Public comment release

Public Health Assessment

Precision Castparts Corporation (PCC)

PCC Structurals Large Parts Campus
4600 SE Harney Drive
Portland, OR 97227
Oct. 29, 2018

Prepared by the
Environmental Health Assessment Program
Public Health Division
Oregon Health Authority
Under cooperative agreement with the
Agency for Toxic Substances and Disease Registry

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Oregon Health Authority
PUBLIC HEALTH DIVISION
Environmental Health Assessment Program

Public comment version

This report is being released for public comment as an opportunity for anyone to review and provide feedback on this document. Comments submitted by the date indicated on the front cover will be addressed in the final version. To submit public comments, either submit them via email to ehap.info@state.or.us or via postal mail addressed to:

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Foreword

This report was supported in part by funding through a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), US Department of Health and Human Services. It was completed in accordance with approved methodologies and procedures existing at the time the Public Health Assessment was initiated. Editorial review was completed by the cooperative agreement partner.

The Oregon Health Authority (OHA), in cooperation with state and federal partners, prepared this Public Health Assessment (PHA). ATSDR and its Oregon cooperative agreement partner, OHA's Environmental Health Assessment Program (EHAP), conducts public health assessments to evaluate environmental data and community concerns. A PHA reviews available information about hazardous substances at a site and evaluates whether exposure to them might cause harm to people.

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Introduction

Through a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), the Oregon Health Authority Environmental Health Assessment Program's (EHAP) priority is to ensure that the community around a site with potential environmental exposures has the best information possible to protect its health.

In 2015, the United States Forest Service (USFS) analyzed moss samples collected around the city of Portland for concentrations of heavy metals. USFS found the highest concentrations of nickel in moss samples collected near the Precision Castparts Large Parts Campus at 4600 SE Harney Drive in Portland, Oregon.

Precision Castparts Corporation (PCC) is a large manufacturer of precision metal castings (known as "investment castings"), forged products and airframe parts based in Portland. PCC is in a mixed commercial, industrial and residential area. It sits on the border of Multnomah and Clackamas counties. The Oregon Department of Environmental Quality collected data on levels of metals and other contaminants in air, water, soil, sediment and crayfish tissue in the area around PCC.

A community group asked EHAP to perform this public health assessment (PHA) to evaluate the potential public health risks of contaminants detected near PCC. A PHA reviews available information about hazardous substances at a site and evaluates whether exposure to them might cause harm to people. PHAs do not determine whether specific environmental exposures caused existing health issues in people.

Limitations of the PHA include the lack of historical sampling data, uncertainties around how well the available monitoring data represent typical ongoing exposures, the inability to differentiate between PCC emissions and emissions from other sources, and uncertainties about potential effects on sensitive populations.

Conclusions

Based on currently available science, monitoring data and guidance from federal agencies, EHAP reached six conclusions about the Precisions Castparts site:

Conclusion 1

Measured concentrations of metals in air near PCC are not likely to harm health.

Cumulative exposure to all metals detected in the air around PCC may be predicted to elevate lifetime cancer risk by as many as 20 additional cases of cancer per 1 million people exposed continuously for a lifetime. EHAP considers this to be very low risk. The estimated cancer risk is similar for current conditions and for conditions prior to HEPA filter installation. These risk calculations are based on the cautious assumption that nickel detected in air monitoring is in its most toxic form. It is likely that nickel emissions from PCC are in an alloy form that may be less available to the body and, therefore, less carcinogenic.

Conclusion 2

Measured concentrations of metals in soil from areas around the PCC facility are not likely to harm health.

DEQ sampled soil near the facility, including locations near residences and in community gardens. No soil concentrations exceeded comparison values.

Conclusion 3

Measured concentrations of chemicals in surface water of Johnson Creek are not likely to harm health.

The levels of chemicals detected in surface water are below health-based comparison values designed to be protective of drinking water. TCE was detected at a level slightly above the cancer CV in one sample in 2009 but was not detected in subsequent samples. Johnson Creek, like many urban streams, has had high levels of bacteria that can make people sick. While bacteria in Johnson Creek is not a focus of this PHA and is not believed to be related to PCC, it has the potential to affect public health.

Conclusion 4

Measured concentrations of PCBs and PAHs in Johnson Creek's sediment near the storm water outfall are not likely to harm the health of people who regularly come in contact with it.

Weekly year-round exposure to sediment is not high enough to harm health. While extremely frequent (daily year-round) contact with Johnson Creek sediment could result in a slight increased risk of both non-cancer and cancer health effects, the likelihood of this degree of contact is quite low. Risk calculations were based on cumulative exposure to maximum concentrations of all PCBs, PAHs and metals of potential concern detected in the creek. Each exposure was assumed to involve full contact of hands, forearms, feet and lower legs with sediment. The biggest health risk from this degree of contact with the creek is the potential for bacterial infections.

Conclusion 5

Residents may safely eat crayfish from Johnson Creek in moderation.

Based on cumulative risk from metals and PCBs, residents can eat up to five meals of Johnson Creek crayfish each month without exceeding health-protective exposure guidelines.

Conclusion 6

There is not enough known about past air emissions from PCC to calculate past health risks before 2016.

No historical monitoring data are available to support a quantitative evaluation of potential health effects of previous exposures. Based on historical trends in emissions reported by PCC to EPA's Toxic Release Inventory, we cannot rule out the possibility that past air concentrations could have been high enough to harm health. Emissions reported to TRI since 1987 indicate that emissions of some chemicals may have been 10 and 100 times higher than current emissions during some periods of PCC's past operations. Historical emissions of trichloroethylene and tetrachloroethylene would have also contributed to past risks of cancer and developmental defects.

For more information

If you have questions about this report, you can contact EHAP by calling 971-673-0977 or toll free 1-877-290-6767 or by emailing ehap.info@state.or.us.

Abbreviations and acronyms

As	arsenic
ATSDR*	Agency for Toxic Substances and Disease Registry
BW	body weight
CAC	community advisory committee
Cd	cadmium
CDC	Centers for Disease Control and Prevention
COC	contaminant of concern
Cr	chromium
Cr6+	hexavalent chromium
CREG	cancer risk guide
CSF	cancer slope factor
CTE	central tendency exposure
CV*	comparison value
DEQ	Oregon Department of Environmental Quality
ED	exposure duration
EHAP	Oregon Environmental Health Assessment Program
EJ	environmental justice
EMEG*	environmental media evaluation guide
EPA*	US Environmental Protection Agency
CALEPA	California Environmental Protection Agency
HEPA	high-efficiency particulate air
HQ	hazard quotient
HVOC	halogenated volatile organic compound
IARC	International Agency for Research on Cancer
IR*	ingestion rate
IRIS	Integrated Risk Information System
IUR	inhalation unit risk

* Abbreviations with an asterisk are defined in the glossary (Appendix H).

LOAEL	lowest observed adverse effect level
MCL	maximum contaminant level
mg/kg*	milligrams per kilogram
MRL	minimal risk level
Ni	nickel
ND	not detected
ng/m3	nanograms per cubic meter
NOAEL*	no observed adverse effect level
NPDES	National Pollutant Discharge Elimination System
OHA	Oregon Health Authority
OSHA	Occupational Safety and Health Administration
PAHs	polycyclic aromatic hydrocarbons
PCBs	polychlorinated biphenyls
PCC	Precision Castparts Corp.
PCE	perchloroethylene
PHA*	public health assessment
ppb	parts-per-billion
ppm	parts-per-million
REL	recommended exposure level
RfC	reference concentration
RfD*	reference dose
RME	reasonable maximum exposure
RSL	regional screening level
SPAQ	South Portland Air Quality
TCE	trichloroethylene
TRI	Toxics Release Inventory
UCL	upper confidence limit
USFS	United States Forest Service
µg/L	microgram per liter
VOC	volatile organic compound

* Abbreviations with an asterisk are defined in the glossary (Appendix H).

Purpose

This public health assessment (PHA) was prepared in response to a request by a neighborhood advocacy group, the South Portland Air Quality (SPAQ), on June 3, 2016. This PHA addresses the potential public health effects of contaminants detected around the Precision Castparts Corp. Large Parts Campus straddling the border between Portland, Oregon, and Milwaukie, Oregon, in Multnomah and Clackamas counties, respectively. The assessment was informed by input from a community advisory committee and focuses on the potential health effects for residents of the nearby Portland neighborhoods of Brentwood-Darlington, Woodstock and Eastmoreland, and the Milwaukie neighborhoods of Lewelling and Ardenwald.

Background

Site description

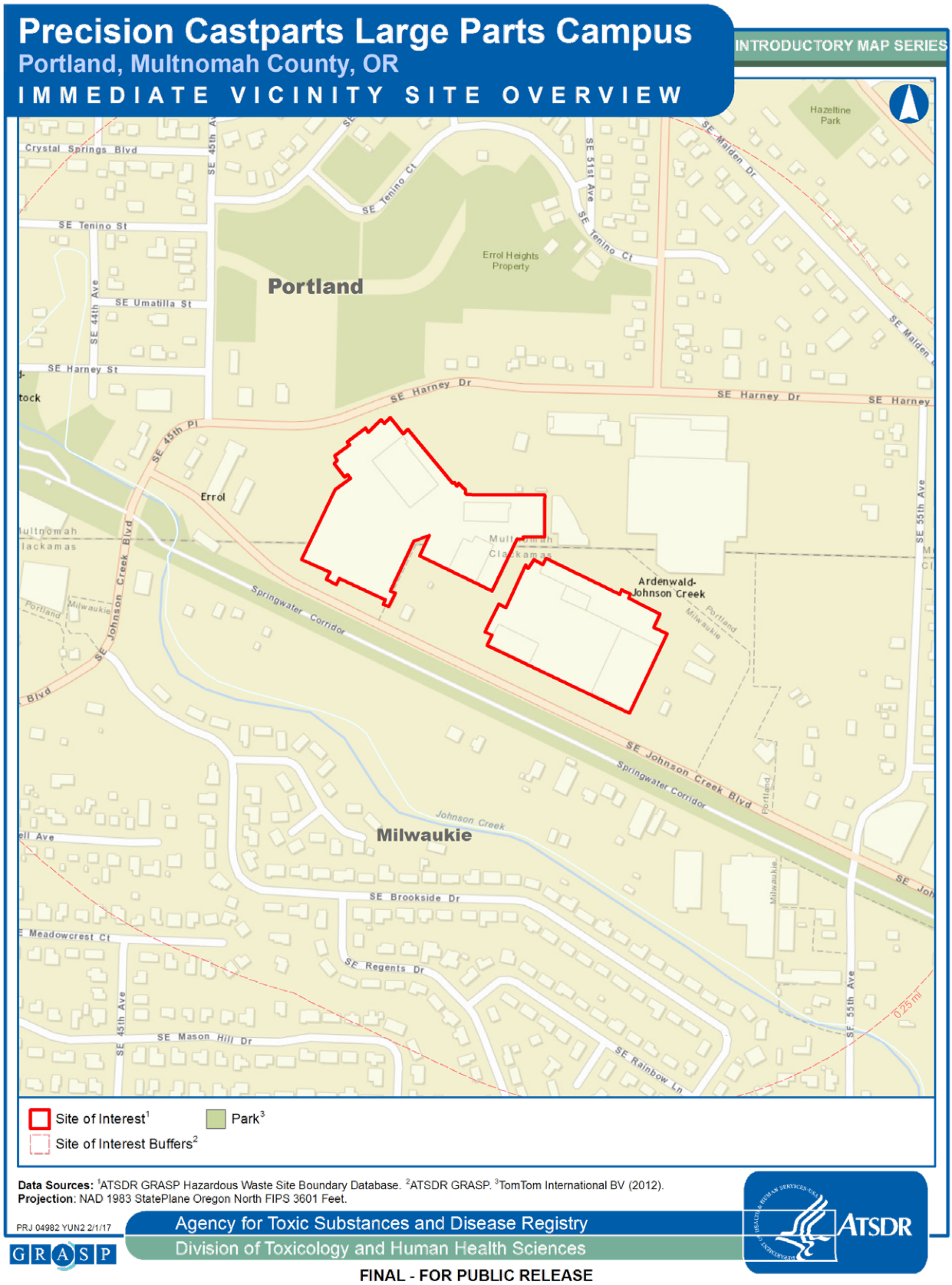
Precision Castparts Corporation (PCC) is a large manufacturer of precision metal castings (known as "investment castings"), forged products and airframe parts based in Portland, Oregon. It ranked 282 on the Fortune 500 list in 2016 (1) and has 162 plants worldwide with multiple manufacturing locations in Oregon (2). PCC is a subsidiary of Berkshire Hathaway (3).

The focus of this public health assessment (PHA) is the PCC Structurals Large Parts Campus located at 4600 SE Harney Drive in Portland, Oregon. The Large Parts Campus (referred to as 'PCC' throughout this document) is PCC's original manufacturing site. At this location, PCC uses investment casting to manufacture parts for a wide range of applications, including aircraft engines, airframes, gas turbines, military armaments and medical devices. The campus houses a stainless-steel casting foundry that uses nickel and cobalt-based alloys and a titanium casting foundry that uses titanium alloys.

PCC is in a mixed commercial, industrial and residential area (Figure 1). It sits on the border of Multnomah and Clackamas counties, with addresses of different buildings on site in both counties. Several small businesses and industrial sites, private residences, Errol Heights City Park, the multi-use Springwater Corridor trail, and Johnson Creek surround the facility. All storm water that falls on the PCC campus is collected in the onsite conveyance system and is treated by a storm water treatment facility that PCC installed in 2016. The treated storm water discharges to a city storm water pipe that drains into a U-shaped bend in Johnson Creek, northwest of the facility. PCC is also near Milwaukie's drinking water aquifer.

PCC began operation at this site in 1957. In addition to PCC, other industrial facilities have operated at this location. The building that currently houses PCC's titanium operations was originally constructed in 1950 for the Oregon Saw Chain Corporation (the original parent company of PCC). In the 1970s the building was used by Code-A-Phone, an electronic communications equipment manufacturer (4).

Figure 1. Area map of the PCC Large Parts Campus and its surroundings



Environmental permits at PCC

PCC operates under several environmental permits that limit emissions allowed from the facility. An Air Contaminant Discharge Permit (ACDP) (5) administered by Oregon Department of Environmental Quality (DEQ) sets PCC's allowable air emissions rates. A National Pollutant Discharge Elimination System (NPDES) permit for storm water discharge, administered by Portland, regulates how the facility directs storm water that falls on facility grounds. As a hazardous waste generator, PCC is also subject to inspections from the Environmental Protection Agency (EPA) and DEQ for hazardous waste treatment, storage and disposal. The DEQ website provides a history of permitting, inspections, penalties and cleanup activities (6). Worker health and safety at the facility is regulated by the federal Occupational Safety and Health Administration (OSHA) in coordination with Oregon OSHA. Records of state and federal OSHA activity at PCC are available online (7).

PCC's ACDP sets a limit on emissions allowed from the facility. The permit requires PCC to report estimates of certain air emissions and perform emissions monitoring. Under this permit, PCC reports air emissions of hazardous air pollutants (including but not limited to nickel, chromium, cobalt, hydrogen chloride, hydrogen fluoride, hexane, lead and manganese) emitted during each 12-month period. As of DEQ's review in 2016, PCC was operating in compliance with the conditions of its permit. However, the most recent EPA National Air Toxics Assessment identified the PCC Large Parts Campus among the facilities in the Portland region with the highest potential to contribute to cancer risk through its air emissions (8). As of the date of this PHA, DEQ is actively working to review PCC's ACDP.

Several additional contaminants — including perchloroethylene (PCE), trichloroethylene (TCE), polychlorinated biphenyls (PCBs) and trace levels of radioactive thorium (9) — were used at the site historically but have since been phased out. While PCC no longer reports use of these chemicals, some have remained in the surrounding environment. Recent monitoring (2009–2015) detected TCE and PCE in groundwater beneath the site and PCBs in solids accumulated in storm water catch basins on site and in Johnson Creek sediment. DEQ's cleanup program initially included thorium, a naturally occurring radioactive substance, among chemicals included in monitoring at the site. Analyses for thorium were discontinued after determining the environmental levels were consistent with naturally occurring background levels. Thorium on site remains regulated by the Oregon Health Authority, under Radioactive Material License No. ORE-90354 (currently Amendment 54, with expiration date April 30, 2022). The license is for natural thorium and is for "possession only of residual contamination in, on, and under facilities, equipment, and surfaces."

Cleanup activities at PCC

In 2008, PCC entered into a voluntary cleanup agreement with DEQ (10). Under this agreement PCC completed extensive soil, storm water, Johnson Creek sediment and groundwater monitoring. PCC recently took several steps to reduce pollution from the facility. In May 2016, PCC added high efficiency particulate air (HEPA) filters to control air emissions from several emissions stacks. PCC also installed a new storm water filtration system to remove metals and PCBs from storm water. In addition, PCC cleaned both the onsite storm water conveyance system and the city storm water lines to the discharge point at Johnson Creek to remove any remaining chemicals that might contribute to ongoing contamination. During 2018, PCC is undertaking soil removal actions and operational facility upgrades and maintenance that will help reduce and control potential pollutant discharges to the onsite storm water conveyance system. DEQ expects to complete its overall site investigation documentation in 2019. The site investigation documentation will comprehensively describe conditions on site and next steps to complete DEQ's regulatory oversight of cleanup activities.

Air toxics concerns in Portland

Some of the recent interest in metal emissions from PCC originated from broader agency efforts to better characterize air pollution sources throughout Portland. In 2009, DEQ developed an air pollution model to predict concentrations of air pollutants at different locations around the city (11). DEQ based the model on several sources of data, including air emissions reported by permitted industrial facilities. DEQ performed air monitoring to evaluate the model. While the model performed well in predicting concentrations of many air pollutants, it underestimated cadmium concentrations. This inconsistency between modeled concentrations and measured air concentrations indicated there were unidentified sources of cadmium emissions in the Portland metropolitan area.

To locate unidentified sources of air toxics in Portland, the US Forest Service and DEQ collaborated in an experimental effort to measure heavy metals in tree moss samples collected throughout the city (12). Moss growing in trees is thought to be a promising indicator of potential air pollution because without contact with soil, contact with air contaminants is the only source of moss exposure to pollution. The moss study identified several locations where metal concentrations in moss were elevated relative to the other locations in Portland (13) (14). These moss study results identified previously unregulated sources of air toxics and ultimately led the Governor to initiate an overhaul of Oregon's industrial air toxics rules (15).

The moss study results brought public attention to elevated concentrations of several metals, including nickel, cobalt, chromium and arsenic in moss samples collected from neighborhoods around PCC. In response, DEQ performed air monitoring (16) to better characterize air pollution around the facility. The study also raised community concerns about potential for metals from air emissions to deposit in soil in nearby neighborhoods' soil. DEQ performed extensive soil sampling to evaluate metal concentrations in soil near PCC (17).

History of community concerns

Community members raised concerns about PCC air emissions prior to 2016. In 2011 a power failure at PCC resulted in the release of a large orange plume of nitrogen oxide from the facility. In response to this emergency, the fire department advised neighbors within a half mile of the facility to stay indoors. Local schools were cancelled for a day to avoid exposure. While PCC has taken steps to avoid similar events in the future, the incident contributed to community concerns around the safety of PCC's operations. In 2013, The Oregonian (18) reported PCC topped a "Toxic 100 Air Polluters Index" produced by the University of Massachusetts (19), prompting neighborhood association and other community calls for the company to move or reduce emissions. After the early 2016 revelations about metals in moss near PCC, in July 2016, six residents of SE Portland filed two separate class-action lawsuits against PCC, stating that toxic air emissions from the PCC facility have harmed their health and affected property values. A new neighborhood advocacy group that formed in 2016, the South Portland Air Quality (SPAQ), has focused on air quality concerns related to PCC. Community meetings on PCC were well attended by SPAQ members, PCC workers, residents, neighbors, gardeners, parents and Springwater Corridor path users. They all voiced concerns about short-term and long-term health effects of facility emissions to air, land and water. In June 2016 SPAQ asked OHA to prepare a public health assessment of PCC emissions.

Potentially affected communities

Residences. PCC is located near several residential neighborhoods, including Brentwood-Darlington, Woodstock and Eastmoreland in Portland (Multnomah County), and Lewelling and Ardenwald in Milwaukie (Clackamas County). The 2010 census reported 2,144 homes and 5,167 residents within one-half mile of the PCC campus (Appendix A).

Small businesses. Immediately neighboring PCC are several small businesses. These firms' employees breathe air near PCC throughout the work day. Businesses at the corner of SE 45th Avenue and SE Harney Drive include a maid service, an equipment rental supplier, restaurants, a carwash, a bakery outlet and a coffee shop with a walk-up window.

Recreation. There are several recreational sites neighboring PCC where people may be exposed to any contaminants present in air, water or soil.

- Errol Heights City Park is north of the facility across Harney Drive (Figure 1). The park is more than 14 acres and contains unpaved walking paths. The Errol Heights Community Garden at the north end of the park holds 28 garden plots (20). Park users may be exposed to air emissions near PCC. Ongoing restoration and park improvement efforts (21) may put workers and volunteers in direct contact with soil in the park and sediment in Errol Creek and associated wetlands. In December 2016, the Portland Parks commissioner announced $5.3 million of funding to support additional park improvement efforts (22) that may temporarily result in additional work crew exposure to local air and soil, and potentially increased park use subsequent to construction.

- Johnson Creek flows along the southern border of the facility (Figure 1). An oxbow in the creek winds northwest of the facility and is the location of the city storm water outfall that releases storm water from PCC. Residents report wading, swimming and collecting crayfish in various spots along the creek. This oxbow is the subject of substantial habitat restoration and erosion control work completed by Portland, which owns the property, during July and August 2018. This work changed the sediment, gravel and cobble surface of the stream bed and added woody debris to the stream to reduce winter water velocities and provide improved fish habitat. A consortium of state and federal agencies with jurisdiction, in consultation with DEQ, required and approved the city work.
- The Springwater Corridor Trail is a multi-use trail that runs along the southern border of the PCC campus (Figure 1). Residents and visitors who frequently bicycle, walk and run along the trail may have higher exposure to air emissions as they breathe more heavily during exercise.

Schools and child care facilities. There are no schools immediately neighboring the PCC campus. One daycare is located just under one-half mile away from PCC. There are five other childcare facilities and six schools within one mile of the facility (Appendix A). Small, informal childcare operations, not registered as business operations, may also be present. Depending on the distance traveled by emissions from PCC, children attending these schools and daycare facilities may have some exposure.

Demographics

The communities neighboring PCC are similar to many communities in Oregon in terms of racial, ethnic and economic makeup. The 2010 census counted 5,167 people living within one-half mile of the facility. Among those, 87% were white. The Hispanic or Latino population more than doubled between 2000 and 2010 and makes up 7.8% of the total population. The median household income ($55,284) is roughly the same as the median income across Portland as a whole ($55,003).

Environmental justice

Low-income communities and communities of color often face disproportionately high levels of exposure to pollution where they live and work (23). These same communities may also be more susceptible to the health effects of environmental exposures (24) (25) due to social stressors, lack of access to health care, nutritional factors and other conditions in which people are born, grow, live, work and age (26). Limited time and resources and language barriers prevent some communities from becoming meaningfully involved in environmental decisions. To highlight potential environmental justice concerns, EHAP identifies groups that may be more exposed or more susceptible to disease, or face barriers to participation in public decision-making processes.

There are some groups and individuals in the community around PCC who may be sensitive to the health effects of pollution due to economic and psychosocial factors (e.g., stress), age and preexisting health conditions, such as asthma. Data from EPA's EJScreen tool (27) indicate people living within one-half mile of PCC have a greater risk of exposure to various environmental risk factors (e.g., exposure to fine particulate matter and ozone) when compared to the state average. Data from the American Community Survey also show a slightly higher than average percentage of children under 5 (7%), and adults 65 years and older (14%) residing in the surrounding neighborhood, compared to the Portland metro area. Other environmental justice demographic indicator values are below Portland metro area and state averages.

Exposure and health analysis

Data sources

This section describes the data EHAP considered in evaluating whether people's health may be harmed by chemical contaminants detected around PCC. All environmental sampling data used for health effects evaluation in this PHA were obtained using EPA-approved methods and technology by certified professionals and technicians. Some supporting data described below helped define the extent of potential contamination and provide additional context but could not be used for quantifying potential health effects.

Data used for health effects evaluation

Air monitoring (performed by DEQ)

DEQ performed air monitoring at three locations (Appendix B) surrounding the PCC facility from March 30, 2016, through October 2016 (28). This monitoring effort captures one month of monitoring data prior to PCC's installation of additional pollution controls and more than six months of data collected under current conditions. All three monitors measured heavy metal concentrations at 24-hour intervals through October 2016. In addition to metals, one of the three monitors measured volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs) and other air toxics. This full spectrum monitor operated for a year, through May 2017 (16). A nearby meteorological station collected data on wind speed and direction throughout the monitoring period.

Soil monitoring (performed by DEQ)

DEQ tested soil for metals at several locations within one mile of the PCC facility in June 2016 (17). DEQ used incremental sampling methods in which multiple samples were analyzed from a single site. This approach ensures that results accurately reflect average concentrations at sites of interest.

Johnson Creek sediment and surface water monitoring (performed by Landau Associates on behalf of PCC)

Since 2009, Landau Associates has monitored Johnson Creek surface water and Johnson Creek sediment samples both upstream and downstream of the city storm water outfall used by PCC. Between 2009 and 2015, Landau Associates collected individual samples at numerous locations in the oxbow portion of Johnson Creek. During this time, surface water and sediment monitoring collected data on a diverse range of chemicals, including

metals, PCBs, PAHs and VOCs. In 2017, additional sampling was performed using an incremental sampling method in which numerous samples taken from an area are combined to determine average concentrations of metals and PCBs in sediment in that area.

Johnson Creek sediment and crayfish monitoring (performed by DEQ)

As part of its statewide toxics monitoring program in 2016, DEQ tested sediment in Johnson Creek both upstream and downstream from the city storm water outfall used by PCC. A composite sediment sample, in which multiple sediment samples were combined for analysis, was tested for metals and PCBs. In addition, a composite sample of eight crayfish collected downstream of the storm water outfall was tested for metals (29).

Supporting data (these data are referenced, but not used as the basis for any risk calculations)

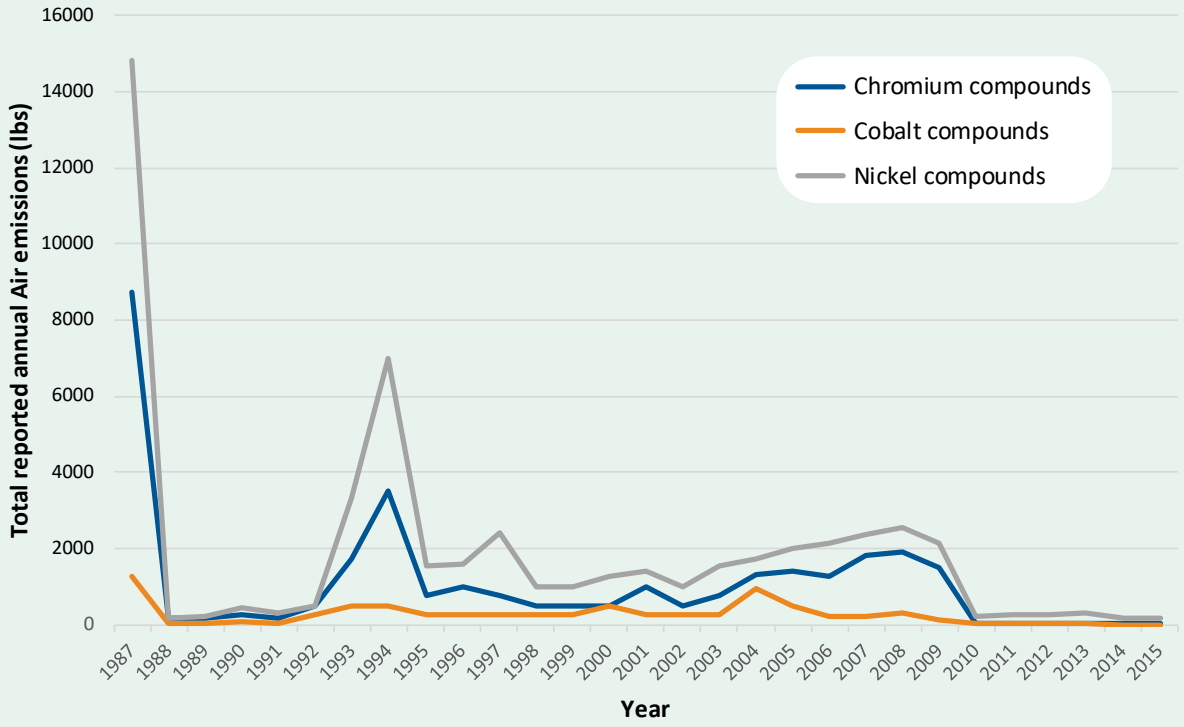
Air emissions reported to the Toxics Release Inventory (submitted by PCC to EPA)

The PCC Large Parts Campus has reported its estimated annual air emissions to EPA's Toxics Release Inventory (TRI) annually since the program began in 1987 (30). The historical emissions trends captured in TRI provide qualitative information about potential historical exposures. PCC emissions reported to TRI indicate that overall air emissions have decreased substantially since 1987 (Figure 2A). In 2015, PCC reported air emissions of aluminum, chromium, cobalt, copper, hydrogen fluoride, nickel and nitric acid. Total reported air releases of nickel, chromium and cobalt compounds have decreased substantially over time (Figure 2B). Trichloroethylene and tetrachloroethylene air emissions were reported historically but were phased out in the early 1990s.

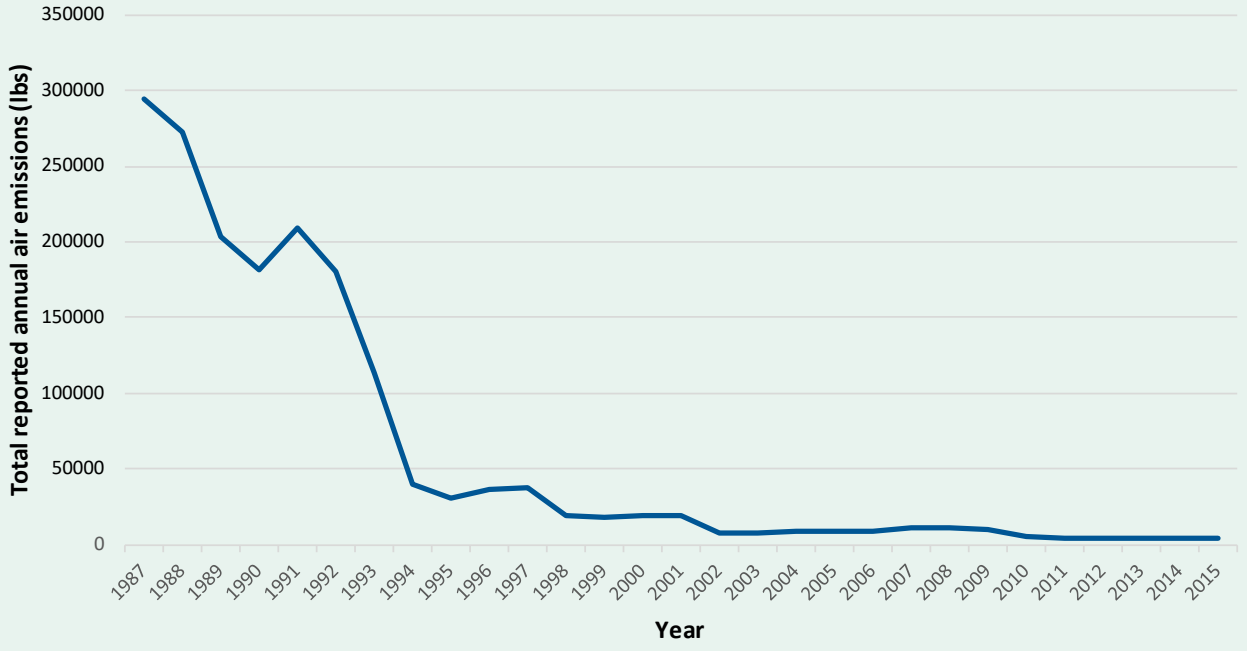
There is uncertainty around emissions reported to TRI. Emissions are estimated based on chemical use and are not confirmed by monitoring data. The methods used to estimate emissions have not been consistent across time, so some changes in emissions reported to TRI simply reflect changes in record keeping. Furthermore, there may be incentive to overestimate reported emissions when those reported emissions are also used to determine emissions limits enforced in permits. Because of these uncertainties, data must be interpreted with caution. TRI data were not used as the basis for risk calculations in this PHA. Additional discussion of appropriate interpretation of TRI data is available on the EPA website (31).

