Exponent®

Screening Risk Assessment for Air Emissions from the Alcoa Fjardaal Aluminum Smelter

Prepared for

Alcoa Fjardaal
Reydarfjordur, Iceland

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Prepared by

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Acronyms and Abbreviations

BaP	benzo[a]pyrene
CSM	conceptual site model
CDI	chronic daily intake
CoPC	chemical of potential concern
cPAH	carcinogenic polycyclic aromatic hydrocarbon
CSF	cancer slope factor
EIA	environmental impact assessment
EPC	exposure point concentration
ERA	ecological risk assessment
FTV	Fjardaal Team Village
GIS	geographic information system
HHRA	human health risk assessment
K_{oc}	organic carbon partition
LOAEL	lowest-observed-adverse-effect level
MEI	maximum exposed individual
NAAQS	national ambient air quality standards
NCP	National Contingency Plan
NOAEL	no-observed-adverse-effect level
PAH	polycyclic aromatic hydrocarbon
PF/HF	particulate fluoride and hydrogen fluoride
PM_{10}	particulate matter <10 μm
$PM_{2.5}$	particulate matter <2.5 μm
RAGS	Risk Assessment Guidance for Superfund
RBC	risk-based concentration
RfC	reference concentration
RfD	reference dose
RME	reasonable maximum exposure
SO_2	sulfur dioxide
tpy	tonnes per year
TRV	toxicity reference value
TSP	total suspended particulates
UCL	upper confidence limit
U.S. EPA	U.S. Environmental Protection Agency

Executive Summary

Alcoa is constructing the Fjardaal aluminum smelter at the Hraun industrial area in Reydarfjordur, Fjardabyggd, East Iceland. After the smelter becomes operational in 2007, it will produce a maximum of 346,000 metric tonnes per year (tpy) of aluminum. Hydro Aluminium AS, Haefi hf., Landsvirkjun, and Reydaral hf., had previously planned to construct a facility (Reydaral) at the same location with a maximum capacity of 420,000 tpy. Unlike the original Reydaral design, the Fjardaal facility will use imported anodes (and will thus not include an anode production plant) and spent pot liner will be exported rather then being placed in a onsite landfill.

In November 2002, Alcoa prepared a report comparing the environmental impacts of the Fjardaal plant with the original Reydaral design and concluded that the Fjardaal facility would meet applicable air emission standards without the need for installing a seawater scrubber system (see Honnun et al. 2002). The objective of this risk assessment is to evaluate risks to human health and the regional ecology of air emissions under two scenarios: 1) Fjardaal without seawater scrubbers (the "base case"), and 2) Fjardaal with seawater scrubbers (the "alternative case"). The risk estimates presented in this report were developed using air dispersion modeling results prepared by Earth Tech, Inc., under separate contract to Alcoa. Alcoa, Reydaral (2001) and Earth Tech (2005a,b) presented initial results of modeling without deposition wherein estimates of the concentration of particulate matter (PM_{10}), sulfur dioxide (SO_2), hydrogen fluoride, and benzo[a]pyrene (BaP) were compared to the corresponding ambient air standards or air quality guidelines as identified in the EIA including standards from Norway, Iceland, or European directives EC 1999 and EU 2004 (Reydaral 2001). Earth Tech provided Exponent with additional modeling results incorporating deposition modeling in order to support the receptor-specific analysis that is required for risk assessment (Earth Tech 2005c) and specifically required for risk assessment of carcinogenic PAHs (cPAH) (Scire 2006a, pers. comm.). The modeling approach is described in detail by Earth Tech (2006) and is based on the CALMET diagnostic meteorological model (Scire et al. 2000a) and the CALPUFF non-steady state dispersion model (Scire et al. 2000b).

The objective of the risk assessments is to determine whether there is a consequential difference in the level of risk to human and ecological receptors from constituents in air emissions from Fjardaal under the base case (without seawater scrubbers) and the alternative case (with seawater scrubbers). This objective is met by developing reasonably conservative exposure estimates for the most highly exposed receptors and comparing those exposure estimates to well-accepted and conservative effects levels. The technical approaches used in this report are consistent with standard practice in risk assessment as documented by U.S. EPA (1989, 1997a,b, 1999, 2005b).

For the seawater scrubber scenario, emissions were modeled assuming that the facility would use anodes made from coke containing 3 percent sulfur. For the no seawater scrubber scenario, emissions were modeled assuming that the facility would use anodes made from coke containing 1.8 percent sulfur. Under the seawater scrubber scenario, there are no emissions of SO_2 from the fume stack; however, there is a small increase in estimated SO_2 emissions from

the potline roof vents and SO_2 would be emitted from the four seawater scrubber stacks. Emissions of fluoride, PM_{10}, and polycyclic aromatic hydrocarbons (PAHs) from the fume stack, would also be decreased with the use of seawater scrubbers. In order to model growing season emissions of fluoride conservatively, the estimated fluoride emissions for the potline roof vents were increased based on observed summer emission rates from a similar facility, Deschambault smelter in Quebec, Canada.