Figure 2. Total estimated air emissions (stack and fugitive emissions of all chemicals) reported to TRI by PCC for all chemicals and for selected metals over time

All chemicals



Selected metals



Metals detected in moss (performed by USFS in collaboration with DEQ)

The US Forest Service (USFS) measured concentrations of heavy metals in moss collected throughout Portland in October 2015 (12). There were no sampling locations neighboring PCC, but moss sampling sites closest to the facility had the highest nickel concentrations in the city (Appendix C). While moss data were useful in identifying areas in need of further air monitoring, the relationship between metal concentrations detected in moss and concentrations detected in air is not understood. Moss data provided an indication of elevated air concentrations but required confirmation from air monitoring.

PCC storm water (performed by both Landau Associates and the city of Portland)

Landau Associates, Inc. as well as Portland have directly monitored storm water from the city storm water pipe used by PCC. Past storm water data provide evidence that PCBs may have entered the creek from the storm water outflow. Since installation of its new storm water treatment system, PCC has analyzed storm water samples collected after treatment but prior to entering the city pipes. According to results submitted by PCC to the city of Portland, under the DEQ issued National Pollution Discharge Elimination (NPDES) permit, no PCBs or PAHs were present at detectable levels in treated storm water samples in 2016 or 2017. While storm water data provide some information about the extent to which storm water from PCC may have increased contamination in Johnson Creek, there is no direct human contact with the storm water itself. Johnson Creek surface water and sediment monitoring data are the focus in this PHA because they represent the potential points of human exposure.

Exposure pathways

For a chemical contaminant to harm human health, there must be a way for people to come into contact with the chemical. An "exposure pathway" describes how a chemical moves from its source and comes into physical contact with people. An exposure pathway has five elements:

- A contaminant source or release
- A way for the chemical to move through the environment to a place where people could come into contact with it
- A place where people could contact the contaminant
- A route of exposure to a contaminant (breathing it, swallowing it, absorbing it through skin, etc.)
- A population that comes in contact with the contaminant

An exposure pathway is considered "completed" if all five of the elements are known to be in place and occurring. If one or more of the elements is unknown, then the exposure pathway is considered a "potential" pathway. If it is known that one of the five elements does not occur, that pathway is "eliminated."

With input from the community advisory committee, EHAP identified four complete exposure pathways (Table 1) and several potential and eliminated pathways (Table 2 and Table 3). In this PHA, we considered potential health effects of contact with chemicals through completed and potential exposure pathways. Eliminated exposure pathways are not evaluated for health effects because no exposure is occurring.

Table 1. Completed exposure pathways

Environ-mental media	Contaminants measured	Potential source of exposure	Potential point of exposure	Exposure route	Potential exposure population	Notes
COMPLETE EXPOSURE PATHWAYS						
AIR	Metals, historical VOC emissions	Air releases from PCC and neighboring sources	Air at nearby households, workplaces, schools, daycare facilities, etc.	Breathing the air	Adults and children living, working and going to school nearby	DEQ air monitoring at three locations around the facility captures one month prior to and > six months following installation of new pollution controls. There is no historical air monitoring data on emissions of metals and TCE.
SOIL	Metals, PCBs, halogenated and non-halogenated VOCs	Soil deposition of air emissions from the facility and direct releases to soil onsite	Soil in yards, residential and community gardens, nature parks (e.g., Errol Heights Nature Park), playgrounds, schoolyards, and construction sites and road paving sites near the PCC facility	Ingestion of soil and produce grown in soil, skin contact with soil, dust inhalation	Adults and children living, playing and gardening nearby; outdoor work/ volunteer crews	DEQ has measured metals in soil offsite to determine how air emissions may have affected soil. PCC has monitored onsite soil for PCBs and VOCs to determine occupational risks of onsite exposures to excavation workers. Recently announced nature park restoration efforts raised concerns about exposures during the restoration and tree planting efforts.
SURFACE WATER (Johnson Creek near the storm water outflow)	Metals and solvents; solvents include PCE and TCE	PCC storm water outflow and other upstream sources	Surface water from Johnson Creek downstream from storm water outfall	Ingestion of water and skin contact with water	Adults and children in contact with Johnson Creek for recreation and restoration efforts	Community members report that people come into contact with Johnson Creek water and sediment (wading, fishing, garden irrigation, etc.).
SEDIMENT (Johnson Creek)	PCBs, PAHs, metals	PCC storm water run-off and storm water outfall into Johnson Creek. Runoff from streets to city conveyance	Sediment in creek, at or downstream from the PCC outfall, or places downstream (where sediment has been transported)	Ingestion of sediment and skin contact with sediment	Adults and children in contact with Johnson Creek for recreation and restoration	Community members report that people come into contact with Johnson Creek water and sediment (wading, fishing, etc.).

Table 2. Potential exposure pathways

Environ-mental media	Contaminants measured	Potential source	Potential point of exposure	Exposure route	Population	Notes
POTENTIAL EXPOSURE PATHWAYS						
INDOOR AIR	Metals	Air releases from PCC and neighboring sources that enter homes and nearby businesses	Air and dust inside nearby households, workplaces, schools, daycare facilities, etc. (Indoor air has not been tested.)	Breathing the air and dust	Adults and children living, working and going to school nearby	There is no monitoring data available for indoor air near PCC. We do not know the extent to which outdoor emissions travelled indoors. Risk calculations in this health assessment assume that people living nearby were exposed to concentrations measured outdoors continuously.
AIR	Metals	Air releases from PCC and neighboring sources that enter homes and nearby businesses	Air at nearby households, workplaces, schools, daycare facilities, etc.	Skin contact with air and dust (Degree of exposure through skin is unknown.)	Adults and children living, working and going to school nearby	The degree of exposure to metals in air through skin is unknown and the potential health effects of exposure through skin are generally not well known.
SOIL	Metals	Soil deposition of air emissions from the facility and direct releases to soil onsite	Locally grown produce	Ingestion of water and skin contact with water	Adults and children in contact with Johnson Creek for recreation and restoration efforts	Community members report that people come into contact with Johnson Creek water and sediment (wading, fishing, garden irrigation, etc.).

Table 3. Eliminated exposure pathways

Environ-mental media	Contaminants measured	Potential source	Potential point of exposure	Exposure route	Population	Notes
ELIMINATED EXPOSURE PATHWAYS						
AIR (vapor intrusion)	Halogenated VOCs, including TCE and PCE	Ground water (migration to soil) or soil (migration from particles into soil gas)	Indoor air from soil gas migration into nearby household or other building (vapor intrusion)	Breathing the air	Adults and children	DEQ continues to monitor potential for TCE and PCE vapor intrusion through remedial investigation as part of the voluntary cleanup agreement (10). While there is some uncertainty about the potential for migration of soil gas onsite to neighboring properties, DEQ has concluded that the solvent concentrations detected in monitoring wells are below levels that would indicate a concern for vapor intrusion offsite.
GROUND WATER	TCE and PCE	Residential wells and community aquifers (Milwaukie drinking water source)	Tap water (from well or community water source), vapors from a shower or hot water use (from well), indoor air (vapor intrusion) at nearby residence or other building	Ingestion, dermal contact and inhalation	Neighboring adults and children on private wells and Milwaukie residents	TCE has been detected in ground water monitoring wells operated by PCC under the voluntary cleanup agreement with DEQ. DEQ has concluded that the plume is not currently at risk of contaminating nearby registered wells or drinking water. All neighboring residents are on public water systems, though it is conceivable that some residents also use unregistered wells that DEQ and OHA are not aware of existing. Milwaukie monitors treated drinking water annually for 300 chemicals, including TCE and PCE. It is in compliance with state and federal law (32).

Screening: Identifying contaminants of concern

To identify contaminants of concern (COCs) that require further evaluation, maximum chemical concentrations detected in air, soil, water and sediment around PCC were evaluated against health-based comparison values (CVs). CVs are chemical concentrations in air, water or soil at which exposure is not expected to harm health. The Agency for Toxic Substances and Disease Registry (ATSDR) and other federal and state government agencies established CVs through a scientific peer-review process based on the health effects data available for each chemical as well as information about how frequently adults and children come in contact with air, water and soil. For each chemical, there are typically several different types of CVs that provide reference concentrations for cancer risk and non-cancer health risks. Reference concentrations also include long-term (chronic) and short-term (acute) exposures, for children and adults. To the extent possible with existing data, CVs are designed to be protective of sensitive health effects in susceptible individuals with frequent exposure.

EHAP screens environmental monitoring data using CVs developed by several different agencies:

- ATSDR cancer risk revaluation guides (CREG)
- ATSDR environmental media evaluation guides (EMEG)
- ATSDR reference dose media evaluation guides (RMEG)
- ATSDR minimal risk levels (MRL)
- EPA regional screening levels (RSL)
- California Environmental Protection Agency (CALEPA) reference exposure levels (REL)
- Oregon DEQ ambient benchmark concentrations (ABC) and action levels for drinking water
- EPA maximum contaminant levels (MCL) and action levels for drinking water for drinking water
- EPA National Ambient Air Quality Standards (NAAQS)

When more than one CV is available for a chemical, EHAP selects CVs according to ATSDR's general hierarchy and best professional judgment (Appendix E). For this screening step, EHAP uses CVs intended to be health-protective of frequent long-term exposures for sensitive populations.

A chemical detected at concentrations above a CV does not necessarily mean harmful health effects will occur. Rather, it indicates the need for closer evaluation of potential risks. In this screening step, chemicals present at concentrations above comparison values are identified as COCs for further evaluation in the "Health effects evaluation" section of this PHA. Chemicals at concentrations below comparison values are not likely to cause health effects, and EHAP/ATSDR does not evaluate them further.

What is a CV?

Comparison values (CVs) are screening tools to identify contaminants of concern at a site. CVs represent the contaminant levels in air, soil or water that people could be exposed to every day and not experience harmful health effects. CVs are not environmental clean-up levels, and chemicals that exceed their CVs will not necessarily pose health risks.

Chemicals detected at concentrations exceeding the selected CV were also compared to alternate CVs for short-term (acute) exposures and for other types of health risks (i.e., cancer vs. non-cancer risks) to ensure that all relevant health effects are evaluated.

Contaminants of concern

Chemicals present at concentrations above health-based comparison values in any media were identified as contaminants of concern requiring closer analysis in the "Health effects evaluation" section of this PHA. Health effects that may be associated with each chemical of potential concern and the sources of health-based comparison values used for screening are described in Appendix F. Contaminants of concern in this PHA include:

- Arsenic
- Cadmium
- Hexavalent chromium
- Nickel
- Polychlorinated biphenyls (PCBs)
- Polycyclic aromatic hydrocarbons (PAHs)

Air screening

Arsenic, cadmium, and hexavalent chromium and nickel are identified as contaminants of concern for cancer risk in air emissions and are evaluated in depth in this PHA. Nickel concentrations detected prior to installation of HEPA filters also exceed non-cancer CVs and are, therefore, evaluated for potential effects on non-cancer health risk.

Concentrations of metals in air detected at DEQ's three daily monitors stationed around PCC were compared to health-based comparison values for each of the metals. Monitoring performed prior to installation of HEPA filters on some PCC emissions stacks was evaluated separately to capture higher metals concentrations that may have been present in the absence of the additional pollution controls (Table 4). There was a decreasing trend in nickel and cobalt concentrations detected after HEPA filter installation (Table 5). Average cadmium concentrations were higher in monitoring performed after filter installation, though the significance of and reason for this increase are unknown. Concentrations of other chemicals didn't change significantly.

Before the installation of HEPA filters, maximum nickel, hexavalent chromium and arsenic concentrations were above CVs based on cancer risk (Table 5; more detailed tables in Appendix E). Maximum nickel concentrations also exceeded the ATSDR MRL (90 ng/m3), a non-cancer comparison value derived from effects on respiratory health. Under current conditions, the maximum concentrations of nickel, hexavalent chromium, arsenic and cadmium exceed comparison values based on cancer risk but are below CVs for non-cancer health endpoints (Table 5; more detailed tables in Appendix E).

Table 4. Air concentrations prior to HEPA filter installation (measured by DEQ March 30–May 16, 2016)

Chemical	Average concentration detected[A] ng/m^3	Maximum concentration detected[B] ng/m^3	Comparison value ng/m^3	Comparison value source (sensitive health endpoint)	Chemical of potential concern?
Arsenic	0.876	5.03	0.23	ATSDR CREG (cancer)	yes
Beryllium	0.007	0.018	0.42	ATSDR CREG (cancer)	no
Cadmium	0.166	0.45	0.56	ATSDR CREG (cancer)	no
Chromium	42.025	60.3	*See hexavalent chromium*		
Cobalt	3.353	36.3	100	ATSDR chronic MRL (respiratory function)	no
Hexavalent chromium	0.306	1.16	0.052	ATSDR CREG (cancer)	yes
Lead	2.260	5.39	150	Oregon ambient benchmark concentration/NAAQS (brain development)	no
Manganese	9.564	31.6	300	ATSDR chronic MRL (neurological function)	no
Nickel[C]	22.279	131	4	EPA Residential RSL (cancer)	yes
Selenium	0.742	1.12	20,000	EPA RSL (selenosis)	no

Contaminants of concern (detected at concentrations exceeding the comparison value) are highlighted in grey.

[a] Highest of average concentrations detected at each of the three monitors

[B] Maximum concentration detected at any of the three monitors

[C] The maximum nickel concentration also exceeds non-cancer comparison values (ATSDR MRL =90ng/m3) based on risk of respiratory effects from chronic exposure.

Table 5. Air concentrations under current conditions (measured by DEQ after installation of HEPA filters; May 17, 2016–Jan. 22, 2017)

Chemical	Average concentration detected[A] ng/m³	Maximum concentration detected[B] ng/m³	Comparison value ng/m³	Comparison value source (sensitive health endpoint)	Chemical of potential concern?
Arsenic	0.663	5.48	0.23	ATSDR CREG (cancer)	yes
Beryllium	0.006	0.018	0.42	ATSDR CREG (cancer)	no
Cadmium	0.683	9.19	0.56	ATSDR CREG (cancer)	no
Chromium	33.554	63.2	*See hexavalent chromium*		
Cobalt	1.181	13.1	100	ATSDR chronic MRL (respiratory function)	no
Hexavalent chromium	0.330	1.7	0.052	ATSDR CREG (cancer)	yes
Lead	1.877	8.65	150	Oregon ambient benchmark concentration/NAAQS (brain development)	no
Manganese	8.807	39.1	300	ATSDR chronic MRL (neurological function)	no
Nickel	9.502	51	4	EPA Residential RSL (cancer)	yes
Selenium	0.729	3.56	20,000	EPA RSL (selenosis)	no

Contaminants of concern (detected at concentrations exceeding the comparison value) are highlighted in grey.

[a] Highest of average concentrations detected at each of the three monitors

[B] Maximum concentration detected at any of the three monitors

Soil screening

No metals exceed ATSDR health guidelines for soil. Therefore, no further analysis is performed on health risks from contact with soil.

The highest metal concentrations detected in DEQ soil samples were compared to health-based CVs for soil. DEQ detected low concentrations of several metals in soil sampling performed near PCC, but none exceeded health-based CVs recommended for use by ATSDR (Table 6). The ATSDR cancer risk guide (CREG) for arsenic is a very conservative (health-protective) value that is below natural background concentrations of arsenic found in soil across the country. For that reason, ATSDR recommends using the environmental media evaluation guide (EMEG) based on chronic child exposures as a comparison value for public health assessment. While arsenic detected in soil near PCC is above ATSDR's CREG for lifetime cancer risk, it is still below ATSDR's recommended EMEG comparison value and within natural background levels typical of Oregon (Table 6).

There is no comparison value available for total chromium in soil. For screening in this PHA, total chromium concentrations were compared to CVs for trivalent chromium. In the absence of independent monitoring for hexavalent chromium, EHAP estimated hexavalent chromium concentrations by multiplying concentrations of total chromium detected in soil near PCC

by 2.2%, the proportion of chromium that EPA and ATSDR estimate will be emitted in the hexavalent format specialty/steel production facilities (33). This EPA estimate is consistent with the air monitoring data near PCC, where average concentrations of measured hexavalent chromium are approximately 1% of average total measured chromium concentrations (Table 4 and Table 5). Using this approach, neither form of chromium exceeded its corresponding comparison value for soil. It should be noted that the hexavalent chromium analyses performed by DEQ did not detect any hexavalent chromium in soil. Therefore, the estimates of 2.2 % may be conservative.

In the absence of a CV for titanium in soil, we used a CV for the more toxic titanium tetrachloride for screening. Maximum concentrations of titanium detected in soil near PCC are below this CV.

Table 6. Soil concentrations (measured by DEQ in June 2016)

Chemical	Average concentration mg/kg (ppm)	Maximum concentration mg/kg (ppm)	Comparison value mg/kg (ppm)	Comparison value source (sensitive health endpoint)	Chemical of potential concern?
Arsenic	4.76	10.9	17	ATSDR chronic child EMEG and RMEG (dermal effects)	no
Beryllium	0.54	0.662	110	ATSDR chronic child EMEG and RMEG (gastrointestinal effects)	no
Cadmium	0.28	0.82	5.7	ATSDR chronic child EMEG (kidney function)	no
Chromium total	53.4	239	86,000	ATSDR child chronic RMEG for trivalent chromium	no
Chromium, hexavalent[A]	1.17	5.26	51	ATSDR chronic child EMEG (intestinal effects)	no
Cobalt	20.17	81	570	ATSDR intermediate child EMEG (blood effects)	no
Iron	27,736.7	36,600	55,000	EPA residential RSL (gastrointestinal effects)	no
Lead	34.17	91.8	400	EPA residential RSL standard for bare soil in children's play areas (brain development)	no
Manganese	706.7	1,030	2,900	ATSDR chronic child RMEG (neurological function)	no
Nickel	123.4	776	1,100	ATSDR chronic child RMEG (decreased body weight)	no
Selenium	0.171	0.36	290	ATSDR chronic child EMEG and RMEG (selenosis)	no
Titanium	1,795	2,680	140,000	EPA residential RSL for titanium tetrachloride; no CVs are available for titanium alone	no
Zinc	100	213	17,000	ATSDR chronic child EMEG (copper deficiency)	no

Contaminants of concern (detected at concentrations exceeding the comparison value) are highlighted in grey.

[A] Estimated by adjusting average and maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33)

Johnson Creek surface water screening

Trichloroethylene (TCE) was the only chemical detected in Johnson Creek surface water above health-based comparison values for drinking water. However, because it was only detected in a single sample taken in 2009, there is insufficient information to calculate potential long-term risk.

Johnson Creek surface water and sediment monitoring data collected for PCC by Landau Associates are evaluated in this PHA because they represent the potential points of human exposure through water. Landau Associates tested surface water for many chemicals, including metals, pesticides, PAHs, PCBs and solvents. Maximum chemical concentrations detected in Johnson Creek surface water at any point between 2009 and 2013 were compared to health-based CVs for drinking water that are designed to be protective of young children. This is a very health-protective comparison because it is unlikely that children drink from or bathe in Johnson Creek as much as they come into contact with drinking water.

Among chemicals detected in Johnson Creek surface water (Table 7), TCE was the only chemical detected above any drinking water CV. Of 12 samples collected in Johnson Creek between 2009 and 2013, TCE was only detected in one set of duplicate samples taken in 2009. TCE was not detected in any samples collected in later years. The level of TCE detected in the 2009 sample was slightly above the drinking water CV for lifetime cancer risk but was below the CV for non-cancer effects on fetal development and the immune system. Cancer risk comparison values are designed to identify levels of contaminants that increase cancer risk over a lifetime of exposure through drinking water. It is not possible to estimate potential long-term exposures from the results of a single surface water sample. Because it is not possible to estimate the potential long-term exposures that would be necessary to calculate cancer risk, no further analysis was done. The failure to detect TCE in subsequent samples means it is unlikely that TCE has been consistently present in Johnson Creek surface water at levels above the drinking water CV.

Water quality monitoring has also detected high concentrations of bacteria in Johnson Creek. E. coli concentrations frequently exceed concentrations of concern for health (34) (35). Risk of bacterial infections is beyond the scope of this PHA, but people who come in contact with the creek should be aware that E. coli in the water does have the potential to make them sick.

Storm water monitoring that detected PCBs indicates that PCBs may have entered the creek from the storm water outflow. However, this data will not be evaluated for human health effects because direct human contact with storm water is expected to be very minimal. No PCBs were detected in storm water analyzed in 2017 following the installation of the new storm water treatment plant.

Groundwater data were not evaluated in this screening analysis because there are no complete exposure pathways through which neighbors would come in contact with groundwater at the onsite locations being monitored by PCC (Table 1, Table 2, Table 3).

Through the voluntary cleanup agreement, DEQ is working with PCC to ensure that existing groundwater contamination does not threaten drinking water sources. Milwaukie performs treatment and monitoring (32) of drinking water fed by the nearby aquifer, providing additional data to confirm that community drinking water is protected.

Table 7. Chemical concentrations in Johnson Creek surface water (measured by Landau Associates 2009–2013)

Chemicals detected	Maximum concentration detected (ppb)	Drinking water comparison Value (ppb)	Comparison value source (sensitive health endpoint)	Chemical of potential concern?
Acetone	1,200	6,300	ATSDR child chronic RMEG (kidney function)	no
Chromium, total	2.3	100	EPA MCLG and EPA MCL (skin reactions)	no
cis-1,2-Dichloroethene	1.4	14	ATSDR child chronic RMEG (kidney weight)	no
Copper	6.8	70	ATSDR child intermediate EMEG (gastrointestinal effects)	no
Lead	1.8	15	EPA action level (brain development)	no
Nickel	2.4	140	ATSDR child chronic RMEG (decreased body weight)	no
Tetrachloroethene	2.66	56	ATSDR child EMEG (color vision impairment)	no
Trichloroethene	1.17	0.43	ATSDR CREG (cancer)	yes
Zinc	20	2,100	ATSDR child EMEG (copper deficiency)	no

Johnson Creek sediment screening

Total PCBs and total PAHs in sediment are evaluated for combined cancer risk. Nickel in sediment is also evaluated for potential non-cancer endpoints.

Maximum concentrations of all chemicals detected in Johnson Creek sediment by Landau Associates and DEQ were compared to soil comparison values. Soil comparison values are designed to be protective of children who play often in contaminated soil in their yard. This is a health-protective comparison. Children are not likely to come in contact with Johnson Creek sediment as much as the soil comparison values assume. Several chemicals have been detected in Johnson Creek sediment at concentrations above soil comparison values (Table 8 and Table 10). These include PCBs, PAHs and nickel.

There are many chemicals that fall in the category of PCBs. Because different PCBs can contribute to the same health effects, the potential health effects for total PCBs are considered both individually and together. Maximum concentrations of total PCBs detected in sampling performed by Landau Associates between 2009 and 2015 were above soil comparison values for cancer risk (Table 8). These PCB concentrations were below non-cancer comparison values designed to be protective of effects on the immune system from PCBs. All PCB concentrations detected by Landau Associates in 2017 were below both cancer and non-cancer comparison values (Table 9).

Like PCBs, PAHs are a class of chemicals that may contribute to the same health effects. The potential health effects of PAHs are, therefore, considered both individually and together. In sampling performed by Landau Associates during 2009–2015, maximum concentrations of total PAHs exceeded soil comparison values for cancer risk. Maximum concentrations of the PAH benzo(a)pyrene were below non-cancer comparison values designed to be protective of neurodevelopmental effects. PAHs were not included in sediment monitoring performed by DEQ in 2016 or by Landau Associates in 2017.

In monitoring performed by Landau Associates during 2009–2015 and by DEQ in 2016, maximum concentrations of nickel in sediment exceeded soil comparison values based on the non-cancer health effects associated with chronic oral exposure (Table 8 and Table 10). In monitoring performed by Landau Associates in 2017, concentrations of nickel and all other metals were below soil comparison values (Table 9).

Table 8. Chemical concentrations detected in Johnson Creek sediment (discrete samples measured by Landau Associates 2009–2015)

Chemicals detected	Max concentration detected (ppm)	Soil comparison value (ppm)	Comparison value source (sensitive health endpoint)	Chemical of potential concern?
Antimony	0.66	23	ATSDR child chronic RMEG (blood glucose and cholesterol regulation)	no
Arsenic	6.56	17	ATSDR child chronic EMEG (dermal effects)	no
Barium	1.05	11,000	ATSDR child chronic EMEG (kidney function)	no
Beryllium	0.41	110	ATSDR child chronic EMEG (gastrointestinal effects)	no
Cadmium	0.67	5.7	ATSDR child chronic EMEG (kidney function)	no
Chromium, Total	1000	86,000	ATSDR chronic child RMEG for trivalent chromium	no
Chromium, Hexavalent[A]	22	51	ATSDR chronic child EMEG (intestinal effects)	no
Copper	100	570	ATSDR child intermediate EMEG (gastrointestinal effects)	no
Lead	61.8	400	EPA residential RSL standard for bare soil in children's play areas (brain development)	no
Mercury	0.20	17	ATSDR child chronic EMEG for methylmercury (brain development)	no
Nickel	2,500	1,100	ATSDR child chronic RMEG (decreased body weight)	yes
Zinc	260	17,000	ATSDR child chronic EMEG (copper deficiency)	no
Total PCB[B]	0.48	0.19	ATSDR CREG (cancer)	yes
Total PAH[B]	0.336	0.12	ATSDR CREG for benzo(a)pyrene (cancer)	yes

Contaminants of concern (detected at concentrations exceeding the comparison value) are highlighted in grey.

[A] Estimated by adjusting maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33)

[B] Reflects the maximum sum of PCB or PAH concentrations detected in any individual sediment sample. Total PAH concentrations are the sum of 'benzo(a)pyrene equivalent' concentrations (the detected concentration multiplied by EPA's chemical-specific relative potency factor) for all PAHs detected in each sample. Complete summaries of individual PAH and PCB (aroclor) concentrations are in Appendix E.

Table 9. Chemical concentrations detected in Johnson Creek sediment (incremental samples measured by Landau Associates in 2017)

Chemicals detected	Max concentration detected (ppm)	Soil comparison value (ppm)	Comparison value source (sensitive health endpoint)	Chemical of potential concern?
Antimony	<0.5[B]	23	ATSDR child chronic RMEG (blood glucose and cholesterol regulation)	no
Arsenic	2.57	17	ATSDR child chronic EMEG (dermal effects)	no
Beryllium	0.478	110	ATSDR child chronic EMEG (gastrointestinal effects)	no
Cadmium	<0.5[B]	5.7	ATSDR child chronic EMEG (kidney function)	no
Chromium, total	23.3	75,000	ATSDR chronic child RMEG for trivalent chromium	no
Chromium, hexavalent[A]	0.51	51	ATSDR chronic child EMEG (intestinal effects)	no
Copper	30.7	570	ATSDR child intermediate EMEG (gastrointestinal effects)	no
Lead	27.9	400	EPA residential RSL standard for bare soil in children's play areas (brain development)	no
Mercury	0.0657[C]	17	ATSDR child chronic EMEG for methylmercury (brain development)	no
Nickel	49.8	1,100	ATSDR child chronic RMEG (decreased body weight)	no
Selenium	<1[B]	290	ATSDR child chronic EMEG and RMEG (selenosis)	no
Silver	<0.5[B]	290	ATSDR child chronic RMEG (dermal effects)	no
Thallium	<0.5[B]	NA	NA	no
Zinc	197	17,000	ATSDR child chronic EMEG (copper deficiency)	no
Total PCB	0.1299[C]	0.19	ATSDR CREG (cancer)	no

NA indicates comparison values are not available.

[A] Estimated by adjusting average and maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33)

[B] The chemical was not detected above the sample quantitation limit shown. These chemicals will not be included in further analysis.

[C] Concentration was estimated because the chemical was detected, but it is below the level that can be accurately quantified.

Table 10. Chemical concentrations detected in Johnson Creek sediment (collected by DEQ in 2016)

Chemical	Result (mg/kg)	Soil comparison value (ppm)	Comparison value source	Chemical of potential concern?
Aluminum, total	16,900	57,000	ATSDR child chronic EMEG (motor function)	no
Antimony, total	0.39	23	ATSDR child chronic RMEG (blood glucose and cholesterol regulation)	no
Arsenic, total	2.27	17	ATSDR child chronic EMEG (dermal effects)	no
Barium, total	114	11,000	ATSDR child chronic EMEG (nerve function)	no
Cadmium, total	0.22	5.7	ATSDR child chronic EMEG (kidney function)	no
Chromium, total	476	75,000	ATSDR child chronic RMEG for trivalent chromium	no
Chromium, hexavalent[A]	10.5	51	ATSDR chronic child EMEG (intestinal effects)	no
Cobalt, total	131	570	ATSDR child intermediate EMEG (blood effects)	no
Copper, total	42.4	570	ATSDR child intermediate EMEG (gastrointestinal effects)	no
Lead, total	42.3	400	EPA residential RSL standard for bare soil in children's play areas (brain development)	no
Manganese, total	268	2,900	ATSDR child chronic RMEG (brain effects)	no
Mercury, total	<0.040[B]	17	ATSDR child chronic EMEG for methylmercury (brain development)	no
Nickel, total	1,600	1,100	ATSDR child chronic RMEG (decreased body weight)	yes
Selenium, total	<1.99[B]	290	ATSDR child chronic EMEG and RMEG (selenosis)	no
Silver, total	<0.10[B]	290	ATSDR child chronic RMEG (dermal effects)	no
Thallium, total	<0.10[B]	NA	NA	no
Zinc, total	179	17,000	ATSDR child chronic EMEG (copper deficiency)	no

Contaminants of concern (detected at concentrations exceeding the comparison value) are highlighted in grey.

NA indicates comparison values are not available.

[A] Estimated by adjusting average and maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33)

[B] The chemical was not detected above the reporting limit shown.

Johnson Creek crayfish screening

Arsenic, chromium, cobalt, mercury, nickel, zinc and PCBs were all detected in crayfish samples from Johnson Creek. Levels of these contaminants were considered in calculating the number of Johnson Creek crayfish meals that people can safely eat each month.

DEQ measured metal and PCB concentrations in a combined sample of eight crayfish caught in Johnson Creek downstream of the city storm water outfall used by PCC. There are no screening values available for crayfish. Therefore, all chemicals that were detected in crayfish (Table 11) are included in a more thorough analysis of potential exposures from eating crayfish.

Table 11. Chemical concentrations measured in crayfish collected in Johnson Creek (collected by DEQ in 2016; analyzed in 2017)

Chemical	Concentration in crayfish
Arsenic, total	0.28
Cadmium, total	< 0.03[A]
Chromium, total	0.63
Cobalt, total	0.26
Mercury, total	0.019
Nickel, total	1.08
PCB, total	0.033
Selenium, total	< 0.59[A]
Titanium, total	11.8
Zinc, total	24.1

[A] The chemical was not detected above the sample quantitation limit shown. These chemicals will not be included in further analysis.

Health effects evaluation

To assess whether environmental contaminants at a specific site could harm health, EHAP estimates how much of each contaminant could get into people's bodies. In toxicology, this is referred to as the "dose." EHAP uses a process similar to EPA's human health risk assessment to calculate the exposure doses people might get from contact with chemicals at a site. In the screening step of this PHA, EHAP identified COCs in air under current and past conditions and in sediment at Johnson Creek. Here we evaluate potential health effects by calculating exposure doses for each of the COCs and comparing calculated doses to health-based guidelines for cancer and non-cancer related health risk.

EHAP calculated exposure doses for a set of exposure scenarios designed to capture worst case scenarios in which people are exposed consistently over long periods of time (Table 12). EHAP also identified exposure scenarios for which there is insufficient data to calculate health risks (Table 13). EHAP considered input from local residents on specific exposure scenarios and assumptions that may occur near PCC. We evaluated potential for cancer and non-cancer health effects based on exposure doses calculated from these worst-case exposure scenarios. In cases where multiple chemicals affect the same health outcomes, EHAP evaluated the cumulative risks of all relevant chemicals across all pathways.

This section describes how doses were calculated for each scenario and how they were compared with cancer and non-cancer health guidelines to determine potential risk. It then summarizes the health implications for people in each of the three exposure scenarios.

Table 12. Exposure scenarios evaluated in health risk calculations (for each complete exposure pathway containing COCs)

Exposure scenario	Exposure routes	Rationale
1. Long-term residents exposed to air concentrations measured in 2016 prior to HEPA filter installation (59 years including childhood)[A,B]	Inhalation	Residents who were born, grew up as children and lived as adults around the PCC facility and were exposed to air concentrations measured prior to HEPA filter installation in 2016 for up to 59 years.[B] This hypothetical scenario assumes that 2016 monitoring data would be an accurate reflection of all historical exposures.
2. Long-term residents exposed to current air concentrations, after HEPA filter installation (78 years including childhood)[A]	Inhalation	Residents who are born, grow up as children, and will live as adults around the PCC facility may be exposed to emissions at concentrations measured following HEPA filter installation for up to 78 years.
3. Long-term, frequent recreational contact with Johnson Creek sediment (78 years including childhood)[A]	Ingestion and dermal contact with sediment	Community members raised concerns about potential health effects of contact with contaminants in Johnson Creek. Long-term residents may be exposed over the course of a 78-year lifetime.
4. Long-term, frequent fishing from Johnson Creek	Ingestion of crayfish	Community members raised concerns about potential health effects of eating crayfish from Johnson Creek. The number of crayfish meals that can be safely consumed each month is calculated based on non-cancer risks.

[A] Risk from exposure over a 78-year lifetime was calculated assuming that the first 21 years reflect exposure as a child. Where appropriate, risks of exposure during childhood were adjusted to reflect differences in children's exposure factors (such as frequency or body weight). Risk from early childhood exposure to mutagenic chemicals was weighted as described further in Appendix G.

[B] PCC has been in operation since 1957, so 59 years is the maximum number of years a person may have been exposed to pre-HEPA filter concentrations.

[C] Emissions reported to EPA's Toxics Release Inventory were higher in the past (see pages 20–21 of this assessment).

Table 13. Exposure scenarios for which there is insufficient information to calculate health risks

Exposure scenario	Exposure routes	Rationale
5. Long-term residents exposed to unknown past air concentrations (59 years including childhood)[A,B]	Inhalation	Residents who were born, grew up as children, and lived as adults around the PCC facility were exposed to unknown historical levels of air emissions for up to 59 years.[B] Historical exposures were likely higher than what was measured in 2016 air monitoring based on required company reports to the EPA Toxics Release Inventory showing a decline in the use of COCs over time.[C] There is insufficient information to quantify those past risks.

[A] Risk from exposure over a 78-year lifetime was calculated assuming that the first 21 years reflect exposure as a child. Where appropriate, risks of exposure during childhood were adjusted to reflect differences in children's exposure factors (such as frequency or body weight). Risk from early childhood exposure to mutagenic chemicals was weighted as described further in Appendix G.

[B] PCC has been in operation since 1957, so 59 years is the maximum number of years a person may have been exposed to pre-HEPA filter concentrations.

[C] Emissions reported to EPA's Toxics Release Inventory were higher in the past (see pages 20–21 of this assessment).

Approach to dose calculation

To calculate a dose, we determined the frequency and duration with which people come into contact with the COCs through each exposure pathway. Wherever possible, EHAP uses site-specific information, but when that information is unavailable, we use default values established by ATSDR or the EPA. Where default values are unavailable, EHAP uses best professional judgment. For the complete list of the exposure assumptions and formulas used to calculate doses of COCs in this report, see Appendix G.

To calculate long-term doses in this PHA, EHAP used health-protective assumptions to estimate potential chemical concentrations that people may be exposed to in air consistently over many years. This helps to account for uncertainties around how well monitoring data collected over a limited period reflect what is typically in the air (average concentration). Health protective estimates of average concentrations were calculated by defining a range that we can have 95% confidence will include the true average. The high end of this range is the upper confidence limit. EHAP used EPA's ProUCL software to identify upper confidence limits for average air concentrations based on available monitoring data at each location (resulting UCLs are included in air screening tables in Appendix D). In risk calculations, EHAP used the upper confidence limits identified in ProUCL to represent potential average long-term exposures to air contaminants. To calculate long-term doses to contaminants detected in sediment we use the maximum concentrations detected because there is not enough data at each sampling location to define confidence limits.

Approach to estimating cancer risk

There is no threshold below which cancer-causing chemicals are considered completely safe. Every additional exposure, no matter how small, has the potential to contribute toward lifetime risk of getting cancer. Cancer risk from a specific exposure is, therefore, expressed as a probability, which can be thought of in terms of additional cancer cases in a population. Cancer risk from a particular environmental exposure is considered in addition to the background risk of developing cancer over a lifetime. The American Cancer Society estimates that one in three women and one in two men will develop some type of cancer over the course of their life (36). These background cancers are attributed to a combination of genetic mutations, inherited conditions (traits that are passed on to children), tobacco use, lifestyle factors, common environmental exposures and

What is an ATSDR MRL?

Minimal risk levels (MRLs) are estimates of daily human exposure to a hazardous substance. They represent the amount of a substance that is not expected to cause non-cancer health effects. Exposure doses that are greater than MRLs do not necessarily mean that people will experience the associated adverse effects.

ATSDR develops MRLs for acute (14 days or less), intermediate (between 15 and 364 days) and chronic (one or more years) exposure durations.

occupational exposures. The contributions of each factor to the incidence of cancer in individuals and communities is difficult to predict or quantify.

Cancer risk is generally expressed in terms of chances in a million ($1x10^{-6}$ or 0.000001). For example, a one-in-a-million cancer risk means that for every 1 million people with the same site-specific exposure for the same period, one additional person will develop cancer due to that exposure at some point in their lifetime. This one-in-a-million increase of cancer is in addition to the roughly 400,000 people out of 1 million (approximate background rate for men and women) that would be expected to get cancer from all causes combined. It is not possible to determine which one of the 400,001 cancer cases is the additional case due to a site-specific exposure. In a community of 10,000 people, a one-in-a-million cancer risk means that less than one additional cancer case would be expected.

Cancer risk that falls between one additional case of cancer per million people ($1x10^{-6}$) and one additional case per 10,000 people ($1x10^{-4}$) is generally considered low. It is important to know that this range is *in addition* to the one out of three women or one out of two men who will develop cancer over their lifetime from all causes combined.

What is a hazard quotient?

Hazard quotients (HQs) summarize potential risk of non-cancer health effects. They are calculated by dividing the estimated exposure by a health guideline (such as an ASTDR MRL or an EPA reference dose).

An HQ less than one means that estimated exposure is below health guidelines and no non-cancer health effects are expected.

An HQ greater than one means that estimated exposure exceeds health guidelines and further analysis is needed to determine whether health could be harmed.

To calculate lifetime cancer risk, EHAP uses cancer slope factors (CSF) identified by EPA for each cancer-causing chemical. Cancer slope factors (or in the case of air exposures, inhalation unit risk) describe the increased cancer risk associated with each additional unit of exposure based on the best available data. Cancer risk is estimated by multiplying the calculated dose by the cancer slope factor (Appendix G). In this PHA, when more than one chemical contributed to cancer risk in a given exposure scenario, the risks from all chemicals were added together for an estimate of cumulative cancer risk.

Approach to estimating non-cancer risk

For many non-cancer health effects, there is thought to be a threshold of exposure below which no health effects are expected. Federal health guidelines are intended to identify a daily dose of a chemical that is below this threshold for each chemical and, therefore, unlikely to harm health. To calculate risks for non-cancer health outcomes, EHAP compares the daily doses calculated for each exposure scenario with health guideline doses at which no health effect is anticipated for that chemical.

In this PHA, EHAP used the health guidelines established by ATSDR, called minimal risk levels (MRLs), whenever available. When a specific chemical does not have an appropriate MRL, EHAP uses a reference dose (RfD) or, in the case of inhalation exposures, a reference concentration (RfC) established by the EPA. Appendix F describes the potential health effects and derivation of MRLs and RfDs for each of the COCs identified in this PHA. No contaminants of concern were detected at concentrations high enough to indicate potential acute or intermediate health risks. We evaluated potential long-term health risks by comparing chronic MRLs or RfDs to doses calculated based on long-term exposures.

EHAP divides calculated doses by the health guideline for each chemical (Appendix G). The resulting number is called the hazard quotient (HQ). An HQ greater than 1 indicates that potential exposures exceed the MRL or RfD. When an HQ is less than or equal to 1, the exposure is lower than or equal to the health guideline, and it is unlikely that non-cancer health effects will occur. If it is greater than 1, the exposure is higher than the health guideline and a more in-depth analysis is needed to determine whether an exposed person could experience adverse health effects that are not cancer. In this PHA, nickel was the only chemical evaluated for non-cancer health endpoints because it was the only chemical to exceed non-cancer comparison values for air or sediment concentrations.

Results of risk calculations

Exposure Scenario 1: Long-term residents with hypothetical exposure to air concentrations assumed to constantly be at levels measured in 2016 prior to HEPA filter installation

This hypothetical scenario reflects risks that would occur if people were exposed to concentrations detected prior to HEPA filter installation in 2016 for as long as the facility has been in operation. Because PCC has only been operating since 1957, total lifetime exposures under pre-HEPA filter conditions cannot exceed 59 years of lifetime exposure. It is important to note that in the absence of historical monitoring data, risk estimates calculated in this scenario only reflect risk of long-term exposure to levels of metals detected in 2016 monitoring prior to HEPA filter installation. They do not reflect risks from higher rates of emissions reported historically (described in Exposure Scenario 5 on page 40).

Risk associated with air concentrations detected in 2016 prior to installation of HEPA filters was calculated for levels detected at each of the three air monitoring locations. Exposure doses were calculated based on the upper confidence limit of average air concentrations calculated for each location (Appendix G). Exposure was assumed to be constant for 24 hours a day, 365 days a year over 59 years, including childhood.

Cancer risk

Cancer risk was evaluated cumulatively for all metals detected in air under pre-HEPA filter conditions. Cadmium was not identified as a COC on its own but was included in the cumulative evaluation to ensure that all potential cancer risk was fully accounted for. The maximum cumulative lifetime cancer risk calculated for any monitoring location was 20 in 1 million (Table 14). EHAP considers this to be a very low cancer risk (see discussion on page 36). ***EHAP concludes that levels of metals measured in air in 2016 prior to HEPA filter installation pose very low cancer risk to long-term residents exposed as both children and adults.***

Non-cancer risk

Under pre-HEPA filter conditions, long-term nickel exposure concentrations calculated in this section were below the ATSDR chronic MRL designed to be protective against respiratory health effects (Table 14). This produced a hazard quotient less than 1, which EHAP considers too low to affect public health. ***EHAP concludes that measured concentrations of metals in air prior to HEPA filter installation were too low to harm the respiratory health of long-term residents exposed as both children and adults.***

Table 14. Chronic risks calculated for each air monitoring location (before HEPA filters were installed)

Monitoring location	Scenario	Exposure assumptions	Cumulative cancer risk of Ni[A], As, Cd, Cr 6+	Hazard quotient for non-cancer risk from Ni
Milwaukie Johnson Creek	Lifetime	Constant exposure from birth to age 59	20 in 1,000,000	0.4
S.E. 45th and Harney Drive	Lifetime	Constant exposure from birth to age 59	7 in 1,000,000	NA
S.E. Harney Drive	Lifetime	Constant exposure from birth to age 59	9 in 1,000,000	NA

[A] Assuming nickel is present in the most toxic form

Exposure Scenario 2: Long-term residents exposed to air under current conditions

This scenario assumes that long-term residents may continue to be exposed to concentrations of metals detected in air after HEPA filter installation in 2016 over a lifetime. Health risks associated with air concentrations of COCs detected after HEPA filter installation were calculated separately for each of the three air monitoring locations. At each location, exposure doses were calculated based on the upper confidence limit of average air concentrations (Appendix G). Exposure was assumed to be constant for 24 hours a day, 365 days a year over a 78-year lifetime including childhood.

Cancer risk

Cancer risk for all four COCs in air under current conditions was evaluated cumulatively; that is, the analysis estimated the combined cancer risk of the COCs taken together. Nickel was assumed to be present in its most toxic form, an insoluble particulate such as refinery dust. Because hexavalent chromium causes cancer through gene mutations, early childhood exposures may disproportionately increase lifetime cancer risk. Exposures to hexavalent chromium during childhood were, therefore, given additional weight in the risk calculation, consistent with ATSDR guidance. The maximum cumulative lifetime cancer risk calculated for any monitoring location was 10 in 1 million (Table 15). EHAP considers this to be a very low cancer risk (see discussion on page 42). ***EHAP concludes that metals in air under current conditions pose very low cancer risk to long-term residents exposed as both children and adults.***

Non-cancer risk

None of the metals detected in air under current conditions were present at concentrations high enough to be of concern for non-cancer health risks. ***EHAP concludes that concentrations of metals in air under current conditions are too low to harm the respiratory health of long-term residents exposed as both children and adults.***

Table 15. Risks calculated for each air monitoring location (under current conditions)

Monitoring location	Scenario	Exposure assumptions	Cumulative cancer risk of Ni[A], As, Cd, Cr 6+	Hazard quotient for non-cancer risk from Ni
Milwaukie Johnson Creek	Lifetime	Constant exposure from birth to age 78	10 in 1,000,000	NA
S.E. 45th and Harney Drive	Lifetime	Constant exposure from birth to age 78	6 in 1,000,000	NA
S.E. Harney Drive	Lifetime	Constant exposure from birth to age 78	10 in 1,000,000	NA

[A] Assuming nickel is present in the most toxic form

Exposure Scenario 3: Long-term frequent recreational contact with Johnson Creek sediment via both ingestion and skin contact

Exposure to chemicals in sediment may occur through skin (dermal) contact as well as through incidental ingestion of sediment. Because methods and locations of sediment sampling efforts vary, data are not directly comparable. Therefore, the data can't be integrated to confidently predict average concentrations across sampling efforts. Potential exposure doses were calculated based on maximum levels of PCBs, PAHs and nickel detected in Johnson Creek sediment sampled by Landau Associates or DEQ. Exposure doses were calculated assuming a high frequency of contact with creek sediment. Substantial contact with creek sediment was assumed to occur weekly, year-round (equivalent to four

days a week in the summer months only) between ages 1 and 21 years and for 33 years as an adult (this is ATSDR's default residential occupancy period). These exposure scenarios use conservative assumptions. Dermal exposure is assumed to occur with sediment in direct contact with hands, forearms, feet and lower legs; high rates of absorption are assumed. Oral ingestion was calculated based on the assumption that children may swallow 200mg and adults swallow 100mg of sediment each day they come in contact with the creek. These estimates are derived from EPA's upper bound estimates for soil ingestion rates (37).

In response to community advisory committee members' requests for exposure scenarios that reflect an extreme worst case, EHAP also considered an alternate extreme exposure scenario in which the same high degree of contact with sediment occurred daily all year-round (Appendix G). This scenario used the same assumptions as above about the extent of dermal contact and ingestion that occurs with each exposure. While we are not aware of any individuals with this amount of contact, this extreme scenario provides an upper limit for potential risk.

Cancer risk

To calculate cancer risk from exposure to COCs in sediment, risks from exposure through skin contact and through ingestion were considered cumulatively. Cumulative cancer risk was calculated for total PCBs and total PAHs across both exposure pathways. Because some PAHs cause cancer through gene mutations, early childhood PAH exposures may disproportionately increase lifetime cancer risk. Exposures to total PAHs during childhood were, therefore, given additional weight in the risk calculation, consistent with ATSDR guidance. No cancer risk values are available for oral exposure to nickel and hexavalent chromium and were, therefore, not included (Table 16). Cumulative cancer risk of total PCBs and total PAHs over a lifetime of weekly exposure through both pathways was estimated to be 40 in 1 million, which EHAP considers to be a very low cancer risk (see discussion on p.36). In an extreme exposure scenario of daily year-round exposure, cumulative lifetime cancer risk was estimated to be 3 in 10,000. EHAP considers this to be a low increased cancer risk. However, EHAP is not currently aware of any individuals at risk of coming in contact with Johnson Creek sediment with anywhere near this frequency. ***EHAP concludes that PCBs and PAHs in Johnson Creek sediment pose very low lifetime cancer risk for anyone with frequent (weekly year-round) contact.***

Non-cancer risk

Risk of non-cancer health effects of nickel was calculated based on ingestion of soil only because nickel is not readily absorbed through skin. Assuming weekly year-round contact with sediment, non-cancer risk of nickel for all age groups was below a hazard quotient of 1 (Table 16). In an extreme exposure scenario of daily year-round contact, hazard quotients for most age groups in this scenario were below 1. For the 1–2 year-old age group, the hazard quotient associated with daily year-round exposure was 2, indicating the potential for daily exposure to exceed the health-based comparison value for chronic health effects. It is important to note that

there is still a substantial amount of caution built in to this chronic comparison value, making it unlikely that daily exposure at that level would result in health effects. Furthermore, EHAP is not currently aware of any individuals at risk of coming in contact with Johnson Creek sediment with daily frequency. ***EHAP concludes that maximum concentrations of nickel detected in Johnson Creek sediment are too low to have non-cancer health effects for anyone with frequent (weekly year-round) contact.***

Table 16. Cancer risk associated with contact with weekly year-round exposure to PCBs and PAHs at maximum concentrations detected in sediment

Exposure period	Cumulative cancer risk from skin contact and ingestion of PCBs and PAHs in sediment	Hazard quotient for ingestion of nickel in sediment
Child 6 wks to < 1 yr[A]	0	0
Child 1 to < 2 yr[A]	5 in 1,000,000	0.3
Child 2 to < 6 yr[A]	6 in 1,000,000	0.2
Child 6 to < 11 yr[A]	6 in 1,000,000	0.1
Child 11 to <16 yr[A]	5 in 1,000,000	0.1
Child 16 to <21 yr	3 in 1,000,000	0.05
Cumulative child 0–21 years	30 in 1,000,000	NA
Adult for 33 years (95% residential occupancy period)[B]	20 in 1,000,000	0.02
Lifetime (21 years of childhood exposure plus 33 years of adult exposure)[A,B]	40 in 1,000,000	NA

[A] Cancer risks calculated for exposure to PAHs incorporate age-adjustment factors that give more weight to early childhood exposures due to the mutagenic mode of action of some PAHs (described in more detail in Appendix G).

[B] 33 years is the default duration of residential exposures used by ATSDR based on the 95% residential occupancy period.

Exposure Scenario 4: Long-term, frequent consumption of crayfish from Johnson Creek

Health risks associated with eating crayfish caught in Johnson Creek were evaluated using the same method used in Oregon Health Authority's fish advisory program (38). The concentrations of metals and PCBs detected in crayfish collected from Johnson Creek were used to calculate the number of Johnson Creek crayfish meals that can be safely eaten in a month.

Cancer risk

Fish advisories in Oregon are not based on small increases in cancer risk because the small increased risk of cancer needs to be balanced by the health benefits of eating fish. Among the chemicals DEQ detected in Johnson Creek crayfish, arsenic and PCBs are the only chemicals associated with increased risk of cancer when exposure occurs through ingestion.

Non-cancer risk

The concentrations of metals and PCBs detected in crayfish were used to calculate the amount of crayfish that could be eaten in a month without exceeding non-cancer comparison values for oral exposure to those contaminants (Appendix G). The health risks of all contaminants detected in the crayfish are considered for each chemical alone as well as for combined risk from chemicals that affect the same organ system (Table 17). Based on cumulative risk from metals and PCBs, residents can safely eat up to five meals of Johnson Creek crayfish each month. Crayfish caught in Johnson Creek by DEQ weighed between 9 and 19 grams. The average weight was 13.3 grams, or approximately one-half ounce. This means that, on average, an eight-ounce crayfish meal would consist of approximately 20 whole crayfish (including shells) or many more crayfish if only meat is consumed. Meal portion size is proportional to body weight and the calculation methods are designed to protect sensitive populations. The recommended limit on crayfish meals that should be consumed by children is the same as for adults. ***EHAP concludes that residents can safely eat up to five meals of Johnson Creek crayfish each month.***

Table 17. Estimated number of crayfish meals that are safe to eat each month based on potential metal and PCB exposures

Basis for fish consumption recommendations	Number of crayfish meals that can be eaten each month without exceeding exposure guidelines[A]
Cumulative risk from all chemicals that target brain development (mercury and PCBs)	5 eight-ounce meals
Cumulative risk from all chemicals that target the immune system (zinc and PCBs)	5 eight-ounce meals
Cumulative risk from all chemicals that target skin (arsenic and PCBs)	5 eight-ounce meals
Cumulative risk from all chemicals that target blood (zinc, chromium and cobalt)	12 eight-ounce meals
Risk from total PCBs[B]	6 eight-ounce meals
Risk from arsenic alone[C]	100 eight-ounce meals
Risk from chromium alone[D]	13.4 eight-ounce meals
Risk from cobalt alone	361 eight-ounce meals
Risk from mercury alone[E]	148 eight-ounce meals
Risk from nickel alone	174 eight-ounce meals
Risk from zinc alone	117 eight-ounce meals

[A] Higher number of meals indicates lower health risks. Meal size is based on adults.

[B] Based on cumulative risk from the sum of all PCB congeners

[C] Assumes that 10% of the arsenic detected is in its more toxic, inorganic form. The consensus in the scientific literature is that approximately 10% of the arsenic typically found in the edible parts of fish and shellfish is inorganic arsenic (39).

[D] Based on the unlikely but health-protective assumption that 100% of chromium detected is in the more toxic, hexavalent form

[E] Based on the health-protective assumption that 100% of mercury detected is in the more toxic, methylmercury form

Analysis of exposure scenarios with insufficient information

Exposure Scenario 5: Long-term residents with exposure to unknown past air concentrations

There is not enough data to support a quantitative evaluation of health effects of historical exposures that occurred before any monitoring was conducted. Emissions reported by PCC to EPA's Toxic Release Inventory (30) indicate that historical emissions for some COCs may have been between 10 and 100 times higher than recent emissions. The presence of additional chemicals, which have since been phased out, would have also contributed to past risk. However, given the limitations and uncertainties of the Toxics Release Inventory, no quantitative conclusions can be drawn. ***EHAP concludes that there is insufficient data to determine whether exposure to historical air emissions near PCC may have harmed health.***

Uncertainties and data gaps

In any public health assessment there are uncertainties and limitations. Calculating and interpreting risk requires the use of assumptions, judgments and limited data sets. This section summarizes potential sources of uncertainty and data gaps and the extent to which they were addressed in this analysis. Estimated risks presented in this PHA should be interpreted in the context of these limitations.

Characterization of toxicity. The health guideline comparison values used to assess toxicity (i.e., MRLs and RfDs) pass through a rigorous scientific peer-review process. However, there is uncertainty in health effects data used to generate these guideline values. For example, health effects of a chemical can vary across species, life stages and individuals in a population. There may also be gaps in the health effects data used to generate health-based comparison values. Typically, these uncertainties are addressed by incorporating a margin of safety into comparison values. To calculate CVs, chemical doses at or below the point where health effects were observed in people or animals are divided by uncertainty factors ranging from 10 to 1,000 to account for remaining uncertainties, sensitive populations and data gaps.

Current CVs may not reflect all the latest evidence or protect against potential health effects that have not yet been well characterized. The chemical-specific comparison values used in this PHA reflect the latest peer-reviewed conclusions of federal scientists and scientific advisory panels based on the weight of evidence from the scientific literature. However, new evidence is continually reshaping our understanding of potential health effects of environmental exposures. For example, in this PHA, non-cancer risk of nickel is evaluated based on an ATSDR chronic MRL derived from studies on respiratory effects in rats. Since the ATSDR MRL was published in 2005, there have been several additional studies finding a correlation between nickel concentrations in air and asthma symptoms in children

(discussed in Appendix F). These studies suggest the potential for nickel to contribute to asthma symptoms at concentrations comparable to what has been detected near PCC. However, these studies alone do not provide conclusive evidence that nickel causes these asthma symptoms and cannot be used to support quantitative health effects analysis in this PHA. Generally, findings from new studies must be replicated and corroborated by other studies with different designs, settings and populations before previously established guidelines or standards can be updated.

Toxicity can also vary with the specific form a chemical takes. In this PHA, there is uncertainty around which specific forms of nickel are present in air. PCC uses nickel alloys that are thought to be less bioavailable and, therefore, less carcinogenic than other forms of nickel (40). However, because monitoring data do not distinguish between the different forms of nickel, we cannot confirm that nickel emitted from PCC remains in an alloy form. We also do not know whether all the nickel present came from PCC. In this PHA we calculate potential health effects based on the health-protective assumption that all nickel detected near the facility may be in the most toxic form.

Risk to sensitive populations. Some groups of people may be particularly sensitive to contaminants of concern identified near PCC. Emerging research has demonstrated that several factors influence our susceptibility to the health effects of environmental exposures. Comparison values are designed to be protective of sensitive populations, but we are not yet able to clearly quantify the role each of these factors plays in influencing risk and how they interact.

- ***Genetic variability.*** Genetic variation may make some individuals particularly susceptible to the health effects of metals. For example, variants in genes involved in processing chemicals mean that some people may be slower to process and excrete chemicals in their bodies than other people (41). Genetic differences can put some people at higher risk of disease, including respiratory disease (42) and cancer (43).
- ***Epigenetic programming.*** Epigenetic factors that influence how genes are turned on and off in our bodies also have an important effect on health and susceptibility (44). Epigenetic gene regulation can be influenced by a range of factors including nutrition, stress, previous chemical exposures and even exposures that occurred during gestation (45) or in previous generations (46).
- ***Sensitive life stages.*** Children, developing fetuses, pregnant women and the elderly may be particularly susceptible to environmental exposures due to differences in how their bodies process and respond to chemicals (47).
- ***Preexisting disease.*** Some people may be more susceptible to the effects of chemical exposure due to preexisting diseases. For example, people with preexisting respiratory conditions such as asthma or chronic obstructive pulmonary disease may be more sensitive to exposures that affect respiratory health (48).

- ***Cumulative chemical exposures.*** Multiple chemicals from a variety of sources at home and at work may act cumulatively to produce the same health outcomes (49) (50).
- ***Social determinants of health.*** Social factors such as poor nutrition and stress may interact with chemical exposures to magnify health effects (25) (26).

Characterization of exposure. There are two main sources of uncertainty in calculating human doses to environmental contaminants based on environmental monitoring data. First, there is uncertainty in environmental monitoring data used to determine the chemical concentrations in air, water and soil with which people may come in contact. Monitoring data may not adequately capture the most contaminated samples or may not include all contaminants that are present. Second, there is uncertainty around the amount of contact people have with contaminated air, water and soil. In this PHA we calculated risk based on health-protective assumptions. We assume that some people may be continuously exposed (24 hours/day) to air concentrations at the upper confidence limit of average monitored air concentrations. We also assume a high frequency of contact with contaminated water, sediment or soil containing the maximum chemical concentrations detected in monitoring efforts.

There is some additional uncertainty around how far air emissions travel and the extent to which they deposit in soil. In this PHA we assume that air monitors located near the facility capture the highest level of emissions because emissions tend to disperse with distance. Dispersion dynamics vary depending on the height of the emissions stack, the temperature of what is emitted and the rate of flow from the stack. Additional emissions modeling that takes these factors into account could better define the geographic area most affected by emissions.

A lack of historical emissions monitoring data means that there is also uncertainty around the extent of historical exposures. This is particularly true of incidents that resulted in short-term elevated emissions. In this PHA we do not calculate risks from historical emissions because there is too much uncertainty around the extent of those exposures. It is possible that high past exposures make some long-term residents more susceptible to ongoing exposures, but there is insufficient information to be able to quantify that effect in this PHA.

Source of the contamination. The air, soil, water and sediment monitoring data used in this PHA determine concentrations of chemicals present in the environment, but they do not identify the source of these chemicals. Other nearby industrial facilities may contribute to total air emissions, and many of the contaminants detected in Johnson Creek may be from upstream sources. This PHA evaluates the potential health effects of all chemicals detected in the environmental monitoring, regardless of source.

Health outcome data

Evaluations of health outcome (i.e., mortality and morbidity) data (HOD) in public health assessments are done using specific guidance in ATSDR's Public Health Assessment Guidance Manual (51). The main requirements for evaluating HOD are the presence of a completed human exposure pathway; high enough contaminant levels to result in measurable health effects; sufficient number of people in the completed pathway for health effects to be measured; and a health outcome database in which disease rates for the population of concern can be identified (51).

This site does not meet the requirements for including an evaluation of HOD in this public health assessment. Although completed human exposure pathways exist at this site, the geographic area and, therefore, the exposed population are not sufficiently defined. In addition, a registry does not exist to track the type of health effects evaluated in the PHA (e.g., respiratory symptoms).

Children's health

EHAP and ATSDR recognize that infants and children may be more vulnerable to exposures than adults in communities faced with contamination of their air, water, soil or food. This vulnerability is a result of the following factors:

- Children's developing body systems can sustain permanent damage if toxic exposures occur during critical growth stages.
- Children are more likely to play outdoors and bring food into contaminated areas.
- Children are shorter, resulting in a greater likelihood to breathe dust, soil and heavy vapors close to the ground.
- Children are smaller and breathe more rapidly, resulting in higher doses of chemical exposure per body weight.
- Children are more likely to swallow or drink water during bathing or when playing in and around water.
- Children are more prone to mouthing objects and eating non-food items such as toys and soil.
- Children's bodies are often different than adults' bodies in their ability to process and remove chemicals to which they are exposed.

Children depend on adults for risk identification and management decisions. The health-based screening values EHAP used for air, soil, water and sediment in this PHA were derived from health guidelines that incorporate a high level of protectiveness for children and other sensitive individuals.

To the extent possible with existing evidence, this PHA considers the special vulnerabilities of children. Children were identified as the most vulnerable to health problems caused by metals in the air and by PCBs and PAHs in Johnson Creek sediment. In each exposure scenario evaluated, EHAP used body weights and ingestion rates that are specific for children at different ages. EHAP also addressed special concerns around childhood exposures to carcinogens. Early childhood exposures to mutagenic carcinogens (those that cause genetic mutations in cells of the body) such as hexavalent chromium and PAHs were given extra weight because those early life exposures may have greater effect on lifetime cancer risks.

Conclusions

Based on currently available science, monitoring data and guidance from federal agencies, EHAP concludes:

Conclusion 1: Measured concentrations of metals in air near PCC are not likely to harm health.

Cumulative exposure to all metals detected in the air around PCC may be predicted to elevate lifetime cancer risk by as many as 20 additional cases of cancer per 1 million people exposed continuously for a lifetime. EHAP considers this to be very low risk. The estimated cancer risk is similar for current conditions and for conditions prior to HEPA filter installation. These risk calculations are based on the cautious assumption that nickel detected in air monitoring is in its most toxic form. It is likely that nickel emissions from PCC are in an alloy form that may be less available to the body and, therefore, less carcinogenic.

Conclusion 2: Measured concentrations of metals in soil from areas around the PCC facility are not likely to harm health.

DEQ sampled soil near the facility, including locations near residences and in community gardens. No soil concentrations exceeded comparison values.

Conclusion 3. Measured concentrations of chemicals in surface water of Johnson Creek are not likely to harm health.

The levels of chemicals detected in surface water are below health-based comparison values designed to be protective of drinking water. TCE was detected at a level slightly above the cancer CV in one sample in 2009 but was not detected in subsequent samples. Johnson Creek, like many urban streams, has had high levels of bacteria that can make people sick. While bacteria in Johnson Creek is not a focus of this PHA and is not believed to be related to PCC, it has the potential to affect public health.

Conclusion 4: Measured concentrations of PCBs and PAHs in the sediment of Johnson Creek near the storm water outfall are not likely to harm health of people who regularly come into contact with it.

Weekly year-round exposure to sediment is not high enough to harm health. While extremely frequent (daily year-round) contact with Johnson Creek sediment could result in a slight increased risk of both non-cancer and cancer health effects, the likelihood of this degree of contact is quite low. Risk calculations were based

on cumulative exposure to maximum concentrations of all PCBs, PAHs and metals of potential concern detected in the creek. Each exposure was assumed to involve full contact of hands, forearms, feet and lower legs with sediment. The biggest health risk from this degree of contact with the creek is the potential for bacterial infections.

Conclusion 5: Residents may safely eat crayfish from Johnson Creek in moderation.

Based on cumulative risk from metals and PCBs, residents can eat up to five meals of Johnson Creek crayfish each month without exceeding health-protective exposure guidelines.

Conclusion 6: There is insufficient information about historical air emissions of metals and solvents at PCC to calculate past health risks.

No historical monitoring data are available to support a quantitative evaluation of potential health effects of previous exposures. Based on historical trends in emissions reported by PCC to EPA's Toxic Release Inventory, we cannot rule out the possibility that past air concentrations could have been high enough to harm health. Emissions reported to TRI since 1987 indicate that emissions of some chemicals may have been 10 and 100 times higher than current emissions during some periods of PCC's past operations. Historical emissions of trichloroethylene and tetrachloroethylene would have also contributed to past risks of cancer and developmental defects.

Recommendations

Based on this analysis of the available information, this report does not identify any levels of exposure that are expected to harm public health and, therefore (in accordance with ATSDR guidance), EHAP does not currently have any recommendations to reduce health risks.

Public health action plan

A public health action plan describes the specific actions EHAP has taken and will take with the goal of preventing and reducing people's exposure to hazardous substances in the environment. EHAP has implemented or will implement the actions listed below in collaboration with community members and partner agencies.

Completed public health actions

Between the spring of 2016 and fall of 2018, EHAP:

- Collaborated with Oregon DEQ on soil sampling plans and placement of air monitors following identification of elevated concentrations of some metals in moss around PCC to ensure that data would be representative of public health
- Convened a community advisory committee to identify the health concerns and help guide the questions addressed in the PHA and met periodically with the committee to provide updates and receive feedback
- Attended and participated in several community meetings organized by DEQ, community advocates and PCC to convey what we knew and didn't know about health risks of air toxics around PCC at the time
- Hosted a webinar to help residents understand when and how different types of public health investigations are used
- Held a public "SoilSHOP" event to screen community members' soil from their gardens and provide guidance on best health practices when gardening in urban areas
- Provided healthy gardening resources to residents concerned about safety of gardening in potentially contaminated soil.

Planned public health actions

In the future, EHAP will:

- Review results of ongoing or future air monitoring by DEQ, Portland State University and PCC to evaluate the potential for health effects
- Continue working with DEQ on the statewide Cleaner Air Oregon effort that aims to implement regulations that ensure that all industrial facility emissions are below levels that may harm public health
- Ensure this public health assessment is made available to all interested community members and stakeholders
- Solicit comments on the draft PHA from community members and stakeholders and update the PHA in response to public comment.

Report preparation

This public health assessment was prepared by the Oregon Environmental Health Assessment Program (EHAP) under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies and procedures existing at the date of publication. The document was reviewed by Oregon DEQ partners.

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Endnotes

1. Fortune 500. Fortune 500: 282 Precision Castparts [Internet]. Fortune 500; 2016 [cited 2018 Oct 12]. Available from: http://fortune.com/fortune500/2016/precision-castparts/.

2. Precision Castparts (PCC). Precision Castparts Corp. [Internet]. Precision Castparts Corps.; 2018 [cited 2018 Oct 12]. Available from: http://www.precast.com/.

3. Precision Castparts (PCC). Berkshire Hathaway completes acquisition of Precision Castparts [Internet]. Precisions Castparts Corp.; 2016 [cited 2018 Oct 12]. Available from: http://precast.com/investors/press_release/?id=673.

4. Precision Castparts (PCC). Our history [Internet]. Precisions Castparts Corp.; 2018 [cited 2018 Oct 12]. Available from: http://www.pccstructurals.com/about-us/history/.

5. Oregon Department of Environmental Quality (DEQ). Standard air contaminant discharge permit [Internet]. Oregon Department of Environmental Quality; 2007 [cited 2018 Oct 12]. Available from: https://www.oregon.gov/deq/FilterDocs/PCCAirPermit.pdf.

6. Oregon Department of Environmental Quality (DEQ). Site summary full report — details from site ID 274, PCC Structurals, Inc. [Internet]. Oregon Department of Environmental Quality Environmental Cleanup Site Information Database; 2018 [cited 2018 Oct 12]. Available from: http://www.deq.state.or.us/lq/ECSI/ecsidetailfull.asp?seqnbr=274#actions.

7. Occupational Safety and Health Administration. PCC Structurals establishment search results. [Internet]. Occupational Safety and Health Administration; 2018 [cited 2017 March 10]. Available from: https://www.osha.gov/pls/imis/establishment.search?p_logger=1&establishment=PCC+structurals&State=OR&officetype=all&Office=all&p_case=all&p_violations_exist=all&startmonth=01&startday=01&startyear=1972&endmonth=03&endday=10&endyear=2017.

8. US Environmental Protection Agency (EPA). National air toxics assessment [Internet]. US Environmental Protection Agency; 2017 [cited 2018 June 5]. Available from: https://www.epa.gov/national-air-toxics-assessment.

9. US Nuclear Regulaotry Commission. PCC Structurals, Inc. (State of Oregon) [Internet]. US Nuclear Regulatory Commission facility locator; 2018 [cited 2018 Oct 12]. Available from: https://www.nrc.gov/info-finder/decommissioning/complex/pcc-structurals-inc.html.

10. Oregon Department of Environmental Quality. Voluntary agreement for remedial investigation/feasibility study and IRAM DEQ NO. LQVC-NWR-04-14 [Internet]. Oregon Department of Environmental Quality; 2018 [cited 2018 March 14]. Available from: http://www.oregon.gov/deq/FilterDocs/PCC-volagreement.pdf.

11. Oregon Department of Environmental Quality (DEQ). Portland air toxics solutions (PATS) [Internet]. Oregon Department of Environmental Quality; 2009 [cited 2018 Oct 12]. Available from: https://www.oregon.gov/deq/aq/air-toxics/Pages/PATS.aspx.

12. US Forest Service (USFS). The Portland moss and air quality study [Internet]. US Forest Service Pacific Northwest Research Station; 2017 [cited 2017 June 5]. Available from: https://www.fs.fed.us/pnw/research/moss/.

13. Gatziolis D, Jovan S, Donovan G, Amacher M, Monleon V. Elemental atmospheric pollution assessment via moss-based measurements in Portland, Oregon [Internet]. US Department of Agriculature; 2016 [cited 2018 Oct 12]. Available from: https://www.fs.fed.us/pnw/pubs/pnw_gtr938.pdf.

14. US Forest Service (USFS). Interactive map of moss-derived element concentrations in Portland, Oregon [Internet]. US Forest Service; 2016 [cited 2017 June 5]. Available from: https://usfs.maps.arcgis.com/apps/webappviewer/index.html?id=14766acdb73e4eb194ba3ada0ce8539d.

15. Oregon Department of Environmental Quality (DEQ). Cleaner Air Oregon website [Internet]. Oregon Department of Environmental Quality; 2017 [cited 2017 June 5]. Available from: http://cleanerair.oregon.gov/.

16. Oregon Department of Environmental Quality (DEQ). Precision Castparts area-wide air sampling and analysis plan [Internet]. Oregon Department of Environmental Quality; 2016 [cited 2017 June 5]. Available from: http://www.deq.state.or.us/nwr/docs/metalsem/sap17.pdf.

17. Oregon Department of Environmental Quality (DEQ). Precision Cast Parts area-wide soil sampling report [Internet]. Oregon Department of Environmental Quality; 2016 [cited 2017 June 5]. Available from: http://www.deq.state.or.us/nwr/docs/metalsem/PCCSoilReport082416.pdf.

18. Read, R. Portland's Precision Castparts ranked nation's top industrial air polluter in University of Massachusetts study [Internet]. The Oregonian/OregonLive; 2013 Aug 26 [cited 2018 Oct 12]. Available from: https://www.oregonlive.com/business/index.ssf/2013/08/portlands_precision_castparts_2.html.

19. Political Economny Research Institute. Toxic 100 air polluters index: 2013 report, based on 2010 data [Internet]. University of Massachusetts Amherst; 2013 [cited 2018 Oct 12]. Available from: https://www.peri.umass.edu/toxic-100-air-polluters-index-2018-report-based-on-2015-data.

20. City of Portland. Errol Heights Community Garden [Internet]. City of Portland Parks and Recreation; 2017 [cited 2017 June 5]. Available from: https://www.portlandoregon.gov/parks/finder/index.cfm?action=ViewPark&PropertyID=1419.

21. Johnson Creek Watershed Council. Restoration projects –– Errol Heights and Wetlands [Internet]. Johnson Creek Watershed Council 2017 [cited 2017 June 5]. Available from: http://jcwc.conservationregistry.org/projects/15308#fulldescr.

22. City of Portland. Commissioner Fritz announces funding for PP&Rs Errol Heights Park enhancement [Internet]. City of Portland Parks and Recreation; 2016 Dec 2 [cited 2017 June 5]. Available from: https://www.portlandoregon.gov/parks/article/619707.

23. Institute of Medicine (US) Committee on Environmental Justice. Toward environmental justice: research, education, and health policy needs [Internet]. Washington, DC: National Academy Press; 1999 [cited 2018 Oct 12]. Available from: http://www.nationalacademies.org/hmd/Reports/1999/Toward-Environmental-Justice-Research-Education-and-Health-Policy-Needs.aspx.

24. Kruize H, Droomers M, van Kamp I, Ruijsbroek A. What causes environmental inequalities and related health effects? An analysis of evolving concepts. International Journal of Environmental Research and Public Health. 2014;11(6):5807–5827. Available from: doi:10.3390/ijerph110605807.

25. Cooney, CM. Stress-pollution interactions: an emerging issue in children's health research. Environmental Health Perspectives. 2011;119(10):A431-5. Available from: doi:10.1289/ehp.119-a430.

26. World Health Organization. A conceptual framework for action on the social determinants of health [Internet]. World Health Organization; 2010 [cited 2018 Oct 12]. Available from: http://www.who.int/sdhconference/resources/ConceptualframeworkforactiononSDH_eng.pdf.

27. US Environmental Protection Agency (EPA). EJScreen: Environmental justice screening and mapping tool [Internet]. Environmental Protection Agency; 2017 [cited 2017 June 5]. Available from: https://www.epa.gov/ejscreen.

28. Oregon Department of Environmental Quality (DEQ). Precision Castparts area air monitoring data [Internet]. Oregon Department of Environmental Quality; 2016 [cited 2018 Oct 12]. Available from: https://www.oregon.gov/deq/Programs/Pages/Precision-Castparts-Corp.aspx.

29. Mulvey, Michael. Analytical report, Toxic Monitoring Program –– tissue, Willamette and Northeast Oregon sampling event: 1702017. Oregon Department of Environmental Quality Laboratory and Environmental Assessment Program; 2018.

30. US Environmental Protection Agency (EPA). Toxics release inventory facility report: PCC Structurals Inc Large Parts Campus (97206LRGST4600S) [Internet]. US Environmental Protection Agency; 2017 [cited 2017 June 5]. Available from: https://www3.epa.gov/enviro/facts/tri/ef-facilities/#/Release/97206LRGST4600S.

31. US Environmental Protection Agency (EPA). Factors to consider when using toxics release inventory data [Internet]. US Environmental Protection Agency; 2015 [cited 2017 June 5]. Available from: https://www.epa.gov/sites/production/files/2015-06/documents/factors_to_consider_6.15.15_final.pdf.

32. City of Milwaukie. Water quality reports [Internet]. City of Milwaukie Public Works, Water Division; 2017 [cited 2017 June 5]. Available from: https://www.milwaukieoregon.gov/publicworks/water-quality-reports.

33. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for chromium [Internet]. Agency for Toxic Substances and Disease Registry; 2012 [cited 2018 Oct 12]. Available from: http://www.atsdr.cdc.gov/toxprofiles/TP.asp?id=62&tid=17.

34. Johnson Creek Watershed Council. Johnson Creek water quality 2009–2014 [Internet]. Johnson Creek Watershed Council; 2015 [cited 2018 March 23]. Available from: http://www.jcwc.org/wp-content/uploads/2017/07/Johnson-Creek-Water-Quality-2009-to-2014-FINAL-lo-res.pdf.

35. Oregon Department of Environmental Quality (DEQ). Lower Willamette Subbasin: DEQ's water quality status and trends analysis for the Oregon Department of Agriculture's biennial review of the agricultural area rules and plans [Internet]. Oregon Department of Environmental Quality; 2017 [cited 2018 Oct 12]. Available from: http://www.oregon.gov/deq/FilterDocs/LWillstatusTrends.pdf.

36. American Cancer Society. Lifetime probability of developing or dying from cancer [Internet]. American Cancer Society; 2016 [cited 2018 Oct 12]. Available from: http://www.cancer.org/cancer/cancerbasics/lifetime-probability-of-developing-or-dying-from-cancer.

37. U Environmental Protection Agency (EPA). Exposure factors handbook 2011 edition (final report). Washington, DC: US Environmental Protection Agency; 2011. EPA/600/R-09/052F.

38. Oregon Health Authority (OHA). Fish consumption advisory standard operating guidance (SOG), Oregon Health Authority Fish Advisory Program [Internet]. Oregon Health Authority; 2018 [cited 2018 Oct 12]. Available from: http://www.oregon.gov/oha/PH/HEALTHYENVIRONMENTS/RECREATION/FISHCONSUMPTION/Documents/Fish-Consumption-Advisory-SOG.pdf.

39. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for arsenic [Internet]. Agency for Toxic Substances and Disease Registry; 2007 [cited 2018 Oct 12]. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=22&tid=3.

40. Goodman JE, Prueitt RL, Thakali S, Oller AR. The nickel ion bioavailability model of the carcinogenic potential of nickel-containing substances in the lung. Critical Reviews in Toxicology. 2011;41(2):142-74. Available from: doi: 10.3109/10408444.2010.531460.

41. Meyer UA, Gut J. Genomics and the prediction of xenobiotic toxicity. Toxicology. 2002;181–182:463–6.

42. Martinez FD. Genes, environments, development, and asthma: a reappraisal. 2007, European Respiratory Journal. 2007;29(1):179–84. Available from: doi: 10.1183/09031936.00087906.

43. Perez-Losada J, Castellanos-Martin A, Mao, JH. Cancer evolution and individual susceptibility. Integrated Biology (Camb). 2011;3(4);316–328. Available from: doi: 10.1039/c0ib00094a.

44. Baccarelli A, Bollati V. Epigenetics and environmental chemicals. Current Opinion in Pediatrics. 2009;21(2):243–51.

45. Barouki, R, Gluckman PD, Grandjean P, Hanson M, Heindel JJ. Developmental origins of non-communicable disease: Implications for research and public health. Environmental Health. 2012;11:42.

46. Skinner M, Geurrero-Bosagna C. Environmental signals and transgenerational epigenetics. Epigenomics. 2009;1(1): 111–117.

47. Klaassen, C. Casarett & Doull's toxicology. 8th ed. New York: McGraw-Hill Education/Medical; 2013.

48. Lagorio S, Forastiere F, Pistelli R, Iavarone I, Michelozzi P, Fano V, et al. Air pollution and lung function among susceptible adult subjects: a panel study. Environmental Health. 2006;5:11. Available from: doi: 10.1186/1476-069X-5-11.

49. Solomon G, Morello-Frosch R, Zeise L, Faust JB. Cumulative environmental impacts: science and policy to protect communities. Annual Review of Public Health. 2016 Mar 18;37:83–96.

50. Goodson III WH, Lowe L, Carpenter DO, Gilbertson M, Manaf Ali A, Lopez de Cerain Salsamendi A, et al. Assessing the carcinogenic potential of low-dose exposures to chemical mixtures in the environment: the challenge ahead. Carcinogenesis. 2015 Jun 1;36(Suppl_1):S254–96.

51. Agency for Toxic Substances and Disease Registry (ATSDR). Public health assessment guidance manual (Update) [Internet]. Agency for Toxic Substances and Disease Registry; 2005 [cited 2018 Oct 12]. Available from: http://www.atsdr.cdc.gov/HAC/PHAmanual.

52. Northwest Center for Public Health Practice. Effective adult learning: a toolkit for teaching adults [Internet]. Seattle : School of Public Health, University of Washington in partnership with the Network for Public Health Law; 2012 [cited 2018 Oct 12]. Available from: http://www.nwcphp.org/training/opportunities/toolkits-guides/effective-adult-learning-a-toolkit-for-teaching-adults.

53. Agency for Toxic Substances and Disease Registry (ATSDR). Public health assessment guidance manual [Internet]. Agency for Toxic Substances and Disease Registry; 2005 [cited 2018 Oct 12]. Available from: https://www.atsdr.cdc.gov/hac/phamanual/toc.html.

54. Precision Castparts Corp. (PCC). Frequently asked questions [Internet]. Precision Castparts Corp.; 2018 [cited 2018 Oct 12]. Available from: http://pccstructuralscommunity.com/faq.html.

55. Maul Foster Alongi. Storm water pollution control plan, prepared for PCC Structurals, Inc Large Parts Campus. DEQ permit file No.: 100481. Maul Foster Alongi; 2017.

56. US Environmental Protection Agency (EPA). Development of a relative potency factor (RPF) approach for polycyclic aromatic hydrocarbons (PAH) mixtures. Washington, DC: US Environmental Protection Agency; 2010. EPA/635/R-08/012A [cited 2018 Oct 12]. Available from: https://cfpub.epa.gov/si/si_public_record_report.cfm?Lab=NCEA&dirEntryId=194584.

57. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for cadmium [Internet]. Agency for Toxic Substances and Disease Registry; 2012 [cited 2018 Oct. 12]. Available from: https://www.atsdr.cdc.gov/toxprofiles/TP.asp?id=48&tid=15.

58. Larsson SC, Orsini N, Wolk, A. Urinary cadmium concentration and risk of breast cancer: a systematic review and dose-response meta analysis. American Journal of Epidemiology. 2015;182:375–80.

59. Wei Z, Song X, Shaikh ZA. Cadmium promotes the proliferation of triple-negative breast cancer cells through EGFR-mediated cell cycle regulation. Toxicology and Applied Pharmacology. 2015;289:98:108.

60. Siewit CL, Gengler B, Vegas E, Puckett R, Louie MC. Cadmium promotes breast cancer cell proliferation by potentiating the interaction between ER· and c-Jun. Molecular Endocrinology. 2010 May 1;24(5):981–92.

61. Wang Y, Chen L, Gao Y, Zhang Y, Wang C, Zhou Y, et al. Effects of prenatal exposure to cadmium on neurodevelopment of infants in Shandong, China. Environmental Pollution. 2016 Apr 1;211:67–73.

62. Kippler M, Bottai M, Georgiou V, Koutra K, Chalkiadaki G, Kampouri M, et al. Impact of prenatal exposure to cadmium on cognitive development at preschool age and the importance of selenium and iodine. European Journal of Epidemiology. 2016 Nov 1;31(11):1123–34.

63. Kim Y, Ha EH, Park H, Ha M, Kim Y, Hong YC, et al. Prenatal lead and cadmium co-exposure and infant neurodevelopment at 6 months of age: the Mothers and Children's Environmental Health (MOCEH) study. Neurotoxicology. 2013 Mar 1;35:15–22.

64. Navy and Marine Corps Public Health Center (NMCPHC). Risk characterization for carcinogens that have a mutagenic mode of action, supplemental Navy guidance for conducting human health risk assessments [Internet]. Navy and Marine Corps Public Health Center; 2008 [cited 2018 Oct 12]. Available from: http://www.med.navy.mil/sites/nmcphc/Documents/environmental-programs/risk-assessment/Risk_Characterization_for_Chemicals_with_Mutagenic_MOA_Feb_2008.pdf.

65. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for nickel [Internet]. Agency for Toxic Substances and Disease Registry; 2005 [cited 2018 Oct 12]. Available from: https://www.atsdr.cdc.gov/ToxProfiles/tp15.pdf.

66. Korten I, Ramsey K, Latzin P. Air pollution during pregnancy and lung development in the child. Paediatric respiratory reviews. 2017 Jan 1;21:38–46.

67. Goldizen FC, Sly PD, Knibbs LD. Respiratory effects of air pollution on children. Pediatric Pulmonology. 2016 Jan;51(1):94–108.

68. Patel MM, Hoepner L, Garfinkel R, Chillrud S, Reyes A, Quinn JW, et al. Ambient metals, elemental carbon, and wheeze and cough in New York City children through 24 months of age. American Journal of Respiratory and Critical Care Medicine. 2009 Dec 1;180(11):1107–13.

69. Rosa MJ, Benedetti C, Peli M, Donna F, Nazzaro M, Fedrighi C, et al. Association between personal exposure to ambient metals and respiratory disease in Italian adolescents: a cross-sectional study. BMC pulmonary medicine. 2016 Dec;16(1):6.

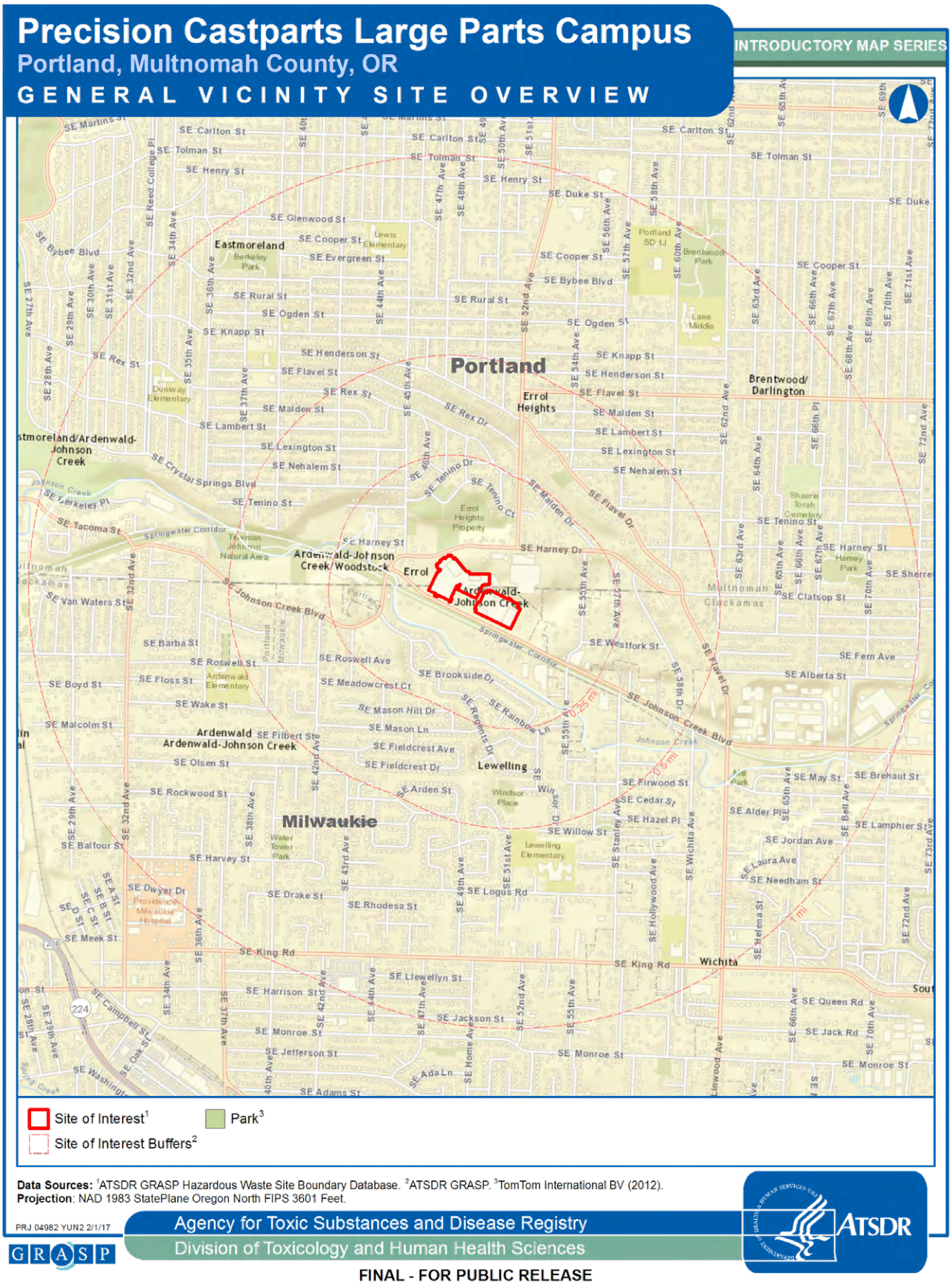
71. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for polycyclic aromatic hydrocarbons [Internet]. Agency for Toxic Substances and Disease Registry August; 1995 [cited 2018 Oct 12]. Available from: https://www.atsdr.cdc.gov/toxprofiles/tp69.pdf.

72. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for polychlorinated biphenyls (PCBs) [Internet]. Agency for Toxic Substances and Disease Registry; 2000 [cited 2018 Oct 12]. Available from: https://www.atsdr.cdc.gov/toxprofiles/tp17.pdf.

73. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment Air Toxicology and Epidemiology Branch. Technical support document for cancer potency factors: methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures [Internet]. California Environmental Protection Agency; 2009 [cited 2018 Oct 12]. Available from: http://oehha.ca.gov/air/hot_spots/2009/TSDCancerPotency.pdf.

74. Skol AD, Sasaki MM, Onel K. The genetics of breast cancer risk in the post-genome era: thoughts on study design to move past BRCA and towards clinical relevance. Breast Cancer Research. 2016 Dec;18(1):99.

Appendix A. Area maps

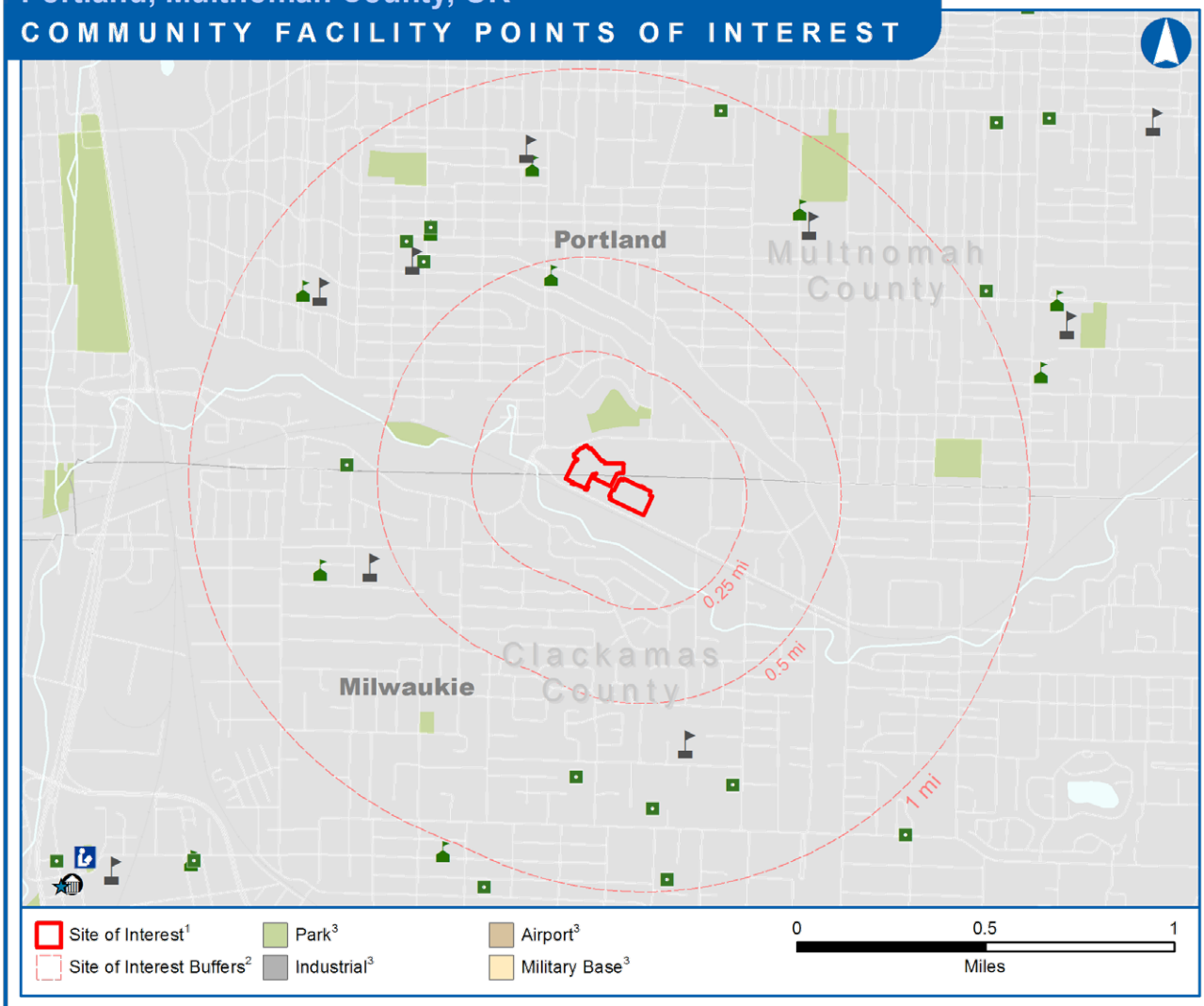


Precision Castparts Large Parts Campus

Portland, Multnomah County, OR

COMMUNITY FACILITY POINTS OF INTEREST

INTRODUCTORY MAP SERIES



The **Community Facility Points of Interest Map** depicts the site of interest and community gathering centers in the local area. Information on number, type, and distribution of these facilities is important to efforts to communicate findings to the local population.

Within a **5-mile buffer** of this site are located **702** community gathering centers of which **37%** are designated as places of worship.

Community Facility Points of Interest

Within specified distance of site boundary. Not all buffers may be shown on map

Facility	0.25 mile	0.5 mile	1 mile	3 miles	5 miles
Libraries[3]	0	0	0	6	17
Schools[4]	0	0	6	57	147
Colleges/Universities[4]	0	0	0	3	16
City Halls[5]	0	0	0	1	5
Civic Centers[3]	0	0	0	3	25
Court Houses[3]	0	0	0	1	8
Places of Worship[3]	0	0	9	101	265
Day Care Centers[4]	0	1	6	37	128
Parks[3]	1	1	4	31	91

Data Sources: [1]ATSDR GRASP Hazardous Waste Site Boundary Database. [2]ATSDR GRASP. [3]TomTom International BV (2015). [4]Homeland Security Infrastructure Program 2015. [5]NAVTEQ 2012 Q1 data
Projection: NAD 1983 StatePlane Oregon North FIPS 3601 Feet.

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GRASP

Agency for Toxic Substances and Disease Registry
Division of Toxicology and Human Health Sciences

ATSDR

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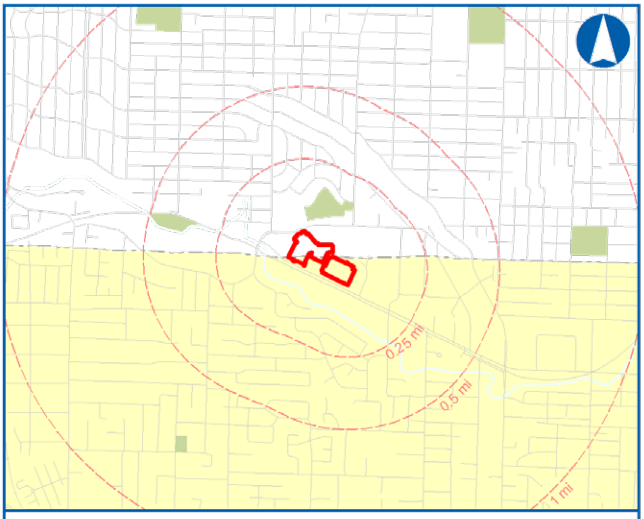
Precision Castparts Large Parts Campus

Portland, Multnomah County, OR

DEMOGRAPHIC SITE PROFILE

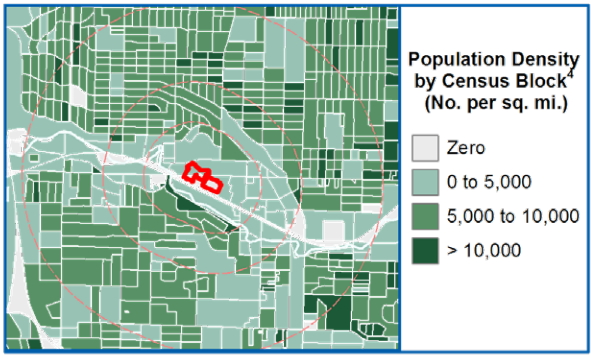
INTRODUCTORY MAP SERIES

Site Vicinity Map

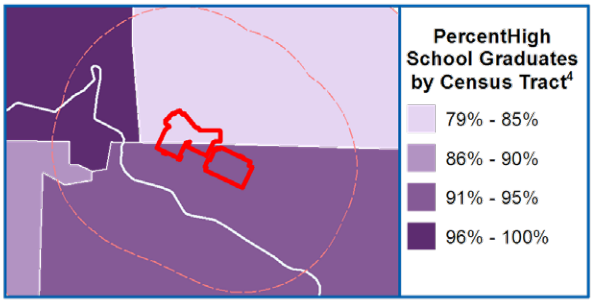


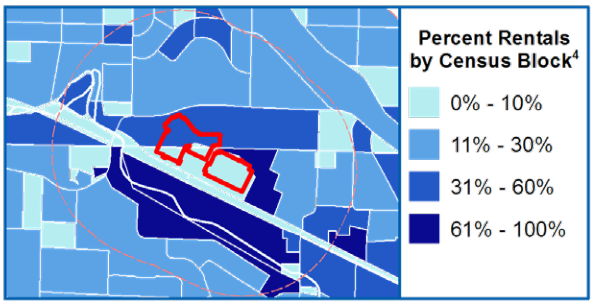
The **General Site Profile Map** depicts the hazardous waste site of interest, along with any airport, industrial, military, or park land uses. It also provides community demographic and housing statistics.

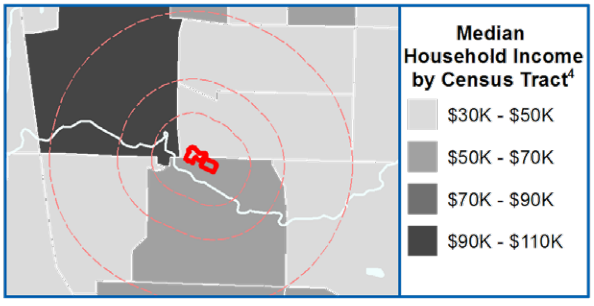
General Population Density



Demographic Indicators







Demographic Statistics[4,5]

Within 0.5 Miles buffer of site boundary, in Portland, and in Oregon

Measure	0.5 miles	Portland	Oregon
Total Population	5,167	566,686	3,939,233
White Alone	86.6%	72.9%	77.2%
Black Alone	1.3%	6.2%	1.8%
Am. Indian & Alaska Native	1.5%	0.7%	0.9%
Asian Alone	3.5%	7.1%	3.9%
Native Hawaiian & Other Pacific Islander Alone	0.3%	0.6%	0.4%
Some Other Race Alone	2.4%	0.2%	0.1%
Two or More Races	4.4%	3.6%	3.3%
Hispanic or Latino[6]	7.6%	8.8%	12.3%
% High School Graduate	88.2%	88.9%	88.6%
% Bachelor's Degree	26.4%	42.2%	28.6%
% Renters	17.9%	43.6%	37.8%
Median Household Income	$55,284	$55,003	$49,260

Data Sources: [1]ATSDR GRASP Hazardous Waste Site Boundary Database. [2]ATSDR GRASP. [3]TomTom International BV (2012). [4]US Census 2010. **Notes:** [5]Calculated using area-proportion spatial analysis method. [6]Individuals identifying origin as Hispanic or Latino may be of any race.

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GRASP

Agency for Toxic Substances and Disease Registry

Division of Toxicology and Human Health Sciences

ATSDR

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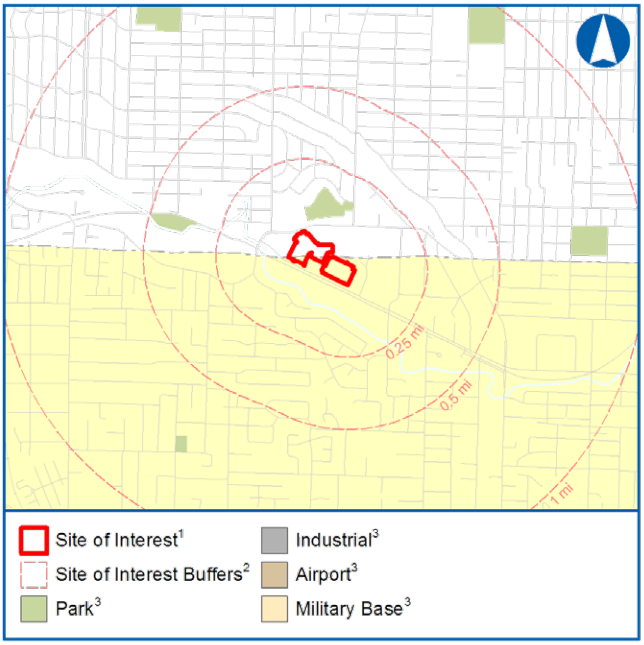
Precision Castparts Large Parts Campus

Portland, Multnomah County, OR

POPULATION SITE PROFILE

INTRODUCTORY MAP SERIES

Site Vicinity Map



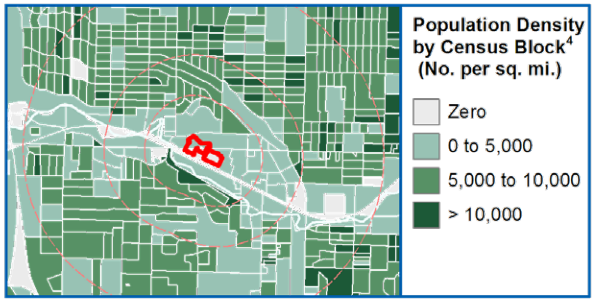
The **General Site Profile Map** depicts the hazardous waste site of interest, along with any airport, industrial, military, or park land uses. It also provides community demographic and housing statistics.

Demographic Statistics[4,5]

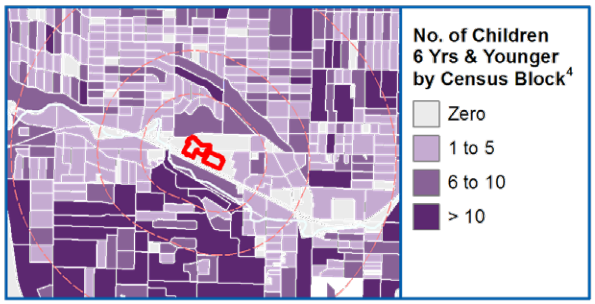
Within 0.5 Miles buffer of site boundary

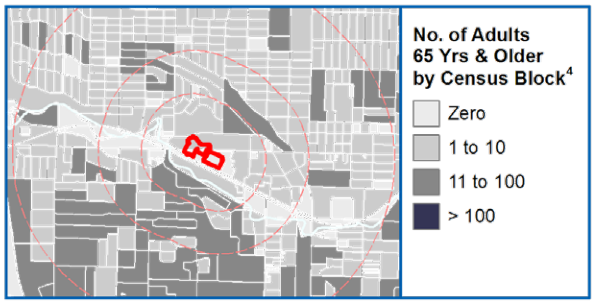
Measure	2000	2010	Change
Total Population	5,082	5,167	+1%
White Alone	4,503	4,476	+0%
Black Alone	36	67	+86%
Am. Indian & Alaska Native	67	77	+14%
Asian Alone	235	182	-22%
Native Hawaiian & Other Pacific Islander Alone	16	14	-12%
Some Other Race Alone	81	123	+51%
Two or More Races	140	227	+62%
Hispanic or Latino[6]	184	393	+113%
Children Aged 6 and Younger	479	433	-9%
Adults Aged 65 and Older	592	561	-5%
Females Aged 15 to 44	1,088	1,146	+5%
Housing Units	2,093	2,144	+2%
Housing Units Pre 1950	1,734	929	-46%

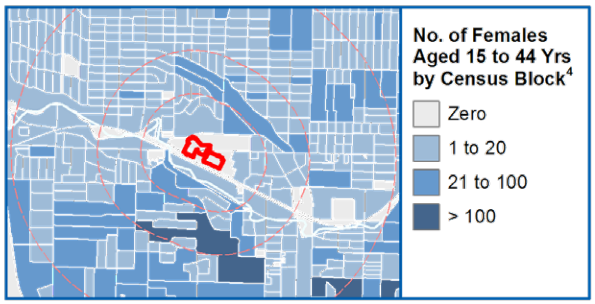
General Population Density



Sensitive Populations







Data Sources: [1]ATSDR GRASP Hazardous Waste Site Boundary Database. [2]ATSDR GRASP. [3]TomTom International BV (2012). [4]US Census 2010. **Notes:** [5]Calculated using area-proportion spatial analysis method. [6]Individuals identifying origin as Hispanic or Latino may be of any race.

PRJ 04982 YUN2 2/1/17

GRASP

Agency for Toxic Substances and Disease Registry

Division of Toxicology and Human Health Sciences

ATSDR

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Appendix B. DEQ monitoring locations

Figure B1. Map of DEQ air monitoring locations (courtesy of DEQ). Locations of three metal particulate monitors are labeled MJC, PFH and PHD. MJF is the meteorological monitoring location. Monitoring details available in the sampling and analysis plan (16).

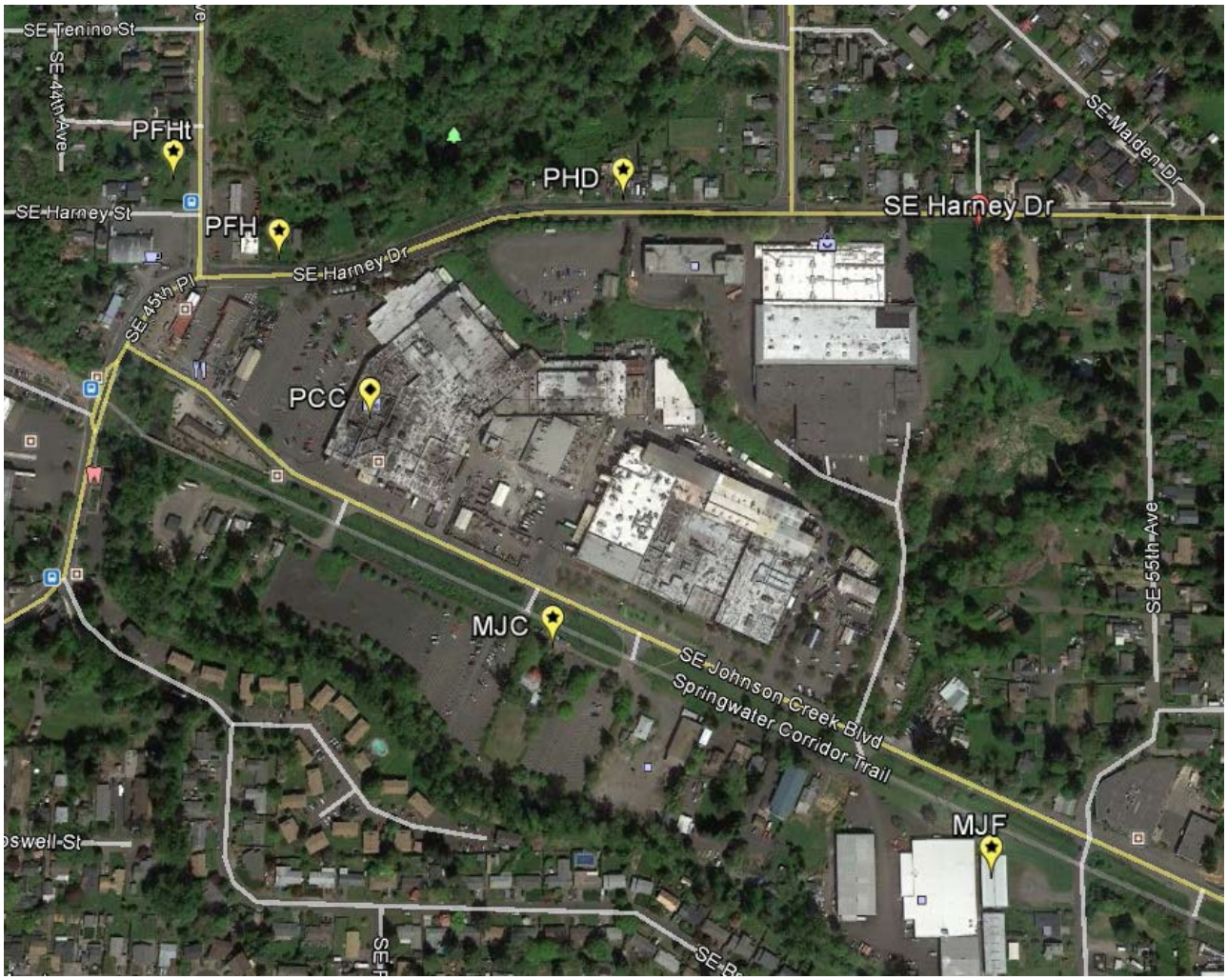
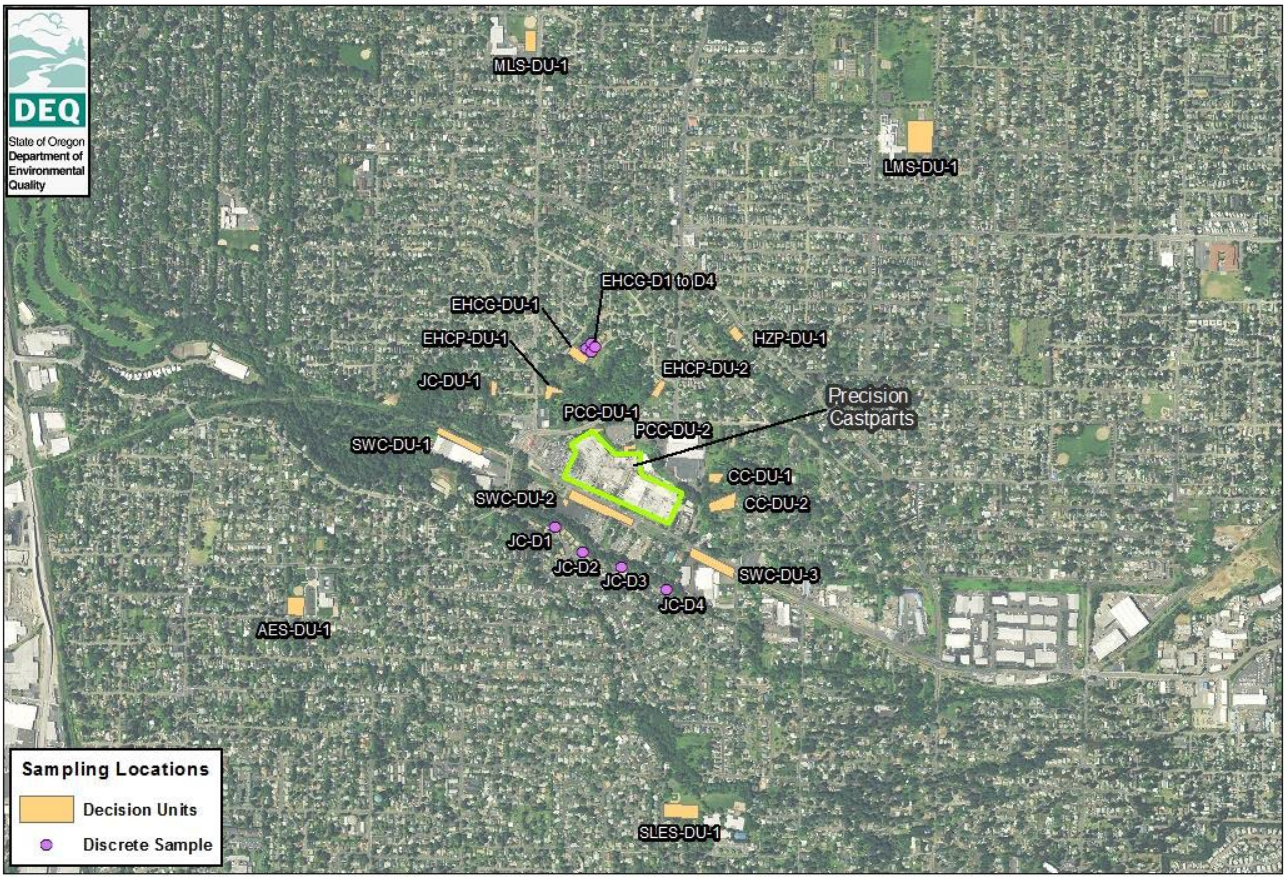


Figure B2. Map of DEQ soil sampling locations (courtesy of DEQ). Details of sampling and analysis methods available in the soil sampling report (17).



Appendix C. Moss sampling results near PCC

Figure C1. Map of approximate US Forest Service moss sampling locations (screenshot from https://usfs.maps.arcgis.com/apps/webappviewer/index.html?id=14766acdb73e4eb194ba3ada0ce8539d).

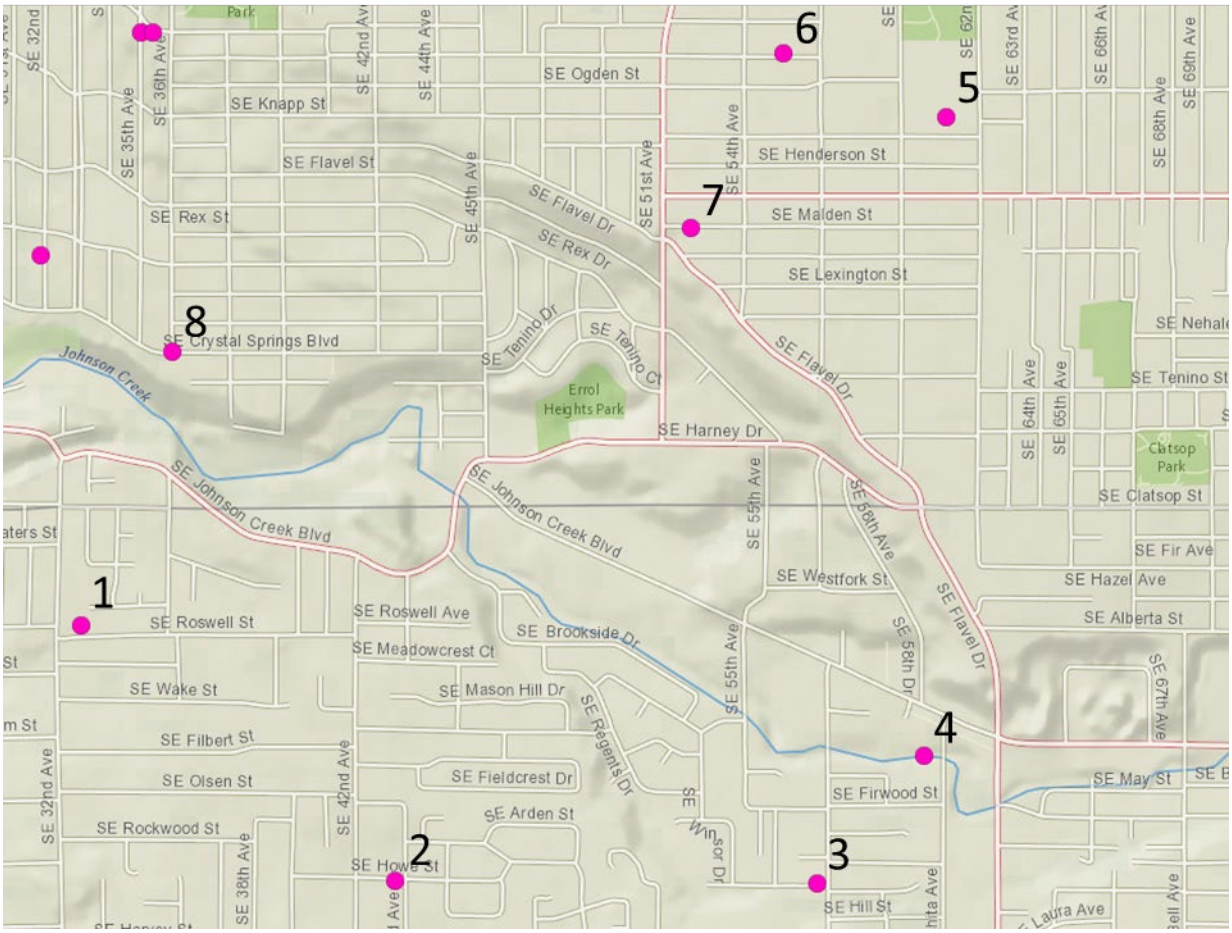


Table C1. Percent rank of moss concentrations for selected metals detected near PCC in comparison with concentrations at all other Portland moss sampling locations Percent ranks closer to 100 indicate higher concentrations relative to moss tested at other locations in Portland

Approximate Location	Nickel	Chromium	Cobalt	Arsenic	Lead
1. 32nd and Roswell	94%	56%	53%	<1%	24%
2. 43rd and Howe	98%	51%	66%	ND	16%
3. SE Stanley	99%	72%	84%	ND	13%
4. SE Wichita Ave	95%	37%	81%	ND	12%
5. SE Knapp and 62nd	96%	62%	82%	96%	62%
6. SE Rural and 57th	100%	88%	95%	90%	47%
7. SE Malden and 52nd	100%	99%	100%	99%	67%
8. Crystal Springs and 36th	95%	85%	91%	24%	68%

ND - Not Detected

Appendix D. Community involvement in the PHA

Community participation helped identify public health concerns, define the scope of the PHA, check assumptions used in risk calculations, and provide guidance on communication strategies for reaching the broader public. EHAP has taken the following steps to ensure meaningful community involvement throughout the PHA process:

Convened a community advisory committee (CAC).

- **Recruitment and composition:** EHAP prioritized residents living in close proximity to the site (within 0.5-mile radius) and populations most sensitive and vulnerable to the effects of exposure to air emissions of metals. EHAP:
    a. Created targeted CAC recruitment materials,
    b. Visited several community locations as part of an in-person outreach strategy, including: Roswell Market, 52nd Coin Laundry, Sparkles Laundromat, Impact NW at the Brentwood Darlington Community Center, Wichita Feed Store, Johnson Creek Market, Brookside Apartments, Brentwood Community Gardens, Lane Middle School and Ardenwald School,
    c. Issued a press-release announcing the CAC recruitment,
    d. Recruited 13 CAC members representing diverse perspectives, including parents of young children, long-time residents of the neighborhood, residents with autoimmune and chronic health conditions, gardeners, and small business owners.
- **CAC meeting logistics:** EHAP convened three formal CAC meetings. To remove barriers for participation, EHAP held meetings outside of daytime work hours at a neighborhood location, served food for participants, and allowed children. Meetings were held in the evening over the span of dinner mealtime hours (from 6:00 PM to 8:00 PM). EHAP leveraged resources beyond the Agency for Toxic Substances and Disease Registry (ATSDR) Cooperative Agreement to provide food at every meeting. EHAP was not able to provide childcare at meetings. EHAP did not translate materials or directly target non-English speaking residents due to the limitations imposed by a tight timeline, funding, and staff constraints.
- **CAC meeting content:** The CAC meetings were structured to provide the opportunity for meaningful participation[1]. EHAP used evidence-based strategies for effective presentations and adult education (52). The content and training explained the PHA process. The presentations, interactive activities, handouts and visual displays were informed by learning objectives with the goal of increasing participants' understanding of the PHA process. This allowed the PHA-CAC members to make

[1] "Meaningful participation" means engaging a diverse group of stakeholders who are representative of the communities that policies and programs will affect, not only in consultative roles to provide input, but also to co-plan or lead program development efforts, have access to data and resources to make informed decisions, have decision-making authority, and participate in the analysis of data and program effect efforts.

informed decisions when advising EHAP on specific elements of the PHA process. Every meeting included time for community advisors to make suggestions, ask questions, and share concerns. EHAP compiled list of CAC concerns, questions and advice and provided responses with resources. This information is summarized within the "Community concerns" section of this PHA.

- **Ongoing dialogue with CAC members**: Informal meetings and conversations have continued with some CAC members who have requested additional information. In addition, EHAP has kept CAC members apprised of timeline changes, PHA updates, opportunities for additional input, and other relevant events (webinars, workshops, etc.).

Attended and Participated in Public Forums. Alongside local partners, EHAP participated in several public forums to learn more about community concerns and to communicate about the PHA process. These public meetings ranged in attendance from 30-200 people and occurred in 2016 and 2017.

***Provided online communication*.** To keep the public informed, EHAP created a webpage for the PCC Larger Parts Campus PHA at www.healthoregon.org/ehap. The page links to relevant documents, other PHAs and contact information for EHAP. It will continue to be updated as needed.

Responded to phone and email contact. EHAP had direct phone and email contact with several individuals through a dedicated phone line, personal contact with EHAP staff, and the EHAP program e-mail.

Community concerns

ATSDR developed its PHA protocols specifically to address community concerns related to environmental health. OHA follows these protocols under the terms of its ATSDR cooperative funding agreement that funds OHA's Environmental Health Assessment Program. Through the PCC Community Advisory Committee (CAC), public forums, and phone and email communication with individuals, EHAP identified a set of environmental health concerns shared by community members. These concerns and responses from EHAP are summarized below.

Specific Exposure Scenarios

Community members wanted to understand risks associated with several specific exposure scenarios, including breathing the neighborhood air, gardening and eating local produce, playing in Johnson Creek, and children's exposures at nearby schools and daycares. Community members also asked EHAP to consider the effects to volunteer workers in the park and creek.

This PHA evaluates the potential health risks of contact with air, water, soil, and sediment measured around the PCC facility. To evaluate risk, EHAP used 'worst-case' scenario assumptions about the frequency and intensity of exposure.

To evaluate risk of exposure to emissions currently in the air, EHAP assumed neighbors of PCC are exposed to concentrations detected immediately surrounding the facility, 24 hours a day for a lifetime. Cancer risk of all air contaminants was evaluated cumulatively. Using these health-protective assumptions, EHAP concluded that current air emissions are not expected to harm health. Because air emissions generally decrease with distance from the source, this also means that there is little risk expected from air at homes, schools, and parks farther away from PCC.

To evaluate risk from contact with contaminants in Johnson Creek sediment, EHAP assumed weekly year-round contact (or 4 times a week in the summer months only) that resulted in sediment containing the maximum chemical concentrations detected at any point in monitoring covering lower legs and feet, hands, and forearms. Using these health-protective assumptions, EHAP concluded that the occasional contact with chemical contaminants in Johnson creek water and sediment that occurs during recreation and volunteering is not expected to pose a health risk. EHAP also considered an extreme exposure scenario assuming daily contact with sediment year-round. This extreme exposure scenario slightly increased lifetime cancer risk and non-cancer effects of nickel exposure, but EHAP is not aware of any individuals that come in contact with the Creek frequently enough for this to be a public health concern. Concentrations of contaminants detected in Johnson Creek surface water were below comparison values for water and are therefore not expected to harm health.

Concentrations of metals detected in soil surrounding PCC were below health-based comparison values for soil. These comparison values are designed to be protective of gardeners and children playing in the soil. EHAP concluded that exposure to soil through gardening, eating local produce, and playing in dirt is not expected to harm health. For those concerned about contaminants in soil, resources for safe gardening are available at www.healthoregon.org/gardening.

Exposure pathways and risk calculations are described in greater detail in the "Health effects evaluation" section of this PHA and in Appendix G.

Historical Exposures

Community members want more information on historical exposures (including emergency releases of hazardous materials) that may have affected health.

There is very limited information on the historical exposures to emissions from PCC. The "Health effects evaluation" section of this PHA includes a discussion of the potential for historical emissions to harm health based on emission rates reported by PCC to the EPA since 1987. However, the data have limitations and only provides information about general emissions trends. Based on reported emissions rates, it is possible that historical air emissions were high enough to harm health. However, there is no historical air monitoring data available. EHAP concludes there is insufficient data to support a quantitative assessment.

Similarly, there is limited information about the amount of exposure that may have occurred during accidental releases that occurred in the past. Emergency releases can result in high, short-term exposures. However, EHAP does not have information about exposures during these past events. EHAP is not able to address risks of accidental short-term exposures in this assessment.

Environmental monitoring data

Community members wanted to know whether monitoring station locations were appropriate for identifying the maximum concentrations people may be exposed to and whether there are any additional types of data that would help to inform potential health risks. They also wanted to know how monitoring distinguishes between different forms of nickel.

DEQ selected air monitor locations (16) to capture metals concentrations near the source on three sides of the facility (Appendix B). The locations were selected based on information about emissions, wind directions, and access to properties where monitors could be placed. Nearby weather stations collected data on wind direction and wind speed. Some community members expressed concern that DEQ's monitoring locations were very close to PCC and may not adequately capture 'worst case' air concentrations if emissions spread farther through air before falling to the ground. Researchers at Portland State University also performed monitoring at additional locations farther away from the facility, on nearby residents' properties that may provide more information about air concentrations near homes. Once it is available, this information will be available from the PSU Sustainable Atmospheres Research (STAR) lab website: https://star.research.pdx.edu/PNAQ.html.

Different species of nickel have different degrees of toxicity. However, the air monitoring data that are available around PCC report total nickel concentrations and do not distinguish between different species. To make assumptions that protect health, EHAP calculated potential health risks under the assumption that all the nickel detected is in a more toxic form.

Health outcomes

Community members expressed concerns about cancer rates in the neighborhood and asked about the availability of additional health outcome data. They also asked if other health outcomes in the neighborhood such as immune disorders, autism, and other neurodevelopment conditions are related to air emissions.

Health outcome data (i.e., incidence of health outcomes such as cancer) can sometimes help identify increased risk of disease among people affected by environmental exposures. Use of health outcome data in PHAs is determined based on specific guidance in ATSDR's *Public Health Assessment Guidance Manual* (53). The main requirements for evaluating health outcome data are the presence of a completed human exposure pathway, contaminant levels high enough to

result in measurable health effects, a sufficient number of people in the completed pathway for health effects to be measured, and a health outcome database in which disease rates for the population of concern can be identified (53). When these requirements are not met, a health outcome study is unlikely to be able to detect health effects in a community even if they are present.

SPAQ made a formal request for a cancer analysis to be done using the Oregon State Cancer Registry (OSCaR). OHA denied the request because the situation does not meet its criteria for a cancer investigation. As described in OHA's formal response to SPAQ's request:

> *"The purpose of the Oregon State Cancer registry is two-fold: 1) to provide opportunities for Oregonians diagnosed with cancer to participate in scientific research projects aimed at improving the quality of cancer treatment; and 2) to monitor overall rates and trends in cancer in the population to target and evaluate prevention efforts. Its purpose is not to analyze cancer data to examine rates in small areas (neighborhoods) because such analyses do not yield useful information that assists in identifying environmental contaminants that people may be exposed to."*

OHA only conducts cancer investigations when all the following criteria are met: the cancer(s) of interest are rare, no environmental contaminants have already been identified as potential risk factors for cancer in the community, a defined geographic area is affected, and the time period of concern for cancer diagnoses can be established. In this case, the cancers associated with the chemicals of concern are not rare and the contaminants of concern are defined (the chemicals emitted from PCC). In addition, a lack of information about the extent of individuals' exposure would make it difficult to identify the specific population that should be included in the cancer analysis. We cannot determine how much carcinogen exposure a person near PCC may have had and are not able to control for other exposures that people farther from PCC may have had. Finally, the small population size of the communities around PCC would make it very difficult to detect increased cancer rates. If cancer rates in the community were higher than average, the cancer investigation would not be able to determine the cause; many different factors may contribute to cancer risk and cancer registry data cannot explain what caused any individual cancer case.

Cancer analysis is a public health tool that is helpful for estimating incidence of cancer across a large population. In contrast, health assessments that compare toxicology data to chemical concentrations detected in the environment are often a more sensitive tool for detecting potential health risks when changes in health outcomes are not yet detectable in the population. By comparing chemical concentrations in air, water, soil, and sediment with health-protective concentrations identified by toxicologists, EHAP can estimate very low cancer risks (on the scale of 1 in 1 million). It would not be possible to detect these relatively small increases in cancer risk in a small population.

There is no state registry to report diseases such as autoimmune disorders, autism, and other neurodevelopmental problems to OHA. Therefore, it is not possible to determine if rates found in this neighborhood are more or less or the same as expected.

In this PHA, we also explored recent scientific literature linking exposure to specific contaminants of concern at PCC with specific health outcomes of concern for community members. The potential health effects that have been identified for each chemical are described in Appendix F.

Biological Testing

Some community members expressed confusion about whether they should get their blood or urine tested and what the results would mean for their health.

OHA did not recommend that community members seek medical testing. Blood and urine measurements are not accurate predictors of long-term exposure to several of the metals of concern around PCC (e.g. arsenic, chromium, and nickel) because they do not stay in the body over long periods of time. Also, little is known about what specific concentrations of these metals in blood or urine mean for an individual's health. However, OHA, along with Multnomah County Health Department, developed a clinician guidance document (available at http://www.oregon.gov/oha/ph/newsadvisories/Documents/se-portland-metals-emissions-physician-guidance.pdf) to increase the likelihood that if a heavy metal medical test is performed, it is done correctly. This guidance also provides clinicians with information about how to interpret test results. The Northwest Pediatric Environmental Health Specialty Unit (NW PEHSU) can also help with interpretation, available at 206-221-8671 or visit the NW PEHSU website at www.depts.washington.edu/pehsu.

Sensitive Populations

Community members wanted to know how factors that influence susceptibility (such as epigenetics) and sensitive populations (elderly, children, and developing fetuses) would be addressed in the PHA.

Many factors influence how an individual processes and responds to chemicals in the environment. Genetics, epigenetics (changes in how genes are expressed that can be passed down through generations), life-stage, cumulative chemical exposures, nutrition, stress, pre-existing disease, and other factors can all interact in complex ways to influence our health. For example, children and developing fetuses can be particularly sensitive to chemical exposures because chemicals can change the way their bodies develop.

To the extent possible with existing science, the health effects evaluation in this PHA is designed to be protective of the most sensitive populations. However, scientific understanding of how these factors influence health is still evolving.

Because there is not enough information to support a quantitative assessment of the additional sensitivity of subgroups, we include a discussion of the factors that may influence susceptibility in the "Uncertainties and data gaps" section of this PHA.

Cumulative effects

Community Advisory Members raised concerns over the effects of cumulative exposure to multiple chemicals and pathways as well as additive or synergistic effects from the contaminants of concern.

In this PHA, when there were multiple chemicals with the potential to affect the same health outcomes, EHAP evaluated health effects of all chemicals cumulatively. To evaluate cancer risk associated with air emissions, EHAP evaluated cancer risk of all cancer-causing chemicals together. To evaluate cancer risk associated with Johnson Creek sediment, EHAP evaluated the cumulative cancer risk of all cancer-causing contaminants of concern that people may come in contact with through both skin contact and by swallowing. It is possible for chemicals to interact synergistically (to produce an effect that is greater than an additive effect), but there is no evidence that this is true for the chemicals evaluated in this PHA.

The primary focus of this PHA is to assess health risks from PCC. EHAP acknowledges the concern for exposures from other sources. This PHA does not include an in-depth review of exposure risks from other sources beyond the site. The EPA Transportation and Air Quality and Health program developed frequently asked questions on this issue, available at: https://www3.epa.gov/otaq/nearroadway.htm.

Risk communication

Community members expressed concerns about contamination in Johnson Creek. Some community members requested that signage be posted, warning of health risks due to bacteria or chemicals. Community members also noted that DEQ and OHA need clearer communication with the public.

Based on the results of EHAP's health assessment, occasional contact with chemical contaminants detected in Johnson Creek water and sediment are not expected to harm health. EHAP does not recommend posting warning signs about chemical contamination. However, like many urban streams, Johnson Creek frequently exceeds safe levels of bacterial contamination. Risk of bacterial infections is beyond the scope of EHAPs typical work to evaluate chemical risks, but EHAP recommends that community members take appropriate precautions when coming in contact with Johnson Creek and all urban streams to prevent bacterial infection. Specifically, people should avoid getting water from urban streams in their mouths and use clean water to

wash any parts of their bodies that come in contact with the stream, particularly before eating or drinking.

DEQ uses water quality standards for bacteria to evaluate safety of coastal water for recreational use: https://www.oregon.gov/deq/wq/Pages/WQ-Standards-Bacteria.aspx

OHA's Beach monitoring program provides information on health risks from bacteria in water and recommendations for reducing risk: https://www.oregon.gov/oha/PH/HEALTHYENVIRONMENTS/RECREATION/BEACHWATERQUALITY/Documents/pocketbrochure.pdf)

Emergency Preparedness

Community members expressed concern around PCC's emergency procedures, material storage, and shut down in the event of a disaster. They want to know whether PCC's chemical storage facilities are built to withstand an earthquake and how chemical releases would be prevented in an emergency. There was of particular concern around the potential health effects from sudden releases of materials onsite in the event of an emergency.

PCC has posted some information on emergency planning in the FAQ section of its community outreach website (54). The company reports it has a 'Contingency and Emergency Response Plan' that "includes but is not limited to: shutting off all utilities to prevent fire potential using backup generators to keep critical emissions controls operating. Chemicals are stored within secondary containment (e.g. lined concrete vaults)." Secondary containment practices and spill prevention and response plans are described in the Storm Water Pollution Control Plan submitted to DEQ (55). The company also reports participation in meetings with the Local Emergency Planning Committee.

Community members concerned about emergency preparedness may consider contacting the Multnomah County or Clackamas County Local Emergency Planning Committee. Contact information is available at:

https://www.oregon.gov/OSP/SFM/pages/local_emergency_planning_committees.aspx

The DEQ air program does not regulate emergency preparedness and does not have documentation of PCCs emergency response plans.

Noises and Odors

Community members expressed concern over loud grinding noises and odors coming from the site. They also expressed a desire for a better understanding of what all the stacks at PCC are used for and greater transparency about PCC's processes and emissions.

EHAP cannot identify if any odor is coming from PCC. DEQ enforces nuisance odor complaints in Oregon. EHAP encourages communities to file nuisance odor related complaints with DEQ, see resources to do so below:

- DEQ Odors Complaint Online Form http://www.deq.state.or.us/complaints/dcomplaint.aspx
- OHA Odors fact sheet https://public.health.oregon.gov/HealthyEnvironments/HealthyNeighborhoods/ToxicSubstances/Documents/OdorsAndYourHealth_Final.pdf
- ATSDR Odors Resources https://www.atsdr.cdc.gov/odors

The state of Oregon has noise standards (OAR 340, Division 35) that are enforced by local agencies. Neighbors that are disturbed by noise at PCC can contact city and county officials:

- Portland Noise Control Program: https://www.portlandoregon.gov/oni/63242
- City of Milwaukie: https://www.milwaukieoregon.gov/police/code-enforcement-complaint-form

Appendix E. Comparison values and contaminant screening

This appendix defines the various comparison values (CVs) that were used in this Public Health Assessment and describes the hierarchy by which they were chosen. It also includes more detailed screening tables for environmental monitoring data near PCC. This process is also explained in Chapter 7 of ATSDR's Public Health Assessment Guidance Manual (53). ATSDR uses the hierarchy shown in Figure A1 to choose CVs for screening purposes. CVs used in this document are listed below:

Environmental Media Evaluation Guides (EMEGs)
EMEGs are an estimate of contaminant concentrations low enough that ATSDR would not expect people to have a negative, non-cancerous health effect. EMEGs are based on ATSDR Minimal Risk Levels (MRLs, described below) and conservative assumptions about the public's contact with contaminated media, such as how much, how often, and for how long someone may be in contact with the contaminated media. EMEGs also account for body weight.

Cancer Risk Guides (CREGs)
CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed during their lifetime (70 years). ATSDR's CREGs are calculated from EPA's cancer slope factors (CSFs) for oral exposures or unit risk values for inhalation exposures. These values are based on EPA evaluations and assumptions about hypothetical cancer risks at low levels of exposure.

Reference Dose Media Evaluation Guides (RMEGs)
ATSDR derives RMEGs from EPA's oral reference doses, which are developed based on EPA evaluations. RMEGs represent chemical concentrations in water or soil at which daily human contact is not likely to cause negative, non-cancerous health effects.

Minimal Risk Levels (MRLs)
A MRL is an estimate of daily human exposure – by a specified route and length of time - to a dose of a chemical that is likely to be without a measurable risk of negative, non-cancerous effects. MRLs are based on ATSDR evaluations. Acute MRLs are designed to evaluate exposures lasting 14 days or less. Intermediate MRLs are designed to evaluate exposures lasting from 15-364 days. Chronic MRLs are designed to evaluate exposures lasting for 1 year or longer.
Oral exposures (swallowing the contaminant) are measured in milligrams per kilogram per day [mg/kg/day] and inhalation exposures (breathing the contaminant) are measured in parts per billion [ppb] or micrograms per cubic meter [µg/m3].

Maximum Contaminant Levels (MCL)

MCLs are derived by EPA as enforceable standards for municipal water systems. These standards are not strictly health-based but are set as close to the maximum contaminant level goals (MCLGs) (health goals) as is feasible and are based upon treatment technologies, costs (affordability) and other feasibility factors, such as the availability of analytical methods, treatment technology and costs for achieving various levels of removal.

Regional Screening Levels (RSLs)

RSLs are contaminant concentrations in soil, water, or air, below which any negative health effects would be unlikely. RSLs are derived by EPA's Region 3 Office using
EPA's reference doses (RfDs) and cancer slope factors (CSFs). This ensures that RSLs consider both non-cancer and cancer risks. RSLs are available online at: (http://www.epa.gov/reg3hwmd/risk/human/rbconcentration_table/Generic_Tables/index.htm)

Table E1. Screening of air concentrations prior to HEPA filter installation (3/30/16-5/16/16)

	45th and Harney Monitor (PFH)			S.E. Harney Dive. Monitor (PHD)			Milwaukie Johnson Creek Monitor (MJC)			Comparison Values (CV)					
	Average ng/m³	UCL ng/m³	Max ng/m³	Average ng/m³	UCL ng/m³	Max ng/m³	Average ng/m³	UCL ng/m³	Max ng/m³	Cancer CV ng/m³	Cancer CV Source	Non-cancer CV ng/m³	Non-cancer CV source	Non-cancer health effect	COC?
Arsenic	0.74	0.87	2.25	0.81	0.96	4.40	0.88	1.05	5.03	0.23	ATSDR CREG	NA	NA	NA	yes
Beryllium	0.01	0.01	0.02	<0.086	NA	<0.086	<0.086	NA	<0.086	0.42	ATSDR CREG	NA	NA	NA	no
Cadmium	0.09	0.12	0.23	0.17	0.21	0.45	0.14	0.15	0.24	0.56	ATSDR CREG	10	ATSDR chronic MRL	kidney function	no
Chromium	19.14	NA	31.60	30.91	NA	39.00	42.03	NA	60.30	(see hexavalent chromium)					
Cobalt	1.45	4.80	25.40	1.02	1.38	7.32	3.35	9.50	36.30	NA	NA	100	ATSDR chronic MRL	respiratory function	no
Hexavalent Chromium	0.11	0.15	0.44	0.18	0.23	1.01	0.31	0.39	1.16	0.052	ATSDR CREG	5	ATSDR chronic MRL	upper respiratory effects	yes
Lead	2.20	2.57	5.39	2.26	2.61	5.34	2.08	2.46	4.84	NA	NA	150	ABC/NAAQS	brain development	no
Manganese	7.33	8.98	26.60	9.56	11.57	31.60	7.03	8.90	26.60	NA	NA	300	ATSDR chronic MRL	neurological function	no
Nickel	6.30	11.25	44.50	9.11	12.41	43.00	22.28	31.68	131.00	4	EPA RSL/ ABC	90	ATSDR chronic MRL	respiratory inflammation	yes
Selenium	0.17	0.20	1.06	0.65	NA	0.87	0.74	NA	1.12	NA	NA	20,000	EPA RSL	selenosis	no

NA - Not Available; CV – Comparison Value; UCL – Upper Confidence Limit
CREG – Cancer Risk Evaluation Guide for cancer effects (ATSDR)
RSL – Regional Screening Level; Environmental Protection Agency (EPA)
NAAQS- National Ambient Air Quality Standards (EPA)
ABC – Ambient Benchmark Concentration (Oregon DEQ)

Table E2. Screening of air concentrations under current conditions (5/17/16-1/22/17)

	45th and Harney Monitor (PFH)			S.E. Harney Drive Monitor (PHD)			Milwaukie Johnson Creek Monitor (MJC)			Comparison Values					
	Average ng/m³	UCL ng/m³	Max ng/m³	Average ng/m³	UCL ng/m³	Max ng/m³	Average ng/m³	UCL ng/m³	Max ng/m³	Cancer CV ng/m³	Cancer CV Source	Non-cancer CV ng/m³	Non-cancer CV source	Non-cancer health effect	COC?
Arsenic	0.6633	0.92	5.48	0.4621	0.65	3.48	0.448	0.62	3.42	0.23	ATSDR CREG	NA	NA	NA	yes
Beryllium	0.0064	0.01	0.018	<0.086	NA	<0.086	<0.086	NA	<0.086	0.42	ATSDR CREG	NA	NA	NA	no
Cadmium	0.1302	0.21	1.3	0.6826	3.16	9.19	0.1186	0.13	0.214	0.56	ATSDR CREG	10	ATSDR chronic MRL	kidney function	yes
Chromium	1.7308		4.83	31.2		31.2	33.554		63.2	(see hexavalent chromium)					
Cobalt	0.134	0.15	0.54	0.3874	0.52	2.63	1.1807	1.42	13.1	NA	NA	100	ATSDR chronic MRL	respiratory function	no
Hexavalent Chromium	0.0772	0.08	0.243	0.1197	0.13	0.589	0.3297	0.37	1.7	0.052	ATSDR CREG	5	ATSDR chronic MRL	upper respiratory effects	yes
Lead	1.8774	2.03	8.65	1.4416	1.56	5.12	1.4352	1.56	5.99	NA	NA	150	ABC/NAA QS	brain development	no
Manganese	5.8653	6.44	36.3	7.6014	8.29	35.8	8.8073	9.80	39.1	NA	NA	300	ATSDR chronic MRL	neurological function	no
Nickel	0.7998	0.88	2.93	2.6301	3.68	15.4	9.5025	11.03	51	4	EPA RSL/ ABC	90	ATSDR chronic MRL	respiratory inflammation	yes
Selenium	0.1971	0.21	1.48	0.4975	0.63	1.03	0.7286	1.76	3.56	NA	NA	20,000	EPA RSL	selenosis	no

NA - Not Available; CV – Comparison Value; UCL – Upper Confidence Limit
CREG – Cancer Risk Evaluation Guide for cancer effects (ATSDR)
RSL – Regional Screening Level; Environmental Protection Agency (EPA)
NAAQS- National Ambient Air Quality Standards (EPA)
ABC – Ambient Benchmark Concentration (Oregon DEQ)

Table E3. Screening of soil concentrations detected June 2016

Contaminant	Average Concentration mg/kg (ppm)	Max Concentration mg/kg (ppm)	Cancer Comparison Value mg/kg (ppm)	Cancer CV source	Non-cancer Comparison Value mg/kg (ppm)	Non-cancer CV source	Chemical of Potential Concern?
Arsenic, Total	4.76	10.90	0.25	ATSDR CREG	17	ATSDR chronic child EMEG and RMEG (dermal effects)	no
Beryllium, Total	0.54	0.66	NA	NA	110	ATSDR chronic child EMEG and RMEG (gastrointestinal effects)	no
Cadmium, Total	0.28	0.82	NA	NA	5.7	ATSDR chronic child EMEG (kidney function)	no
Chromium, Total	53.36	239.00	NA	NA	86,000	ATSDR RMEG for trivalent chromium	no
Chromium, Hexavalent	1.17	5.26	NA	NA	51	ATSDR chronic child EMEG (intestinal effects)	no
Cobalt, Total	20.17	81.00	NA	NA	570	ATSDR intermediate child EMEG (hematological effects)	no
Iron, Total	27,736.67	36,600.00	NA	NA	55,000	EPA residential RSL (gastrointestinal effects)	no
Lead, Total	34.17	91.80	NA	NA	400	EPA lead standard for bare soil in children's play areas (impaired neurodevelopment)	no
Manganese, Total	706.67	1,030.00	NA	NA	2,900	ATSDR chronic child RMEG (neurological function)	no
Nickel, Total	123.43	776.00	NA	NA	1,100	ATSDR chronic child RMEG (decreased body weight)	no
Selenium, Total	0.17	0.36	NA	NA	290	ATSDR chronic child EMEG and RMEG (selenosis)	no
Titanium, Total	1795.00	2680.00	NA	NA	140,000	EPA residential RSL for titanium tetrachloride; no CVs are available for titanium alone	no
Zinc, Total	100.05	213.00	NA	NA	17,000	ATSDR chronic child EMEG (copper deficiency)	no

Table E4. Screening for chemicals in Johnson Creek sediment (Landau Associates 2009-2015)

Chemicals Detected	Max concentration in sediment mg/kg (ppm)	Soil Cancer CV (ppm)	Cancer CV Source	Soil Non-cancer CV (ppm)	Non-cancer CV Source	Chemical of Potential Concern?
Antimony	0.66	NA	NA	23	ATSDR child chronic RMEG	no
Arsenic	6.56	0.25	ATSDR CREG	17	ATSDR child chronic EMEG	no
Barium	1.05	NA	NA	11,000	ATSDR child chronic EMEG	no
Beryllium	0.41	NA	NA	110	ATSDR child chronic EMEG	no
Cadmium	0.67	NA	NA	5.7	ATSDR child chronic EMEG	no
Chromium, Total	1000	NA	NA	75,000	ATSDR child chronic RMEG for trivalent chromium	no
Chromium, hexavalent[A]	22	NA	NA	51	ATSDR chronic child EMEG for hexavalent chromium	no
Copper	100	NA	NA	570	ATSDR child intermediate EMEG	no
Lead	61.8	NA	NA	400	EPA lead standard for bare soil in children's play areas	no
Mercury	0.20	NA	NA	17	ATSDR child chronic EMEG for methylmercury	no
Nickel	2500	NA	NA	1,100	ATSDR child chronic RMEG	yes
Zinc	260	NA	NA	17,000	ATSDR child chronic EMEG	no
Total PCB	0.48	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	yes
Total PAH	0.34	0.12	ATSDR CREG	NA	NA	yes

[A]Estimated by adjusting average and maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33).

Table E5. Screening for individual Aroclor mixtures (PCBs) in sediment (Landau Associates 2009-2015).

Chemicals Detected	Max Concentration Detected (ppm)	Soil Cancer Comparison Value (ppm)	Cancer Comparison Value Source	Soil Non-cancer Comparison Value (ppm)	Cancer Comparison Value Source	Chemical of Potential Concern?
Aroclor 1242	0.016	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	no
Aroclor 1254	0.48	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	yes
Aroclor 1260	0.13	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	no
Aroclor 1262	0.008	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	no
Total PCB[A]	0.48	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	yes

[A]Reflects the maximum sum of PCB concentrations detected in any individual sediment sample.

Table E6. Screening for individual PAHs in sediment (Landau Associates 2009-2015).

PAHs Detected	Max Concentration Detected (ppm)	Relative Potency Factor (RPF)[A]	RPF-adjusted Concentration	Cancer Comparison Value (ppm) for soil	Cancer Comparison Value Source	Non-cancer comparison value (ppm) for soil	Non-cancer Comparison Value Source	Chemical of Potential Concern?
Acenaphthene	0.026	0.001[B]	0.000026	0.12	ATSDR CREG	3,400	ATSDR chronic child RMEG	no
Acenaphthylene	0.027	0.001[B]	0.000027	0.12	ATSDR CREG	NA	NA	no
Anthracene	0.027	0	0	0.12	ATSDR CREG	17,000	ATSDR chronic child RMEG	no
Benzo(a)anthracene	0.12	0.2	0.024	0.12	ATSDR CREG	NA	NA	no
Benzo(a)pyrene	0.17	1	0.17	0.12	ATSDR CREG	17	ATSDR chronic child RMEG	yes
Benzo(b)fluoranthene	0.12	0.8	0.096	0.12	ATSDR CREG	NA	NA	no
Benzo(g,h,i)perylene	0.13	0.009	0.00117	0.12	ATSDR CREG	NA	NA	no
Benzo(k)fluoranthene	0.11	0.03	0.0033	0.12	ATSDR CREG	NA	NA	no
Chrysene	0.15	0.1	0.015	0.12	ATSDR CREG	NA	NA	no
Fluoranthene	0.38	0.08	0.0304	0.12	ATSDR CREG	2,300	ATSDR chronic child RMEG	no
Indeno(1,2,3-cd)pyrene	0.13	0.07	0.0091	0.12	ATSDR CREG	NA	NA	no
Phenanthrene	0.32	0	0	0.12	ATSDR CREG	NA	NA	no
Pyrene	0.55	0	0	0.12	ATSDR CREG	1,700	ATSDR chronic child RMEG	no
Total PAH[C]			0.34	0.12	ATSDR CREG	NA	NA	yes

[A]Benzo(a)pyrene is the only PAH with an ATSDR CREG. EPA has developed 'Relative Potency Factors' (RPF) that quantify the cancer-causing potency of other PAHs relative to benzo(a)pyrene (56). Cancer comparison values should be compared to RPF-adjusted concentrations.

[B]EPA did not assign RPFs for acenaphthene and acenaphthylene, so alternate values cited by EPA were used instead.

[C]Reflects the maximum sum of PAH concentrations detected in any individual sediment sample. Total PAH concentrations are the sum of 'benzo(a)pyrene equivalent' concentrations (the detected concentration multiplied by EPA's chemical-specific Relative Potency Factor) for all PAHs detected in each sample. Note that this is not equal to the sum of RPF-adjusted concentrations for individual chemicals because maximum concentrations for individual chemicals were not all at the same location

Table E7. Screening for chemicals in Johnson Creek sediment (Landau Associates incremental sampling 2017)

Chemicals Detected	Max concentration in sediment mg/kg (ppm)	Soil Cancer CV (ppm)	Cancer CV Source	Soil Non-cancer CV (ppm)	Non-cancer CV source	Chemical of Potential Concern?
Antimony	<0.5[B]	NA	NA	23	ATSDR child chronic RMEG	no
Arsenic	2.57	0.25	ATSDR CREG	17	ATSDR child chronic EMEG	no
Beryllium	0.478	NA	NA	110	ATSDR child chronic EMEG	no
Cadmium	<0.5[B]	NA	NA	5.7	ATSDR child chronic EMEG	no
Chromium, Total	23.3	NA	NA	75,000	ATSDR chronic child RMEG for trivalent chromium	no
Chromium, hexavalent	0.51	NA	NA	51	ATSDR chronic child EMEG	no
Copper	30.7	NA	NA	570	ATSDR child intermediate EMEG	no
Lead	27.9	NA	NA	400	EPA residential RSL standard for bare soil in children's play areas	no
Mercury	0.0657[C]	NA	NA	17	ATSDR child chronic EMEG for methylmercury	no
Nickel	49.8	NA	NA	1,100	ATSDR child chronic RMEG	no
Selenium	<1[B]	NA	NA	290	ATSDR child chronic EMEG and RMEG	no
Silver	<0.5[B]	NA	NA	290	ATSDR child chronic RMEG	no
Thallium	<0.5[B]	NA	NA	NA	NA	no
Zinc	197	NA	NA	17,000	ATSDR child chronic EMEG	no
Total PCB	0.1299[C]	0.19	ATSDR CREG	1.1	ATSDR child chronic EMEG	no

NA indicates comparison values are not available

[A] Estimated by adjusting average and maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33).

[B] The chemical was not detected above the sample quantitation limit shown; These chemicals will not be included in further analysis.

[C] Concentration was estimated because the chemical was detected, but it is below the level that can be accurately quantified.

Table E8. Screening for chemicals in Johnson Creek sediment (DEQ sampling 2016)

Chemical	Max concentration in sediment mg/kg (ppm)	Soil Cancer CV (ppm)	Cancer CV Source	Soil non-cancer CV (ppm)	Non-cancer CV Source	Chemical of Potential Concern?
Aluminum, Total	16,900	NA	NA	57,000	ATSDR child chronic EMEG	no
Antimony, Total	0.39	NA	NA	23	ATSDR child chronic RMEG	no
Arsenic, Total	2.27	0.25	ATSDR CREG	17	ATSDR child chronic EMEG	no
Barium, Total	114	NA	NA	11,000	ATSDR child chronic EMEG	no
Cadmium, Total	0.22	NA	NA	5.7	ATSDR child chronic EMEG	no
Chromium, Total	476	NA	NA	75,000	ATSDR child chronic RMEG for trivalent chromium	no
Chromium, hexavalent	10.5	NA	NA	51	ATSDR chronic child EMEG for hexavalent chromium	no
Cobalt, Total	131			570	ATSDR child intermediate EMEG	no
Copper, Total	42.4	NA	NA	570	ATSDR child intermediate EMEG	no
Lead, Total	42.3	NA	NA	400	EPA lead standard for bare soil in children's play areas	no
Manganese, Total	268			2,900	ATSDR child chronic RMEG	no
Mercury, Total	<0.040[B]	NA	NA	17	ATSDR child chronic EMEG for methylmercury	no
Nickel, Total	1,600	NA	NA	1,100	ATSDR child chronic RMEG	yes
Selenium, Total	<1.99[B]	NA	NA	290	ATSDR child chronic EMEG and RMEG	no
Silver, Total	<0.10[B]	NA	NA	290	ATSDR child chronic RMEG	no
Thallium, Total	<0.10[B]	NA	NA	NA	NA	no
Zinc, Total	179	NA	NA	17,000	ATSDR child chronic EMEG	no

Contaminants of concern (detected at concentrations exceeding the comparison value) are highlighted in grey.

NA indicates comparison values are not available

[A] Estimated by adjusting average and maximum concentrations of total chromium in soil with EPA's estimate that 2.2% of total chromium will be in the hexavalent form (33).

[B] The chemical was not detected above the reporting limit shown

Table E9. Sources of oral comparison values for calculation of crayfish consumption rates

Chemical	Concentration in Crayfish (mg/kg wet wt)	Non-cancer CV (mg/kg body weight /day)	Non-cancer CV Source	Target Organ
Arsenic, Total	0.28	0.0003	ATSDR Oral MRL	skin
Cadmium, Total	<0.03[A]	0.0001	ATSDR Oral MRL	kidneys
Chromium, Total	0.63	0.0009	ATSDR Oral MRL for hexavalent chromium (makes cautious assumption that all chromium detected is hexavalent)	blood; liver
Cobalt, Total	0.26	0.01	ATSDR intermediate oral MRL	blood
Mercury, Total	0.019	0.0003	ATSDR Oral MRL (makes cautious assumption that all mercury present is methylmercury)	brain; prenatal development
Nickel, Total	1.08	0.02	EPA Oral RfD	decreased body weight
PCBs, Total	0.033	0.00002	EPA Oral RfD for Arochlor 1254	Immune, skin, eyes; brain development
Selenium, Total	<0.59[A]	0.005	ATSDR Oral MRL	skin, blood (selenosis)
Titanium, Total	11.8	NA	NA	NA
Zinc, Total	24.1	0.3	ATSDR Oral MRL	blood, immune

[A] The chemical was not detected above the reporting limit shown

Appendix F. Contaminants of concern and health guideline values used

The chemicals described here were identified as contaminants of concern in the screening portion of this PHA.

Arsenic. Arsenic is a naturally-occurring metal widely distributed in soil. Most arsenic compounds have no smell or special taste (39). Arsenic's toxicity has been recognized since ancient times, and scientists are continuing to learn more about how it works and its additional toxic effects on human health. Arsenic is a known cancer-causing chemical. The types of cancer most often associated with arsenic exposure are skin, bladder, and lung (when inhaled) cancers (39). At higher doses, arsenic can also cause skin conditions that involve discoloration and hardening of the skin as well as appearance of corns or warts on the palms, soles, and torso (39). In addition to these effects on the skin, arsenic can also cause nerve damage (numbness in the extremities) at high doses and more subtle effects on the brain at lower doses over a long time (39).

There is some evidence that inhaled or ingested inorganic arsenic can injure pregnant women or their unborn babies, although the studies are not definitive. We do not know if absorption of inorganic arsenic from the gut in children differs from adults. There is some evidence that exposure to arsenic in early life (including gestation and early childhood) may increase mortality in young adults. Studies in animals show that large doses of inorganic arsenic that cause illness in pregnant females can also cause low birth weight, fetal malformations, and even fetal death. There is also some evidence that suggests that long-term exposure to inorganic arsenic in children may result in lower IQ scores. Arsenic can cross the placenta and has been found in fetal tissues. Arsenic is found at low levels in breast milk.

Soil sampling performed around PCC detected levels of arsenic above ATSDR's CREG for soil. However, the levels of arsenic measured in soil were not different from background levels measured in the Portland area. These background levels are due to Oregon's unique volcanic geology – volcanic soils naturally contain high levels of metals such as arsenic and mercury. The background levels in Portland are similar to background levels statewide. Most (if not all) soils in Oregon will have levels of arsenic that are higher than health screening and cleanup levels. Because normal background levels of arsenic in soil are often above the conservative ATSDR CREG, ATSDR recommends using the ATSDR child EMEG for non-cancer risk of exposure to soil as the comparison value for evaluating public health effects at contaminated sites.

Comparison values for arsenic

- **Inhalation CVs.** The comparison value used for air exposure to arsenic in this PHA is the ATSDR CREG of 0.23ng/m^3 for a 1 in 1 million lifetime cancer risk. The CREG is lifetime cancer risk values derived from EPA's inhalation unit risk for arsenic of 4.3$(ng/m^3)^{-1}$ designed to be protective of lung cancer in people. Non-cancer comparison values are not available for inhalation of arsenic (39).

- **Ingestion CVs.** The comparison value used for arsenic exposure in soil and sediment in this PHA is ATSDR's child EMEG for chronic exposure, 17 mg/kg (ppm). This chronic non-cancer comparison value is derived from EPA's reference dose of 0.3ug/kg/day and is designed to be protective of effects on the heart and skin (39). An alternate CV is the ATSDR CREG for arsenic lifetime cancer risk in soil and sediment, 0.25 mg/kg (ppm). This conservative (health-protective) cancer risk value is below natural background concentrations of arsenic found in soil across the country. ATSDR therefore recommends using the EMEG for chronic child exposures instead of the CREG as a comparison value for public health assessments.

Cadmium. Cadmium is a soft, silver-white metal that occurs naturally in the earth's crust. Cadmium is not usually present in the environment as a pure metal, but as a mineral combined with other elements. It is most often present in nature as complex oxides, sulfides, and carbonates in zinc, lead, and copper ores. Cadmium has many industrial uses and is used in consumer products including batteries, pigments, metal coatings, plastics, and some alloys (57).

Low levels of cadmium are present in most foods with the highest levels present in shellfish, liver, and kidney meats (57). Cigarette smoke also contains cadmium and can double the daily intake when compared to a non-smoker. Ingestion of high levels of cadmium in contaminated food or water can severely irritate the stomach, leading to vomiting and diarrhea, and sometimes death. Cadmium is a cumulative toxicant and ingestion of lower levels for a long period (above the chronic Minimal Risk Level [MRL] of 10 ng/m^3) of time can lead to a buildup of cadmium in the kidneys and, possibly, kidney damage. The kidney is the main target organ for cadmium toxicity following chronic-duration exposure by both oral and inhalation routes. Cadmium interferes with proper functioning of the kidney by damaging the proximal tubules and impairing the kidneys' ability retain and resorb large molecules. Cadmium also prevents the kidney from retaining calcium, so prolonged exposure can lead to calcium depletion and loss of bone density (57).

A few studies in animals indicate that younger animals absorb more cadmium than adults. Animal studies also indicate that the young are more susceptible than adults to a loss of bone and decreased bone strength from exposure to cadmium. Cadmium is found in breast milk and a small amount will enter the infant's body through breastfeeding. The amount of cadmium that can pass to the infant depends on how much exposure the mother may have had. We do not know whether cadmium can cause birth defects in people. Studies in animals exposed to high enough levels of cadmium during pregnancy have resulted in harmful effects in the young. The nervous system appears to be the most sensitive target. Young animals exposed to cadmium before birth have shown effects on behavior and learning. There is also some information from animal studies that high enough exposures to cadmium before birth can reduce body weights and affect the skeleton in the developing young (57).

There is some evidence to suggest an association between cadmium and breast cancer. One analysis of multiple case-control studies in people found that each 0.5-µg/g creatinine increment

of urinary cadmium concentration was associated with a 66% increased risk of breast cancer (58). While evidence from epidemiological studies have been inconsistent, the association is plausible based on evidence from laboratory studies indicating that cadmium may influence estrogen signaling (59) (60).

There is also some evidence that cadmium may impair brain development. Young animals exposed to cadmium before birth have shown effects on behavior and learning (57). Recent epidemiological studies have found limited evidence of similar effects in people. For example, a study in China found an association between cadmium in mothers' blood during pregnancy and delayed development in infants (61). In a study of children in Greece, elevated maternal urinary cadmium concentrations (≥0.8 µg/L) during pregnancy were associated with lower cognitive scores, though in that study the effect was limited to mothers who smoked (62). There is also evidence that exposure to lead and cadmium during pregnancy may act synergistically to affect brain development (63).

There is insufficient peer-reviewed data on the association between cadmium and breast cancer and cadmium and brain development to support a quantitative evaluation of their risks in this PHA. The potential effect of cadmium on these other health endpoints should be evaluated in the context of potential cumulative effects from other chemicals. For example, if cadmium affects brain development, concurrent exposures to cadmium and lead in the air around PCC could have had cumulative or synergistic effects.

The exposure route of concern for cadmium in this PHA is inhalation of contaminated air. The EPA has classified cadmium as a probable human carcinogen by inhalation. This is based on limited evidence of an increase in lung cancer in humans from occupational exposure to cadmium fumes and dust. This is further supported by evidence of lung cancer in rats (57).

Comparison values for cadmium

- **Inhalation CVs.** The comparison value used for air exposure to cadmium in this PHA is the ATSDR CREG of 0.56 ng/m^3 for a 1 in 1 million lifetime cancer risk. This lifetime cancer risk is derived from EPA's inhalation unit risk for cadmium, 1.8 (ng/m^3)$^{-1}$, designed to be protective of respiratory cancers. The non-cancer comparison value used for cadmium is the ATSDR chronic EMEG of 10 ng/m^3, based on the ATSDR inhalation MRL, designed to be protective of chronic effects on the kidney (57).
- **Ingestion CVs.** The comparison value used for soil and sediment exposure to cadmium in this PHA is the ATSDR chronic EMEG of 5.7 mg/kg (ppm). This chronic non-cancer risk value is based on the ATSDR ingestion MRL and is designed to be protective of chronic effects on the kidney (57). There are no cancer risk comparison values available for exposure to cadmium through ingestion.

Hexavalent chromium. Chromium is a naturally occurring element found in rocks, animals, plants, and soil. It can exist in several different forms. The trivalent form and hexavalent form

are the most common forms of chromium measured in the environment. Hexavalent chromium is substantially more toxic than trivalent (33). Small amounts of trivalent chromium are considered to be a necessity for human health. Chromium can easily change from one form to another in water and soil, depending on the conditions present. Chromium is widely used in manufacturing and is found in products such as treated wood, tanned leather and stainless-steel cookware (33).

The main health problems seen in animals following ingestion of hexavalent chromium are anemia and irritation and ulcers in the stomach and small intestine. Trivalent chromium compounds are much less toxic and do not appear to cause these problems. Sperm damage and damage to the male reproductive system have also been seen in laboratory animals exposed to hexavalent chromium. Skin contact with certain hexavalent chromium compounds can cause skin ulcers (33). Some people are extremely sensitive to hexavalent chromium or trivalent chromium. Allergic reactions consisting of severe redness and swelling of the skin have been noted.

ATSDR, the International Agency for Research on Cancer (IARC), and EPA have determined that hexavalent chromium compounds are "known" human carcinogens through the exposure route of inhalation. In workers, inhalation of hexavalent chromium has been shown to cause lung cancer. Hexavalent chromium also causes lung cancer in animals. An increase in stomach tumors was observed in humans and animals exposed to hexavalent chromium in drinking water (33).

Children are more sensitive than adults to the cancer effects because hexavalent chromium has a "mutagenic mode of action". This means that the carcinogen reacts and binds to the DNA in our cells (64). Children are assumed to be at increased risk for cancer and tumor development following exposure to mutagenic compounds because their bodies are growing – their cells are rapidly replicating during this time. It is thought that a child's DNA repair mechanisms may not be able to keep up with the rapid cell replication (64).

Scientific studies of chromium haven't fully demonstrated if exposure to chromium could result in birth defects or other developmental effects in people. Some developmental effects have been observed in animals exposed to hexavalent chromium. In animals, some studies show that exposure to high doses during pregnancy may cause miscarriage, low birth weight, and some changes in development of the skeleton and reproductive system. Birth defects in animals may be related, in part, to chromium toxicity in the mothers (33).

Comparison values for hexavalent chromium

- **Inhalation CVs.** The comparison value used for air exposure to hexavalent chromium in this PHA is the ATSDR CREG of 0.052 ng/m^3 for a 1 in 1 million lifetime cancer risk**.** This lifetime cancer risk value is based on EPA's inhalation unit risk for hexavalent chromium, 1.2 (ug/m^3)$^{-1}$ designed to be protective of lung cancer. The non-cancer

comparison value used for hexavalent chromium is the ATSDR chronic EMEG of 5 ng/m^3, based on the ATSDR inhalation MRL designed to be protective of upper respiratory effects (33).

- **Ingestion CVs.** The comparison value used for soil and sediment exposure to hexavalent chromium is ATSDR's EMEG, 51 mg/kg (ppm). This EMEG is derived from ATSDR's chronic ingestion MRL, based on intestinal effects in mice (33). There are no cancer risk comparison values available for exposure to hexavalent chromium through ingestion.

Nickel. Pure nickel is a hard, silvery-white metal, which has properties that make it very desirable for combining with other metals to form mixtures called alloys. Some of the metals that nickel can be alloyed with are iron, copper, chromium, and zinc. The toxicity of nickel may vary with the specific form it takes and the route of exposure (65). Nickel and its compounds have no characteristic odor or taste. The nickel that comes out of the stacks of power plants attaches to small particles of dust that settle to the ground or are taken out of the air in rain or snow. It usually takes many days for nickel to be removed from the air. If the nickel is attached to very small particles, it can take more than a month to settle out of the air.

Primary targets of toxicity appear to be the respiratory tract following inhalation exposure, the immune system following inhalation, oral, or dermal exposure, and possibly the reproductive system and the developing organism following oral exposure. The most common harmful health effect of nickel in humans is an allergic reaction. Approximately 10–20% of the population is sensitive to nickel. Once a person is sensitized to nickel, further contact with the metal may produce a reaction (65).

The most serious harmful health effects from exposure to nickel are respiratory effects such as chronic bronchitis, reduced lung function, and cancer of the lung and nasal sinus. The International Agency for Research on Cancer and the US EPA have concluded that some forms of nickel are carcinogenic to humans (65). Effects of nickel on the respiratory system have been documented in animal studies and 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 were much higher than usual (background) levels in the environment (65).

We do not know whether children differ from adults in their susceptibility to nickel. Human studies that examined whether nickel can harm the developing fetus are inconclusive. Animal studies have found increases in newborn deaths and decreases in newborn weight after ingesting nickel. These doses are 1,000 times higher than levels typically found in drinking water. It is likely that nickel can be transferred from the mother to an infant in breast milk and can cross the placenta (65).

Developing lungs may be particularly susceptible to chemicals that affect respiratory health. There is some evidence that children exposed to other forms of air pollution during gestational development and early life are more likely to have decreased lung function and asthma later in life (66) (67).

Nickel used in manufacturing at Precision Castparts is in an alloy form. There is some evidence that alloys may be less bioavailable and therefore less toxic than nickel alone (40). However, nickel monitoring of ambient air near PCC only provides information about total nickel concentrations and does not distinguish between forms of nickel. To be health protective, this health assessment starts from a "worst case" scenario in which all nickel detected is in a more bioavailable form.

The peer-reviewed comparison values used for this PHA may not reflect all the latest research or protect against potential health effects that are currently being studied by scientists. For example, a few recent studies indicate that nickel in air may increase risk of asthma symptoms in children. In one study, a 14 ng/m^3 increase in nickel concentrations was associated with a 28% increase in risk of wheeze in children under 2 years old (68). In another study, a 4 ng/m^3 increase in nickel concentrations was associated with an 11% increase in risk of asthma symptoms in adolescents (69). In both studies, other metals were also present in air, making it difficult to establish the degree to which the effect is due to nickel alone or in combination with other exposures. Other studies have found an association between nickel in air and risk of nickel sensitivity. A study in Germany found that children consistently exposed to nickel concentrations above 12 ng/m^3 were four times more likely to develop an immune sensitivity to nickel than children exposed to less than 2.5ng/m^3 nickel in air (70). These studies suggest the potential for nickel to have respiratory and immune effects at concentrations comparable to what has been detected near PCC. However, these studies alone do not provide conclusive evidence that nickel causes these symptoms and could not be used to support quantitative health effects analysis in this PHA. Generally, findings from new studies must be replicated and corroborated by other studies with different designs, settings, and populations before previously established guidelines or standards can be updated

Comparison values for nickel

- **Inhalation CVs.** The comparison value used for air exposure to nickel in this PHA is EPA's residential screening level of 4ng/m^3 in air for a 1 in a million cancer risk. This value is derived from EPA's inhalation unit risk for cancer risk of nickel of 0.24 (ng/m^3)$^{-1}$ based on data on cancer risk from occupational exposure to nickel refinery dust. Non-cancer risk was evaluated using ATSDR chronic minimal risk level of 90 ng/m^3 designed to be protective of effects of nickel sulfate on the respiratory system (65).
- **Ingestion CVs.** The comparison values used for water, soil, and sediment exposures in this PHA are ATSDR's chronic RMEGs for soil and water. These values are derived from EPA's oral reference dose for nickel ingestion of 0.02 mg/kg/day and is designed to be protective of long-term effects of nickel soluble salts on decreased body weight (65).

Polycyclic aromatic hydrocarbons. Polycyclic aromatic hydrocarbons (PAHs) are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or

other organic substances, such as tobacco and charbroiled meat. There are more than 100 different PAHs. PAHs generally occur as complex mixtures (for example, as part of combustion products such as soot), not as single compounds (71).

Several of the PAHs, including benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno [1,2,3-c,d]pyrene, have caused tumors in laboratory animals when they breathed these substances in the air, when they ate them, or when they had long periods of skin contact with them. Studies of people show that individuals exposed by breathing or skin contact for long periods to mixtures that contain PAHs and other compounds can also develop cancer. Mice fed high levels of benzo[a]pyrene during pregnancy had difficulty reproducing and so did their offspring. The offspring of pregnant mice fed benzo[a]pyrene also showed other harmful effects, such as birth defects and decreased body weight. Similar effects could occur in people, but we have no information to show that these effects do occur (71).

In health assessments, PAHs are typically evaluated as a group because they affect the same health outcomes. The EPA has established 'relative potency factors' that relate the potency of each carcinogenic PAH to the potency of benzo[a]pyrene (56). Relative potency factors are used to weight each PAH according to its potency in evaluation of 'total PAH' toxicity.

In this PHA, PAHs are evaluated because they were measured in Johnson Creek sediment at concentrations above health-based screening levels for soil. While there is no indication that they originated from PCC, they do contribute to the potential health effects of contact with sediment. They are therefore included in the health effects evaluation.

Comparison values for PAHs

- **Inhalation CVs.** PAH's in air were not evaluated in this PHA.
- **Ingestion and dermal contact CVs.** The comparison value used for sediment exposure to PAHs in this PHA is the ATSDR ingestion CREG for the PAH benzo(a)pyrene 0.12 mg/kg (ppm) for a 1 in 1 million lifetime cancer risk. This lifetime cancer risk value is derived from EPA's cancer slope factor for benzo(a)pyrene. The non-cancer effects of benzo(a)pyrene were evaluated against the EPA reference concentration for ingestion of 0.3 ug/kg/day, which is designed to be protective of neurodevelopmental effects of exposure during pregnancy (71).

Polychlorinated Biphenyls. Polychlorinated Biphenyls (PCBs) are a group of synthetic organic chemicals that can cause several different harmful effects. There are no known natural sources of PCBs in the environment. PCBs are either oily liquids or solids and are colorless to light yellow. They have no known smell or taste. PCBs enter the environment as mixtures containing a variety of individual chlorinated biphenyl components, known as congeners, as well as impurities. Once in the environment, PCBs do not readily break down and therefore may remain for very long periods of time. Small amounts of PCBs can be found in almost all outdoor and indoor air, soil, sediments, surface water, and animals. Health effects that have been

associated with exposure to PCBs in humans and/or animals include liver, thyroid, dermal and ocular changes, immunological alterations, neurodevelopmental changes, reduced birth weight, reproductive toxicity, and cancer. Some PCBs can mimic or block the action of hormones from the thyroid and other endocrine glands. Because hormones influence the normal functioning of many organs, some of the effects of PCBs may result from endocrine changes (72).

Studies of workers provide evidence that PCBs were associated with certain types of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate commercial PCB mixtures throughout their lives developed liver cancer. Based on the evidence for cancer in animals, the Department of Health and Human Services (DHHS) has stated that PCBs may reasonably be anticipated to be carcinogens. Both EPA and the International Agency for Research on Cancer (IARC) have determined that PCBs are probably carcinogenic to humans (72).

Children can be exposed to PCBs both prenatally and from breast milk. PCBs are stored in the mother's body and can be released during pregnancy, cross the placenta, and enter fetal tissues. PCBs dissolve readily in fat, meaning they can accumulate in breast milk fat and be transferred to babies and young children. Because the brain, nervous system, immune system, thyroid, and reproductive organs are still developing in the fetus and child, the effects of PCBs on these target systems may be more profound after exposure during the prenatal and neonatal periods, making fetuses and children more susceptible to PCBs than adults (72).

The potential health effects of PCBs are typically evaluated as a group because they affect common health endpoints. In this PHA we add the concentrations of all PCBs detected to determine 'total PCB' concentrations.

Comparison values for PCBs

- **Inhalation CVs.** PCBs in air were not evaluated in the PHA.
- **Ingestion and dermal contact CVs.** The comparison value used for sediment exposure to PCBs in this PHA is the ATSDR ingestion CREG of 0.19 mg/kg (ppm) for a 1 in 1 million lifetime cancer risk. This lifetime cancer risk value is derived from EPA's cancer slope factor for PCBs. The non-cancer effects of PCBs were evaluated against the EPA reference concentration for ingestion of 0.02 ug/kg/day, which is designed to be protective of immunological and developmental effects (72).

Appendix G. Dose and risk calculations

This appendix describes the formulas, methods, and assumptions used to calculate doses of contaminants of concern that may occur under different exposure scenarios. It also presents detailed summaries of health risk calculation results for each scenario. The doses calculated here were used to calculate the risk for people exposed in these scenarios and to determine whether they are at higher risk of illness because of contaminants at or around PCC.

Exposure Dose Calculation Methods

Exposure doses were calculated for each exposure scenario using the equations and assumptions described below.

Dose from exposure to air (chronic exposure)

This formula was used to calculate exposure concentration of metals from inhaling air from the area around PCC:

$$\text{Exposure Concentration} = \frac{CA \times ET \times EF \times ED}{AT}$$

CA	= Chemical-specific 95% UCL of median concentration measured in air (µg/m^3)
ET	= Exposure Time (hours/day)
EF	= Exposure frequency (days/year)
ED	= Exposure duration (years)
AT_{cancer}	= Averaging time for cancer (hours over a 78-year lifetime)
$AT_{non\text{-}cancer}$	= Averaging time for non-cancer (hours over exposure duration)

Dose from exposure to sediment (chronic exposure)

Via ingestion of sediment

This formula was used to calculate exposure doses to PCBs, PAHs and nickel from ingestion of Johnson Creek sediment:

$$\text{Ingested Dose (mg/kg/day)} = \frac{C \times IR \times EF \times CF}{BW}$$

C	= Contaminant concentration in soil or sediment (mg/kg)
IR	= Intake rate of contaminated soil or sediment (mg/day)
EF	= Exposure factor (unitless) = (F x ED)/ AT

F= Exposure frequency (days/year)
ED= Exposure duration (years)
AT_{Cancer}= Averaging Time for cancer (days/78 year lifetime)
$AT_{non\text{-}cancer}$= Averaging Time for non-cancer (days/exposure duration)

CF = Conversion factor (10^{-6} kg/mg)
BW = Body weight (kg)

Via absorption through skin

This formula was used to calculate exposure doses to PCBs, PAHs from skin contact with Johnson Creek sediment:

$$\textbf{Dermal Absorbed Dose (mg/kg/day)} = \frac{\textbf{C x EF x CF x AF X ABS}_{\textbf{d}}\textbf{ x SA}}{\textbf{BW x ABS}_{\textbf{gi}}}$$

C = Contaminant concentration in soil or sediment (mg/kg)
EF = Exposure factor (unitless) = (F x ED)/AT
F= Exposure frequency (days/year)
ED= Exposure duration (years)
AT_{Cancer}= Averaging Time for cancer (days/78 year lifetime)
$AT_{non\text{-}cancer}$= Averaging Time for non-cancer (days/exposure duration)
CF = Conversion factor (10^{-6} kg/mg)
AF = Adherence factor of soil or sediment to skin (mg/cm2)
ABS_d = Dermal absorption fraction
SA = Surface area available for contact
BW = Body weight (kg)
ABS_{gi} = Gastrointestinal absorption

Non-cancer vs. Cancer Averaging Times

Methods for calculating doses for use in assessing non-cancer risk and for cancer risk are identical except the way in which averaging time (AT) is calculated. The rationale for this difference in AT lies in the theory that cancer is the result of multiple defects/mutations in genetic material accumulated over an entire lifetime while non-cancer risks generally occur only when exposure is ongoing.

Non-cancer averaging time is limited to the duration of the exposure:

$AT_{non\text{-}cancer}$ = Exposure duration (years) x 365 (days/year) x 24 (hours/day)

Cancer averaging time represents an entire statistical lifetime (78 years) for agents that cause cancer.

AT_{cancer} = 78 (years/lifetime) x 365 (days/year) x 24 (hours/day) = 683,280 hours

Health Risk Calculation Methods

Once exposure doses were calculated for each exposure pathway, health risks were evaluated for cancer and non-cancer effects using the following equations.

Cancer risk calculation:

For cancer-causing chemicals, EPA uses evidence from scientific research to estimate the amount of increased lifetime cancer risk associated with each additional unit of exposure. These estimates are known as Cancer Slope Factors (CSF) for chemicals ingested or absorbed through skin and Inhalation Unit Risks (IUR) for chemicals in air.

Cancer risk is calculated separately for each age group (i.e., birth to <1 year, 1 to <2 years, 2 to <6 years, 6 to <11, 11 to <16 years, 16 to <21 years, ≥21 years) based on age-specific exposure factors (e.g., body weight, soil ingestion rate, etc.). For example, children consume more soil than adults so daily intake of soil or sediment is assumed to be higher for early life exposures. Lifetime cancer risk from many years of exposure is calculated by adding together cancer risks of all age ranges. This approach provides a lifetime cancer risk that accounts for changes in exposure that occur over a lifetime.

In addition, cancer risk for children was weighted by age for hexavalent chromium and for PAHs because they cause cancer by what is known as "mutagenic mode of action." Mutagenic chemicals are those that can make multiple changes to genes in a cell. For children, mutagens pose a higher risk of cancer when exposures occur early in life. Age-dependent adjustment factors (ADAFs) were applied to reflect the potential for early-life exposure to mutagens to make a greater contribution to lifetime cancer risk (51; 73). For exposures before 2 years of age, a 10-fold adjustment was made. For exposures between 2 and <16 years of age, a 3-fold adjustment was made. For exposures after turning 16 years of age, no further adjustment was made.

Cancer risk equations

Cancer risk from exposure to a chemical during specific age ranges was calculated with the following equations:

For exposure through ingestion or dermal absorption:

$$\textbf{Cancer Risk} = \textbf{Dose (mg/kg/day)} \times \textbf{CSF (mg/kg/day)}^{-1}$$

For exposure through inhalation:

$$\textbf{Cancer Risk} = \textbf{EC } (\mu g/m^3) \times \textbf{IUR } (\mu g/m^3)^{-1}$$

For chemicals with a mutagenic mode of action:

Cancer Risk = Dose (mg/kg/day) x CSF (mg/kg/day)$^{-1}$ x ADAF

Where:

CSF= Cancer Slope Factor
IUR= Inhalation Unit Risk
EC = Exposure Concentration (in air)
ADAF = Age-dependent Adjustment Factor (for mutagens)

Cancer risk from exposure throughout multiple life stages is calculated as the sum of cancer risk from exposure at each phase.

Lifetime Cancer Risk for an individual chemical across all ages of exposure

= Cancer Risk$_{age0\text{-}1}$ + Cancer Risk$_{age1\text{-}2}$ + Cancer Risk$_{age\ 2\text{-}6}$ …etc.

Cumulative cancer risk across multiple chemicals in a pathway was calculated as the sum of cancer risks from each chemical.

Cumulative lifetime cancer risk across multiple chemicals in a pathway

= Cancer Risk$_{chemical\ A}$ + Cancer Risk$_{chemical\ B}$ …etc.

When exposure to cancer-causing chemicals occurred through multiple pathways, aggregate cancer risk was calculated as the sum of cumulative lifetime cancer risks calculated for each pathway.

Aggregate lifetime cancer risk across pathways

= Cancer Risk$_{ingestion}$ + Cancer Risk$_{skin\ absorption}$

Non-cancer risk calculation:

Non-cancer risk is evaluated by comparing calculated exposure doses with health-based guideline concentrations identified by authoritative bodies like EPA and ATSDR. A health guideline is the daily dose of a chemical, below which scientists consider it unlikely to harm people's health. Non-cancer risk is described by hazard quotients, which are the ratio of air concentrations over health guidelines.

$$\textbf{Hazard Quotient} = \frac{\textbf{Time Adjusted Air Concentration}}{\textbf{Health Guideline (MRL, RfD, or RfC)}}$$

A hazard quotient less than one indicates that the sensitive health effects used as the basis for health guideline values are not expected to occur at the predicted dose. A hazard quotient

greater than one requires further investigation. Because health guidelines for different chemicals are based on different health outcomes of varying severity and incorporate different levels of uncertainty, the risk associated with hazard quotients above one are evaluated on a chemical by chemical basis.

Potential for cumulative non-cancer risks is calculated by adding together hazard quotients for each chemical with similar non-cancer effects. The sum of hazard quotients is known as the hazard index.

$$\textbf{Hazard Index} = HQ_{\text{chemical A}} + HQ_{\text{chemical B}} + HQ_{\text{chemical C}} \ldots \textbf{etc.}$$

In this health assessment, EHAP did not calculate any hazard indexes because nickel was the only chemical of concern identified for non-cancer health outcomes

Detailed Summary of Risk Calculation Results

This section presents details of risk calculations and results for each exposure scenario. Final risk estimates are rounded to a single significant figure to reflect the imprecise nature of risk calculations. Because final numbers presented here are rounded, risk estimates summed across life stages, chemicals, and pathways may not be exactly equal to the sum of risks calculated for individual components of total risk.

Exposure Scenario 2- Long-term resident with exposure to air concentrations detected in 2016 prior to HEPA filter installation

Table G1. Summary of cancer and non-cancer risk under air conditions prior to HEPA-filter installation

Monitoring Location	Scenario	Exposure Assumptions	Cumulative Cancer Risk[A] of Ni, As, Cd, Cr 6+	Hazard Quotient for non-cancer risk from Ni
Milwaukie Johnson Creek	Child	constant exposure for 21 years	7 in 1,000,000	0.35
	Adult	constant exposure for 38 years	8 in 1,000,000	0.35
	Lifetime	constant exposure for 21 years as a child and 38 years as an adult	20 in 1,000,000	0.35
S.E. 45th and Harney Drive	Child	constant exposure for 21 years	3 in 1,000,000	NA
	Adult	constant exposure for 38 years	4 in 1,000,000	NA
	Lifetime	constant exposure for 21 years as a child and 38 years as an adult	7 in 1,000,000	NA
S.E. Harney Drive	Child	constant exposure for 21 years	4 in 1,000,000	NA
	Adult	constant exposure for 38 years	5 in 1,000,000	NA
	Lifetime	constant exposure for 21 years as a child and 38 years as an adult	9 in 1,000,000	NA

[A]Cancer risk is rounded to a single significant digit.

Table. G2. Cancer risk calculation details- 45th and Harney Drive monitor (air conditions in the month prior to HEPA filter installation)

	Child						Adult				Lifetime[B]
	Nickel	Arsenic	Cadmium	Hexavalent Chromium (0-2 yrs)	Hexavalent Chromium (2-16 yrs)	Hexavalent Chromium (16-21yrs)	Nickel	Arsenic	Cadmium	Hexavalent Chromium	
Air Concentration[A] (ug/m^3)	0.01125	0.000874	0.000124	0.00015	0.00015	0.00015	0.01125	0.000874	0.000124	0.00015	
Exposure Time (hours)	24	24	24	24	24	24	24	24	24	24	
Exposure Factor (days/year)	365	365	365	365	365	365	365	365	365	365	
Exposure Duration (years)	21	21	21	2	14	5	38	38	38	38	
Averaging Time $_{cancer}$ (hours)	683280	683280	683280	683280	683280	683280	683280	683280	683280	683280	
Exposure Concentration (ug/m^3)	0.003029	0.000235	0.000033	0.000012	0.000004	0.000025	0.005481	0.000426	0.000060	0.000073	
Inhalation Unit Risk $(\mu g/m^3)^{-1}$	2.40E-04	4.30E-03	1.80E-03	1.20E-02	1.20E-02	1.20E-02	2.40E-04	4.30E-03	1.80E-03	1.20E-02	
Age-dependent Adjustment Factor (ADAF) for mutagens	NA	NA	NA	10	3	1	NA	NA	NA	NA	
Estimated Cancer Risk	**7.27E-07**	**1.01E-06**	**6.01E-08**	**4.62E-07**	**9.69E-07**	**1.15E-07**	**1.32E-06**	**1.83E-06**	**1.09E-07**	**8.77E-07**	**7.48E-06**

[A]95% UCL of median concentration measured in air ($\mu g/m^3$)

[B]Cumulative lifetime risk for all chemicals

EC= (CA x ET x EF x ED)/AT

Cancer Risk = EC ($\mu g/m^3$) x IUR $(\mu g/m^3)^{-1}$

Table G3. Cancer risk calculation details - Milwaukie Johnson Creek monitor (air conditions in the month prior to HEPA filter installation)

	Child						Adult				Lifetime[B]
	Nickel	Arsenic	Cadmium	Hexavalent Chromium (0-2 yrs)	Hexavalent Chromium (2-16 yrs)	Hexavalent Chromium (16-21yrs)	Nickel	Arsenic	Cadmium	Hexavalent Chromium	
Air Concentration[A] ($\mu g/m^3$)	0.03168	0.001049	0.000173	0.000386	0.000386	0.000386	0.03168	0.001049	0.000154	0.000386	
Exposure Time (hours)	24	24	24	24	24	24	24	24	24	24	
Exposure Factor (days/year)	365	365	365	365	365	365	365	365	365	365	
Exposure Duration (years)	21	21	21	2	14	5	38	38	38	38	
Averaging Time $_{cancer}$ (hours)	683280	683280	683280	683280	683280	683280	683280	683280	683280	683280	
Exposure Concentration (ug/m^3)	0.00853	0.00028	0.00004	0.00001	0.00006	0.00003	0.01543	0.00051	0.00008	0.00019	
Inhalation Unit Risk $(\mu g/m^3)^{-1}$	2.40E-04	4.30E-03	1.80E-03	1.20E-02	1.20E-02	1.20E-02	2.40E-04	4.30E-03	1.80E-03	1.20E-02	
Age-dependent Adjustment Factor (ADAF) for mutagens				10	3	1					
Estimated Cancer Risk	**2.0E-06**	**1.2E-06**	**8.4E-08**	**1.2E-06**	**2.5E-06**	**3.0E-07**	**3.7E-06**	**2.2E-06**	**1.4E-07**	**2.3E-06**	**1.6E-05**

[A]95% UCL of median concentration measured in air ($\mu g/m^3$)

[B]Cumulative lifetime risk for all chemicals

EC= (CA x ET x EF x ED)/AT

Cancer Risk = EC ($\mu g/m^3$) x IUR $(\mu g/m^3)^{-1}$

Table G4. Cancer risk calculation details – S.E. Harney Drive monitor (air conditions in the month prior to HEPA filter installation)

	Child						Adult				Lifetime[B]
	Nickel	Arsenic	Cadmium	Hexavalent Chromium (0-2 yrs)	Hexavalent Chromium (2-16 yrs)	Hexavalent Chromium (16-21yrs)	Nickel	Arsenic	Cadmium	Hexavalent Chromium	
Air Concentration[A] ($\mu g/m^3$)	0.01241	0.000959	0.000207	0.000227	0.000227	0.000227	0.01241	0.000959	0.000207	0.000227	
Exposure Time (hours)	24	24	24	24	24	24	24	24	24	24	
Exposure Factor (days/year)	365	365	365	365	365	365	365	365	365	365	
Exposure Duration (years)	21	21	21	2	14	5	38	38	38	38	
Averaging Time $_{cancer}$ (hours)	683280	683280	683280	683280	683280	683280	683280	683280	683280	683280	
Exposure Concentration (ug/m^3)	0.00334	0.00026	0.00006	0.00001	0.00004	0.00002	0.00605	0.00047	0.00010	0.00011	
Inhalation Unit Risk $(\mu g/m^3)^{-1}$	2.40E-04	4.30E-03	1.80E-03	1.20E-02	1.20E-02	1.20E-02	2.40E-04	4.30E-03	1.80E-03	1.20E-02	
Age-dependent Adjustment Factor (ADAF) for mutagens				10	3	1					
Estimated Cancer Risk	**8.0E-07**	**1.1E-06**	**1.0E-07**	**7.0E-07**	**1.5E-06**	**1.7E-07**	**1.5E-06**	**2.0E-06**	**1.8E-07**	**1.3E-06**	**9.3E-06**

[A]95% UCL of median concentration measured in air ($\mu g/m^3$)

[B]Cumulative lifetime risk for all chemicals

EC= (CA x ET x EF x ED)/AT

Cancer Risk = EC ($\mu g/m^3$) x IUR $(\mu g/m^3)^{-1}$

Table G5. Non-cancer risk calculation details for Nickel- Milwaukie Johnson Creek monitor[A] (air conditions in the month prior to HEPA filter installation)

	Child	Adult	Lifetime
Nickel Air Concentration[B] (ug/m^3)	0.03168	0.03168	0.03168
Exposure Time (hours)	24	24	24
Exposure Factor (days/year)	365	365	365
Exposure Duration (years)	21	57	78
Averaging Time $_{non\text{-}cancer}$ (hours)	183960	499320	683280
Exposure Concentration	0.03168	0.03168	0.03168
Non-cancer Comparison Value for nickel	0.09	0.09	0.09
Chronic Non-cancer Risk	**0.35**	**0.35**	**0.35**

[A]Non-cancer risk was only calculated for nickel concentrations detected at the Milwaukie Johnson Creek monitor prior to installation of HEPA-filters because nickel concentrations were below comparison values at all other monitoring locations

[B]95% UCL of median concentration measured in air (µg/m^3)

EC = (CA x ET x EF x ED)/AT

Non-cancer Risk = EC/CV

Exposure Scenario 3. Long-term resident with exposure to air under current conditions

Table G6. Summary of cancer and non-cancer risk under current air conditions

Monitoring Location	Scenario	Exposure Assumptions	Cumulative Cancer Risk[A] of Ni, As, Cd, Cr 6+	Hazard Quotient for non-cancer risk from Ni
Milwaukie Johnson Creek	Child	constant exposure for 21 years	5 in 1,000,000	NA
	Adult	constant exposure for 57 years	7 in 1,000,000	NA
	Lifetime	constant exposure for 21 years as a child and 57 years as an adult	10 in 1,000,000	NA
S.E. 45th and Harney Drive	Child	constant exposure for 21 years	2 in 1,000,000	NA
	Adult	constant exposure for 57 years	4 in 1,000,000	NA
	Lifetime	constant exposure for 21 years as a child and 57 years as an adult	6 in 1,000,000	NA
S.E. Harney Drive	Child	constant exposure for 21 years	4 in 1,000,000	NA
	Adult	constant exposure for 57 years	8 in 1,000,000	NA
	Lifetime	constant exposure for 21 years as a child and 57 years as an adult	10 in 1,000,000	NA

[A]Cancer risk is rounded to a single significant digit.

Table G7. Cancer risk calculation details - 45th and Harney Drive monitor (current air conditions)

	Child						Adult				Lifetime[B]
	Nickel	Arsenic	Cadmium	Hexavalent Chromium (0-2 yrs)	Hexavalent Chromium (2-16 yrs)	Hexavalent Chromium (>16yrs)	Nickel	Arsenic	Cadmium	Hexavalent Chromium	
Air Concentration[A] (ug/m^3)	0.000875	0.000919	0.000214	0.0000827	0.0000827	0.0000827	0.000875	0.000919	0.000214	0.0000827	
Exposure Time (hours)	24	24	24	24	24	24	24	24	24	24	
Exposure Factor (days/year)	365	365	365	365	365	365	365	365	365	365	
Exposure Duration (years)	21	21	21	2	14	5	57	57	57	57	
Averaging Time $_{cancer}$ (hours)	683280	683280	683280	683280	683280	683280	683280	683280	683280	683280	
Exposure Concentration (ug/m^3)	0.000236	0.000247	0.000058	0.000002	0.000014	0.000022	0.000639	0.000672	0.000156	0.000060	
Inhalation Unit Risk $(\mu g/m^3)^{-1}$	0.00024	0.0043	0.0018	1.20E-02	1.20E-02	0.012	0.00024	0.0043	0.0018	0.012	
Age-dependent Adjustment Factor (ADAF) for mutagens	NA	NA	NA	10	3	1	NA	NA	NA	NA	
Estimated Cancer Risk	**5.7E-08**	**1.1E-06**	**1.0E-07**	**2.5E-07**	**5.3E-07**	**6.4E-08**	**1.5E-07**	**2.9E-06**	**2.8E-07**	**7.3E-07**	**6.1E-06**

[A]95% UCL of median concentration measured in air ($\mu g/m^3$)

[B]Cumulative lifetime risk for all chemicals

EC= (CA x ET x EF x ED)/AT

Cancer Risk = EC ($\mu g/m^3$) x IUR $(\mu g/m^3)^{-1}$

Table G8. Cancer risk calculation details – Milwaukie Johnson Creek monitor (current air conditions)

	Child						Adult				Lifetime[B]
	Nickel	Arsenic	Cadmium	Hexavalent Chromium (0-2 yrs)	Hexavalent Chromium (2-16 yrs)	Hexavalent Chromium (>16yrs)	Nickel	Arsenic	Cadmium	Hexavalent Chromium	
Air Concentration[A] (ug/m^3)	0.01103	0.000618	0.000131	0.000367	0.000367	0.000367	0.01103	0.000618	0.000131	0.000367	
Exposure Time (hours)	24	24	24	24	24	24	24	24	24	24	
Exposure Factor (days/year)	365	365	365	365	365	365	365	365	365	365	
Exposure Duration (years)	21	21	21	2	14	5	57	57	57	57	
Averaging Time $_{cancer}$ (hours)	683280	683280	683280	683280	683280	683280	683280	683280	683280	683280	
Exposure Concentration (ug/m^3)	0.002970	0.000166	0.000035	0.000009	0.000061	0.000028	0.008060	0.000452	0.000096	0.000268	
Inhalation Unit Risk (µg/m^3)$^{-1}$	0.00024	0.0043	0.0018	0.012	0.012	0.012	0.00024	0.0043	0.0018	0.012	
Age-dependent Adjustment Factor (ADAF) for mutagens	NA	NA	NA	10	3	1	NA	NA	NA	NA	
Estimated Cancer Risk	**7.1E-07**	**7.2E-07**	**6.3E-08**	**1.1E-06**	**2.4E-06**	**2.8E-07**	**1.9E-06**	**1.9E-06**	**1.7E-07**	**3.2E-06**	**1.3E-05**

[A]95% UCL of median concentration measured in air (µg/m^3)

[B]Cumulative lifetime risk for all chemicals

EC= (CA x ET x EF x ED)/AT

Cancer Risk = EC (µg/m^3) x IUR (µg/m^3)$^{-1}$

Table G9. Cancer risk calculation details – S.E. Harney Drive monitor (current air conditions)

	Child						Adult				Lifetime[B]
	Nickel	Arsenic	Cadmium	Hexavalent Chromium (0-2 yrs)	Hexavalent Chromium (2-16 yrs)	Hexavalent Chromium (>16yrs)	Nickel	Arsenic	Cadmium	Hexavalent Chromium	
Air Concentration[A] (ug/m^3)	0.003677	0.000645	0.003155	0.00013	0.00013	0.00013	0.003677	0.000645	0.003155	0.00013	
Exposure Time (hours)	24	24	24	24	24	24	24	24	24	24	
Exposure Factor (days/year)	365	365	365	365	365	365	365	365	365	365	
Exposure Duration (years)	21	21	21	2	14	5	57	57	57	57	
Averaging Time $_{cancer}$ (hours)	683280	683280	683280	683280	683280	683280	683280	683280	683280	683280	
Exposure Concentration (ug/m^3)	0.000990	0.000174	0.000849	0.000003	0.000022	0.000010	0.002687	0.000471	0.002306	0.000095	
Inhalation Unit Risk $(\mu g/m^3)^{-1}$	0.00024	0.0043	0.0018	0.012	0.012	0.012	0.00024	0.0043	0.0018	0.012	
Age-dependent Adjustment Factor (ADAF) for mutagens	NA	NA	NA	10	3	1	NA	NA	NA	NA	
Estimated Cancer Risk	**2.4E-07**	**7.5E-07**	**1.5E-06**	**4.0E-07**	**8.4E-07**	**1.0E-07**	**6.4E-07**	**2.0E-06**	**4.2E-06**	**1.1E-06**	**1.2E-05**

[A]95% UCL of median concentration measured in air ($\mu g/m^3$)

[B]Cumulative lifetime risk for all chemicals

EC= (CA x ET x EF x ED)/AT

Cancer Risk = EC ($\mu g/m^3$) x IUR $(\mu g/m^3)^{-1}$

Exposure Scenario 4. Contact with contaminated Johnson Creek sediment

Table G10. Summary of estimated cumulative cancer risk from exposure to PCBs and PAHs in sediment through ingestion and absorption through skin (assuming *weekly* year-round exposure)

Exposure Period	Cumulative cancer risk from dermal contact with PCBs and PAHs in sediment[A]	Cumulative cancer risk from ingestion of PCBs and PAHs in sediment[A]	Aggregate cumulative cancer risk from dermal contact and ingestion of PCBs and PAHs in sediment[A]
Child 6 wks to < 1 yr	NA	NA	NA
Child 1 to < 2 yr	4 in 1,000,000	0.1 in 1,000,000	5 in 1,000,000
Child 2 to < 6 yr	6 in 1,000,000	0.2 in 1,000,000	6 in 1,000,000
Child 6 to < 11 yr	6 in 1,000,000	0.1 in 1,000,000	6 in 1,000,000
Child 11 to <16 yr	5 in 1,000,000	0.06 in 1,000,000	5 in 1,000,000
Child 16 to <21 yr	3 in 1,000,000	0.03 in 1,000,000	3 in 1,000,000
Cumulative Child 0-21 years	20 in 1,000,000	0.5 in 1,000,000	30 in 1,000,000
Adult for 33 years (95% residential occupancy period)	20 in 1,000,000	0.1 in 1,000,000	20 in 1,000,000
Lifetime (21 years of childhood exposure plus 33 years of adult exposure)	40 in 1,000,000	0.6 in 1,000,000	40 in 1,000,000

[A]Cancer risk is rounded to a single significant digit.

Table G11. Summary of estimated cumulative cancer risk from exposure to PCBs and PAHs in sediment through ingestion and absorption through skin (assuming *daily* year-round exposure)

Exposure Period	Cumulative cancer risk from dermal contact with PCBs and PAHs in sediment[A]	Cumulative cancer risk from ingestion of PCBs and PAHs in sediment[A]	Aggregate cumulative cancer risk from dermal contact and ingestion of PCBs and PAHs in sediment[A]
Child 6 wks to < 1 yr	NA	NA	NA
Child 1 to < 2 yr	30 in 1,000,000	1 in 1,000,000	30 in 1,000,000
Child 2 to < 6 yr	40 in 1,000,000	1 in 1,000,000	40 in 1,000,000
Child 6 to < 11 yr	40 in 1,000,000	0.8 in 1,000,000	40 in 1,000,000
Child 11 to <16 yr	30 in 1,000,000	0.4 in 1,000,000	30 in 1,000,000
Child 16 to <21 yr	20 in 1,000,000	0.2 in 1,000,000	20 in 1,000,000
Adult CR (33 years) -- 95% residential occupancy period	100 in 1,000,000	0.7 in 1,000,000	100 in 1,000,000
Lifetime (21 years of childhood exposure plus 33 years of adult exposure)	300 in 1,000,000	4 in 1,000,000	300 in 1,000,000

[A]Cancer risk is rounded to a single significant digit.

Table G13. Cancer risk calculation details- Absorption of total PCBs through skin (assuming *weekly* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (mg/kg)	0.48	0.48	0.48	0.48	0.48	0.48	
EF	Exposure factor (unit less)= (F x ED)/ATc	0.0018	0.0073	0.0092	0.0092	0.0092	0.0604	
F	Frequency of Exposure (days/week x weeks/year)	52.14	52.14	52.14	52.14	52.14	52.14	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
AT_{cancer}	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor (10^{-6} kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
AF	Adherence factor of soil or sediment to skin (mg/cm^2) based on geometric mean mud adherence for 9-14 year-olds	21	21	21	21	21	21	
ABS_{dermal}	Chemical-specific dermal absorption fraction	0.14	0.14	0.14	0.14	0.14	0.14	
SA	Surface area available for contact -head, hands, forearms, lower legs, and feet (cm^2)	2,299	2,592	3,824	5,454	6,083	6,030	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
ABS_{gi}	Gastrointestinal absorption	1	1	1	1	1	1	
D	**Dermal absorbed dose (mg/kg/day)**	5.2E-07	1.5E-06	1.6E-06	1.2E-06	1.1E-06	6.4E-06	
ADAF	Age Dependent Adjustment Factor for mutagens (unitless)	NA	NA	NA	NA	NA	NA	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	2	2	2	2	2	2	
Risk	**Estimated cancer risk from stage-specific exposures**	1.0E-06	3.1E-06	3.1E-06	2.5E-06	2.2E-06	1.3E-05	2.5E-05

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x EF x CF x AF x ABSdermal xSA)/(BW x ABSgi)

Risk= D x CSF x ADAF

Table G14. Cancer risk calculation details- Absorption of total PAHs through skin (assuming weekly year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (mg/kg)	0.336	0.336	0.336	0.336	0.336	0.336	
EF	Exposure factor (unit less)= (F x ED)/ATc	0.00183	0.00733	0.00916	0.00916	0.00916	0.06044	
F	Frequency of Exposure (days/week x weeks/year)	52.14	52.14	52.14	52.14	52.14	52.14	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
AT_{cancer}	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor (10^{-6} kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
AF	Adherence factor of soil or sediment to skin (mg/cm^2) based on geometric mean mud adherence for 9-14 year-olds	21	21	21	21	21	21	
ABS_{dermal}	Chemical-specific dermal absorption fraction	0.13	0.13	0.13	0.13	0.13	0.13	
SA	Surface area available for contact -head, hands, forearms, lower legs, and feet (cm^2)	2,299	2,592	3,824	5,454	6,083	6,030	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
ABS_{gi}	Gastrointestinal absorption	1	1	1	1	1	1	
D	**Dermal absorbed dose (mg/kg/day)**	3.4E-07	1.0E-06	1.0E-06	8.1E-07	7.1E-07	4.2E-06	
ADAF	Age Dependent Adjustment Factor for mutagens (unitless)	10	3	3	3	1	1	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	1	1	1	1	1	1	
Risk	**Estimated cancer risk from stage-specific exposures**	3.4E-06	3.0E-06	3.0E-06	2.4E-06	7.1E-07	4.2E-06	1.7E-05

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x EF x CF x AF x ABSdermal xSA)/(BW x ABSgi)

Risk= D x CSF x ADAF

Table G15. Estimated cumulative cancer risk from absorption of PCBs and PAHs in sediment through skin (assuming *weekly* year-round exposure)

Exposure Period	Total PCBs	Total PAHs	Sum for all chemicals
Child 6 wks to < 1 yr	NA	NA	NA
Child 1 to < 2 yr	1 in 1,000,000	3 in 1,000,000	4 in 1,000,000
Child 2 to < 6 yr	3 in 1,000,000	3 in 1,000,000	6 in 1,000,000
Child 6 to < 11 yr	3 in 1,000,000	3 in 1,000,000	6 in 1,000,000
Child 11 to <16 yr	3 in 1,000,000	2 in 1,000,000	5 in 1,000,000
Child 16 to <21 yr	2 in 1,000,000	0.7 in 1,000,000	3 in 1,000,000
Adult	10 in 1,000,000	4 in 1,000,000	20 in 1,000,000
Lifetime	30 in 1,000,000	20 in 1,000,000	40 in 1,000,000

Table G16. Cancer risk calculation details- Absorption of total PCBs through skin (assuming *daily* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (mg/kg)	0.48	0.48	0.48	0.48	0.48	0.48	
EF	Exposure factor (unit less)= (F x ED)/ATc	0.013	0.051	0.064	0.064	0.064	0.423	
F	Frequency of Exposure (days/week x weeks/year)	365	365	365	365	365	365	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
AT_{cancer}	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor (10^{-6} kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
AF	Adherence factor of soil or sediment to skin (mg/cm^2) based on geometric mean mud adherence for 9-14 year-olds	21	21	21	21	21	21	
ABS_{dermal}	Chemical-specific dermal absorption fraction	0.14	0.14	0.14	0.14	0.14	0.14	
SA	Surface area available for contact -head, hands, forearms, lower legs, and feet (cm^2)	2,299	2,592	3,824	5,454	6,083	6,030	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
ABS_{gi}	Gastrointestinal absorption	1	1	1	1	1	1	
D	**Dermal absorbed dose (mg/kg/day)**	**3.6E-06**	**1.1E-05**	**1.1E-05**	**8.7E-06**	**7.7E-06**	**4.5E-05**	
ADAF	Age Dependent Adjustment Factor for mutagens (unitless)	NA	NA	NA	NA	NA	NA	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	2	2	2	2	2	2	

Risk	**Estimated cancer risk from stage-specific exposures**	**7.3E-06**	**2.2E-05**	**2.2E-05**	**1.7E-05**	**1.5E-05**	**9.0E-05**	**1.7E-04**

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x EF x CF x AF x ABSdermal xSA)/(BW x ABSgi)

Risk= D x CSF x ADAF

Table G17. Cancer risk calculation details- Absorption of total PAHs through skin (assuming *daily* year-round exposure)

	Description	**Child 1 to < 2 yr**	**Child 2 to < 6 yr**	**Child 6 to < 11 yr**	**Child 11 to <16 yr**	**Child 16 to <21 yr**	**Adult**	**Lifetime Exposure[B]**
C	Contaminant concentration[A] (mg/kg)	0.336	0.336	0.336	0.336	0.336	0.336	
EF	Exposure factor (unit less)= (F x ED)/ATc	0.013	0.051	0.064	0.064	0.064	0.423	
F	Frequency of Exposure (days/week x weeks/year)	365	365	365	365	365	365	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
AT_{cancer}	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor (10^{-6} kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
AF	Adherence factor of soil or sediment to skin (mg/cm^2) based on geometric mean mud adherence for 9-14 year-olds	21	21	21	21	21	21	
ABS_{dermal}	Chemical-specific dermal absorption fraction	0.13	0.13	0.13	0.13	0.13	0.13	
SA	Surface area available for contact -head, hands, forearms, lower legs, and feet (cm^2)	2,299	2,592	3,824	5,454	6,083	6,030	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
ABS_{gi}	Gastrointestinal absorption	1	1	1	1	1	1	
D	**Dermal absorbed dose (mg/kg/day)**	2.4E-06	7.0E-06	7.1E-06	5.6E-06	5.0E-06	2.9E-05	
ADAF	Age Dependent Adjustment Factor for mutagens (unitless)	10	3	3	3	1	1	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	1	1	1	1	1	1	
Risk	**Estimated cancer risk from stage-specific exposures**	**2.4E-05**	**2.1E-05**	**2.1E-05**	**1.7E-05**	**5.0E-06**	**2.9E-05**	**1.3E-04**

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x EF x CF x AF x ABSdermal xSA)/(BW x ABSgi)

Risk= D x CSF x ADAF

Table G18. Estimated cumulative cancer risk from absorption of PCBs and PAHs in sediment through skin (assuming *daily* year-round exposure)

Exposure Period	Total PCBs	Total PAHs	Sum for all chemicals[A]
Child 6 wks to < 1 yr	NA	NA	NA
Child 1 to < 2 yr	7 in 1 ,000,000	20 in 1,000,000	30 in 1,000,000
Child 2 to < 6 yr	20 in 1,000,000	20 in 1,000,000	40 in 1,000,000
Child 6 to < 11 yr	20 in 1,000,000	20 in 1,000,000	40 in 1,000,000
Child 11 to <16 yr	20 in 1,000,000	20 in 1,000,000	30 in 1,000,000
Child 16 to <21 yr	20 in 1,000,000	5 in 1,000,000	20 in 1,000,000
Adult	90 in 1,000,000	30 in 1,000,000	100 in 1,000,000
Lifetime	200 in 1,000,000	100 in 1,000,000	300 in 1,000,000

[A]Cancer risk is rounded to a single significant digit.

Table G19. Cancer risk calculation details- Ingestion of total PCBs in sediment (assuming *weekly* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (ppm or mg/kg)	0.48	0.48	0.48	0.48	0.48	0.48	
IR	Intake rate - upper percentile of daily soil intake for each age group (mg/day)	200	200	200	200	200	100	
EF	Exposure factor= (F x ED)/AT	0.0018	0.0073	0.0092	0.0092	0.0092	0.0604	
F	Frequency of Exposure (days/week x weeks/year)	52	52	52	52	52	52	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
ATc	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor 0.000001 (kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
D	Estimated dose rate from the above equation (mg/kg/day)	1.5E-08	4.0E-08	2.8E-08	1.5E-08	1.2E-08	3.6E-08	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	2	2	2	2	2	2	
ADAF	Age-dependent adjustment factor- *for mutagenic chemicals only* (unitless)	NA	NA	NA	NA	NA	NA	
Risk	Estimated cancer risk from stage-specific exposures	3.1E-08	8.1E-08	5.5E-08	3.1E-08	2.5E-08	7.3E-08	3.0E-07

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x IR x EF x CF) / BW

Risk= D x CSF x ADAF

Table G20. Cancer risk calculation details- Ingestion of total PAHs in sediment (assuming *weekly* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (ppm or mg/kg)	0.336	0.336	0.336	0.336	0.336	0.336	
IR	Intake rate - upper percentile of daily soil intake for each age group (mg/day)	200	200	200	200	200	100	
EF	Exposure factor= (F x ED)/AT	0.0018	0.0073	0.0092	0.0092	0.0092	0.0604	
F	Frequency of Exposure (days/week x weeks/year)	52	52	52	52	52	52	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
ATc	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor 0.000001 (kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
D	Estimated dose rate from the above equation (mg/kg/day)	1.1E-08	2.8E-08	1.9E-08	1.1E-08	8.6E-09	2.5E-08	
CSF	Cancer Slope Factor (mg/kg/day)$^{-1}$	1	1	1	1	1	1	
ADAF	Age-dependent adjustment factor- *for mutagenic chemicals only* (unitless)	10	3	3	3	1	1	
Risk	Estimated cancer risk from stage-specific exposures	1.1E-07	8.5E-08	5.8E-08	3.3E-08	8.6E-09	2.5E-08	3.2E-07

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x IR x EF x CF) / BW

Risk= D x CSF x ADAF

Table G21. Estimated cumulative cancer risk from exposure to PCBs and PAHs in sediment through ingestion (assuming *weekly* year-round exposure)

Exposure Period	Total PCBs	Total PAHs	Sum for all chemicals[A]
Child 6 wks to < 1 yr	NA	NA	NA
Child 1 to < 2 yr	0.03 in 1,000,000	0.1 in 1,000,000	0.1 in 1,000,000
Child 2 to < 6 yr	0.08 in 1,000,000	0.08 in 1,000,000	0.2 in 1,000,000
Child 6 to < 11 yr	0.06 in 1,000,000	0.06 in 1,000,000	0.1 in 1,000,000
Child 11 to <16 yr	0.03 in 1,000,000	0.03 in 1,000,000	0.06 in 1,000,000
Child 16 to <21 yr	0.02 in 1,000,000	0.009 in 1,000,000	0.03 in 1,000,000
cumulative Child for 21 years	0.2 in 1,000,000	0.3 in 1,000,000	0.5 in 1,000,000
Adult CR (33 years) -- 95% residential occupancy period	0.07 in 1,000,000	0.02 in 1,000,000	0.1 in 1,000,000
Lifetime	0.3 in 1,000,000	0.3 in 1,000,000	0.6 in 1,000,000

[A]Cancer risk is rounded to a single significant digit.

Table G22. Cancer risk calculation details- Ingestion of total PCBs in sediment (assuming *daily* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (ppm or mg/kg)	0.48	0.48	0.48	0.48	0.48	0.48	
IR	Intake rate - upper percentile of daily soil intake for each age group (mg/day)	200	200	200	200	200	100	
EF	Exposure factor= (F x ED)/AT	0.013	0.051	0.064	0.064	0.064	0.423	
F	Frequency of Exposure (days/week x weeks/year)	365	365	365	365	365	365	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
ATc	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor 0.000001 (kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
D	Estimated dose rate from the above equation (mg/kg/day)	1.1E-07	2.8E-07	1.9E-07	1.1E-07	8.6E-08	2.5E-07	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	2	2	2	2	2	2	
ADAF	Age-dependent adjustment factor- *for mutagenic chemicals only* (unitless)	NA	NA	NA	NA	NA	NA	
Risk	Estimated cancer risk from stage-specific exposures	2.2E-07	5.7E-07	3.9E-07	2.2E-07	1.7E-07	5.1E-07	2.1E-06

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x IR x EF x CF) / BW

Risk= D x CSF x ADAF

Table G23. Cancer risk calculation details- Ingestion of total PAHs in sediment (assuming *daily* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult	Lifetime Exposure[B]
C	Contaminant concentration[A] (ppm or mg/kg)	0.336	0.336	0.336	0.336	0.336	0.336	
IR	Intake rate - upper percentile of daily soil intake for each age group (mg/day)	200	200	200	200	200	100	
EF	Exposure factor= (F x ED)/AT	0.0128	0.0513	0.0641	0.0641	0.0641	0.4231	
F	Frequency of Exposure (days/week x weeks/year)	365	365	365	365	365	365	
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33	
ATc	Cancer averaging time (days/78 year lifetime)	28468	28468	28468	28468	28468	28468	
CF	Conversion factor 0.000001 (kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001	
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80	
D	Estimated dose rate from the above equation (mg/kg/day)	7.6E-08	2.0E-07	1.4E-07	7.6E-08	6.0E-08	1.8E-07	
CSF	Cancer Slope Factor $(mg/kg/day)^{-1}$	1	1	1	1	1	1	
ADAF	Age-dependent adjustment factor- *for mutagenic chemicals only* (unitless)	10	3	3	3	1	1	
Risk	Estimated cancer risk from stage-specific exposures	7.6E-07	5.9E-07	4.1E-07	2.3E-07	6.0E-08	1.8E-07	2.2E-06

[A]Maximum total PCB concentration detected in any sediment sample

[B]Sum of lifetime cancer risks from all child and adult exposure periods

D= (C x IR x EF x CF) / BW

Risk= D x CSF x ADAF

Table G24. Estimated cancer risk from exposure to PCBs and PAHs in sediment through ingestion (daily year-round exposure)

Exposure Period	Total PCB	Total PAH	Sum for all chemicals[A]
Child 6 wks to < 1 yr	NA	NA	NA
Child 1 to < 2 yr	0.2 in 1,000,000	8 in 10,000,000	1 in 1,000,000
Child 2 to < 6 yr	0.6 in 1,000,000	6 in 10,000,000	1 in 1,000,000
Child 6 to < 11 yr	0.4 in 1,000,000	0.4 in 1,000,000	0.8 in 1,000,000
Child 11 to <16 yr	0.2 in 1,000,000	0.2 in 1,000,000	0.4 in 1,000,000
Child 16 to <21 yr	0.2 in 1,000,000	0.06 in 1,000,000	0.2 in 1,000,000
cumulative Child for 21 years	2 in 1,000,000	2 in 1,000,000	4 in 1,000,000
Adult CR (33 years) -- 95% residential occupancy period	0.5 in 1,000,000	0.2 in 1,000,000	0.7 in 1,000,000
Lifetime	2 in 1,000,000	2 in 1,000,000	4 in 1,000,000

[A]Cancer risk is rounded to a single significant digit.

Table G25. Summary of non-cancer risk from ingestion of nickel in Johnson Creek sediment.

Scenario	Hazard Quotient based on weekly year-round exposure	Hazard Quotient based on daily year-round exposure
Child 6 wks to < 1 yr	0.0	0.0
Child 1 to < 2 yr	0.3	2.2
Child 2 to < 6 yr	0.2	1.4
Child 6 to < 11 yr	0.1	0.8
Child 11 to <16 yr	0.1	0.4

Child 16 to <21 yr	0.05	0.3
Adult CR (33 years) -- 95% residential occupancy period	0.02	0.2

Table G26. Non-cancer risk calculation details for ingestion of nickel in Johnson Creek sediment (assuming *weekly* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult
C	Contaminant concentration (ppm or mg/kg)	2500	2500	2500	2500	2500	2500
IR	Intake rate - upper percentile of daily soil intake for each age group (mg/day)	200	200	200	200	200	100
EF	Exposure factor (F x ED)/AT	0.14	0.14	0.14	0.14	0.14	0.14
F	Frequency of Exposure (days/week x weeks/year)	52	52	52	52	52	52
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33
ATnc	Cancer averaging time (days during exposure duration)	365	1460	1825	1825	1825	12044
CF	Conversion factor 0.000001 (kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80
D	Estimated dose rate from the above equation (mg/kg/day)	6.3E-03	4.1E-03	2.2E-03	1.3E-03	1.0E-03	4.5E-04
RfD	EPA Reference Dose (mg/kg/day)	0.02	0.02	0.02	0.02	0.02	0.02
HQ	Hazard Quotient	0.31	0.21	0.11	0.06	0.05	0.02

D= (C x IR x EF x CF) / BW

HQ= D / RfD

Table G27. Non-cancer risk calculation details for ingestion of nickel in Johnson Creek sediment (assuming *daily* year-round exposure)

	Description	Child 1 to < 2 yr	Child 2 to < 6 yr	Child 6 to < 11 yr	Child 11 to <16 yr	Child 16 to <21 yr	Adult
C	Contaminant concentration (ppm or mg/kg)	2500	2500	2500	2500	2500	2500
IR	Intake rate - upper percentile of daily soil intake for each age group (mg/day)	200	200	200	200	200	100
EF	Exposure factor (F x ED)/AT	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
F	Frequency of Exposure (days/week x weeks/year)	365	365	365	365	365	365
ED	Age-specific Exposure duration (years)	1	4	5	5	5	33
ATnc	Cancer averaging time (days during exposure duration)	365	1460	1825	1825	1825	12044
CF	Conversion factor 0.000001 (kg/mg)	0.000001	0.000001	0.000001	0.000001	0.000001	0.000001
BW	Body weight (kg)	11.4	17.4	31.8	56.8	71.6	80
D	Estimated dose rate from the above equation (mg/kg/day)	4.4E-02	2.9E-02	1.6E-02	8.8E-03	7.0E-03	3.1E-03
RfD	EPA Reference Dose (mg/kg/day)	0.02	0.02	0.02	0.02	0.02	0.02
HQ	Hazard Quotient	2.19	1.44	0.79	0.44	0.35	0.16

D= (C x IR x EF x CF) / BW

HQ= D / RfD

Exposure Scenario 5. Consumption of crayfish from Johnson Creek

Table G28. Calculation of the number of Johnson Creek crayfish meals that can be consumed each month without exceeding health-based exposure guidelines for individual metals and PCBs

	Description	Arsenic, Total	Chromium, Total	Cobalt, Total	Mercury, Total	Nickel, Total	Zinc, Total
BW	Body Weight (kg)	70	70	70	70	70	70
D	Days/month (days)	30.44	30.44	30.44	30.44	30.44	30.44
Kg/meal	Fish/meal (one 8-ounce meal = 0.227 kg)	0.227	0.227	0.227	0.227	0.227	0.227
C	Contaminant concentration detected in crayfish (mg/kg wet weight)	0.028[A]	0.63[B]	0.26	0.019	1.08	24.1
CV	Oral dose comparison value (mg/kg body weight/day)	0.0003	0.0009	0.01	0.0003	0.02	0.3
8-ounce crayfish meals per month based on risk from individual chemicals = (BW x D)/(kg/meal) x (CV/C)		**100.6**	**13.4**	**361.0**	**148.2**	**173.8**	**116.8**

[A] Assumes that 10% of the arsenic detected is in its more toxic, inorganic form. The general consensus in the scientific literature is that about 10% of the arsenic typically found in the edible parts of fish and shellfish is inorganic arsenic (39).

[B] Based on the unlikely but health-protective assumption that 100% of chromium detected is in its more toxic, hexavalent form.

Table G29. Calculation of the number of Johnson Creek crayfish meals that can be consumed each month without exceeding health-based exposure guidelines for combined risk of metals and PCBs that target specific organ systems

Organ systems targeted	**8-ounce crayfish meals that can be consumed each month based on cumulative risk of chemicals that target each organ system**[A]
Brain development (methylmercury and PCBs)	5.4 eight-ounce meals
Immune system (zinc and PCBs)	5.4 eight-ounce meals
Skin (arsenic and PCBs)	5.3 eight-ounce meals
Blood (zinc, chromium, and cobalt)	11.6 eight-ounce meals

[A] Calculated using the formula defined in OHA's fish advisory Standard Operating Guidance (38): Meals per month = (BW x D)/(kg/meal) x (1/ ∑ C/CV))

Appendix H. Glossary

This glossary defines words used in this document.

Absorption:	How a chemical enters a person's blood after the chemical has been swallowed, has come into contact with the skin, or has been breathed in.
Adverse (or negative) Health Effects	A change in body function or cell structure that might lead to disease or health problems
ATSDR:	The **A**gency for **T**oxic **S**ubstances and **D**isease **R**egistry. ATSDR is a federal health agency in Atlanta, Georgia that deals with hazardous substance and waste site issues. ATSDR gives people information about harmful chemicals in their environment and tells people how to protect themselves from coming into contact with chemicals.
Background Level:	An average or expected amount of a chemical in a specific environment or amounts of chemicals that occur naturally in a specific environment.
Bioavailability:	See **Relative Bioavailability**.
Cancer:	A group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.
Chronic Exposure:	A contact with a substance or chemical that happens over a long period of time. ATSDR considers exposures of more than one year to be *chronic*.
Completed Exposure Pathway:	See **Exposure Pathway**.

Comparison Value (CVs):	Concentrations of substances in air, water, food, and soil that are unlikely, upon exposure, to cause adverse health effects. Comparison values are used by health assessors to select which substances and environmental media (air, water, food and soil) need additional evaluation while health concerns or effects are investigated.
Concern:	A belief or worry that chemicals in the environment might cause harm to people.
Concentration:	How much or the amount of a substance present in a certain amount of soil, water, air, or food.
Contaminant:	See **Environmental Contaminant**.
Dermal Contact:	A chemical getting onto your skin. (See **Route of Exposure**).
Dose:	The amount of a substance to which a person may be exposed, usually daily. Dose is often explained as "amount of substance(s) per body weight per day".
Duration:	The amount of time (days, months, years) that a person is exposed to a chemical.
Environmental Contaminant:	A substance (chemical) that gets into a system (person, animal, or the environment) in amounts higher than the **Background Level**, or what would be expected.
Environmental Media:	Usually refers to the air, water, and soil in which chemicals of interest are found. Sometimes refers to the plants and animals that are eaten by humans. **Environmental Media** is the second part of an **Exposure Pathway**.
US Environmental Protection Agency (EPA):	The federal agency that develops and enforces environmental laws to protect the environment and the public's health.

Exposure: Coming into contact with a chemical substance. (For the three ways people can come in contact with substances, see **Route of Exposure**.)

Exposure Assessment: The process of finding the ways people come in contact with chemicals, how often and how long they come in contact with chemicals, and the amounts of chemicals with which they come in contact.

Exposure Pathway: A description of the way that a chemical moves from its source (where it began) to where and how people can come into contact with (or get exposed to) the chemical.

ATSDR defines an exposure pathway as having 5 parts:
1. Source of Contamination,
2. Environmental Media and Transport Mechanism,
3. Point of Exposure,
4. Route of Exposure, and
5. Receptor Population.

When all 5 parts of an exposure pathway are present, it is called a **Completed Exposure Pathway**. Each of these 5 terms is defined in this Glossary.

Frequency: How often a person is exposed to a chemical over time; for example, every day, once a week, or twice a month.

Health Effect: ATSDR deals only with **Adverse Health Effects** (see definition in this Glossary).

Ingestion: Swallowing something, as in eating or drinking. It is a way a chemical can enter your body (See **Route of Exposure**).

Inhalation: Breathing. It is a way a chemical can enter your body (See **Route of Exposure**).

kg	Kilogram or 1000 grams. Usually used here as part of the dose unit mg/kg/day meaning mg (contaminant)/kg (body weight)/day.
µg	Microgram or 1 millionth of 1 gram. Usually used here as part of the concentration of contaminants in water (µg/Liter).
mg	Milligram or 1 thousandth of 1 gram. Usually used here as in a concentration of contaminant in soil mg contaminant/kg soil or as in the dose unit mg/kg/day meaning mg (contaminant)/kg (body weight)/day.
MRL:	**M**inimal **R**isk **L**evel. An estimate of daily human exposure – by a specified route and length of time -- to a dose of chemical that is likely to be without a measurable risk of adverse, non-cancerous effects. An MRL should not be used to predict adverse health effects.
oxbow	A U-shaped bend in the course of a river
PHA:	**P**ublic **H**ealth **A**ssessment. A report or document that looks at chemicals at a hazardous waste site and tells if people could be harmed from coming into contact with those chemicals. The PHA also tells if possible further public health actions are needed.
Point of Exposure:	The place where someone can come into contact with a contaminated environmental medium (air, water, food or soil). Some examples include: the area of a playground that has contaminated dirt, a contaminated spring used for drinking water, or the backyard area where someone might breathe contaminated air.
Population:	A group of people living in a certain area or the number of people in a certain area.
Public Health Assessment(s):	See **PHA**.

Reference Dose (RfD):	An estimate, with safety factors (see **Safety Factor**) built in, of the daily, life-time exposure of human populations to a possible hazard that is not likely to cause harm to the person.
Relative Bioavailability:	The amount of a compound that can be absorbed from a particular medium (such as soil) compared to the amount absorbed from a reference material (such as water). Expressed in percentage form.
Route of Exposure:	The way a chemical can get into a person's body. There are three exposure routes: – breathing (also called inhalation), – eating or drinking (also called ingestion), and – getting something on the skin (also called dermal contact).
Safety Factor:	Also called **Uncertainty Factor**. When scientists don't have enough information to decide if an exposure will cause harm to people, they use "safety factors" and formulas in place of the information that is not known. These factors and formulas can help determine the amount of a chemical that is not likely to cause harm to people.
Source (of Contamination):	The place where a chemical comes from, such as a landfill, pond, creek, incinerator, tank, or drum. Contaminant source is the first part of an **Exposure Pathway**.
Toxic:	Harmful to health. Any substance or chemical can be toxic at a certain dose (amount). The dose is what determines the potential harm of a chemical and whether it would cause someone to get sick.
Tumor:	Abnormal growth of tissue or cells that have formed a lump or mass.
Uncertainty Factor:	See **Safety Factor**.

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