Oxides of nitrogen (NO_x) were not modeled because they are expected to be an insignificant component of air emissions. NO_x emissions are generated only from the potline at a rate of roughly 2.4 kg/hour. Because ambient air standards for SO_2 and NO_x are similar and NO_x emissions are less than 1 percent of the SO_2 emission rate, there was no need to model NO_x emissions. $PM_{2.5}$ and total suspended particulates (TSP) were not modeled for two reasons: 1) there are no Iceland ambient air standards for $PM_{2.5}$ or TSP, and 2) $PM_{2.5}$ is a component of PM_{10} and evaluation of compliance with PM_{10} standards will thus include evaluation of $PM_{2.5}$ (Palazzolo 2005a, pers. comm.). Perfluorocarbons were not modeled because they are expected to be generated sporadically over short time periods and there are no regulatory limits for perfluorocarbons. Furthermore, environmental concern about perfluorocarbons stems from their potential role as a greenhouse gas, not from their potential role in localized human health or ecological effects.

Modeling results are presented for two nested grids of points (termed "receptors" by Earth Tech [2006]) that extend 19 km west, 32 km east, 18 km north, and 13 km south from the Fjardaal smelter. The innermost grid is in the immediate vicinity of the Fjardaal smelter and consists of 4,224 points with a spacing of 100 m between points. The outermost grid consists of 6,560 points with a spacing of 200 m between points. For purposes of evaluating exposure to humans and ecological receptors, the grid was projected geographically using a geographic information system (GIS). This risk assessment employs the data generated from Earth Tech (2005c, Scire 2006a, pers. comm.) using emissions modeled with deposition. Estimated air concentrations and deposition rates, with or without the effect of seawater scrubbers, were then compared on a point-specific basis or examined statistically or probabilistically to describe the nature of exposure of each constituent to sensitive receptors.

Human Health Risk Assessment

Given the site characteristics including current use and future use, and considering the dilution zone surrounding the facility, the following receptors were considered in the risk assessment process:

- Onsite outdoor worker
- Seagoing worker
- Hypothetical maximum exposed individual residing at the facility fenceline
- Future hypothetical resident at Fjardaal Team Village
- Closest resident

- Residents in Reydarfjordur and Eskifjordur villages
- Closest agricultural farmer
- Visitor to the Holmanes Reserve.

Indoor exposures to onsite workers are not considered in this risk assessment because exposures within the future facility will be maintained consistent with occupational health standards set to protect human health. The first step of the human health risk assessment was a screening process that identified potential receptors and chemicals to evaluate further in the quantitative risk assessment. Concentration estimates for SO_2, fluoride, and particulates in ambient air were considered in comparison with all relevant standards and PAHs were compared with health protective screening values in air and in soil. Specifically, air concentration estimates were compared to the relevant standards from Iceland, Norway, and U.S. Environmental Protection Agency (U.S. EPA) for SO_2, hydrogen fluoride, and PM_{10}. Air concentration estimates for PAHs were compared with available standards and with health-protective screening concentrations (i.e., preliminary remediation goals derived by U.S. EPA Region 9).

Estimated concentrations for all constituents evaluated were well within the applicable standards and risk-based concentrations (RBCs). However, in order to provide additional information to risk managers, risk estimates were calculated for PAHs in the areas with the highest estimated concentrations. Thus the following receptors were evaluated in the quantitative risk assessment:

- Onsite outdoor worker—potential inhalation of PAHs in air
- Seagoing worker—potential inhalation of PAHs in air
- Hypothetical maximum exposed individual residing at the facility fenceline—potential inhalation of PAHs in air.

In addition to inhalation exposure, the risk assessment evaluated potential exposure to human receptors via ingestion of constituents deposited in soil. The maximum modeled deposition rates ($\mu g/m^2/s$) from within the facility boundary and in the dilution zone were used to derive soil concentration estimates for fluoride and PAHs. Fluoride and PAHs were the only constituents modeled in this manner because there are no oral toxicity values for the other constituents. The estimated deposition rates represent the highest rates modeled within the receptor grid and in general decreased with distance from the facility. Exposure point concentration estimates for total PAHs and fluoride were then compared to well-accepted RBCs for BaP and for fluoride in soil. For all exposure routes and receptors, conservative assumptions were applied at various stages of the risk assessment to ensure that risk estimates would be biased high. Soil concentration estimates were compared with health protective RBCs for soil. All soil concentrations were well below these values. The potential for the food-chain to be affected by PAHs and hydrogen fluoride is greatly reduced or eliminated by the relatively low contribution, the location of the higher deposition within industrial areas, and the degree to which PAHs are metabolized in tissues. Information on hydrogen fluoride concentrations in soil is somewhat limited but suggests that the contribution related to facility operations would be well below background levels.

The results of the risk assessment indicate that there are no instances in which the model estimates predict that a standard would not be met. Use of seawater scrubbers is expected to decrease average SO_2 air concentration estimates in the short term (i.e., 1-hour, 3-hour and 24-hour); however, annual estimates are lower without seawater scrubbers. In the short term, both with and without seawater scrubbers, the number of exceedances of SO_2 predicted per year are well below the maximum number of allowed exceedances. All carcinogenic risk estimates for cPAHs were lower than the 10^{-6} risk level identified by many regulatory agencies as the lower end of the acceptable risk range of 10^{-6} to 10^{-4} and for noncarcinogenic PAHs, all hazard indices were well below the threshold of 1.0, with the highest estimate being 0.00028. Risk estimates for PAHs were slightly higher for the alternative case, with seawater scrubbers, than the base case, but were still well within acceptable levels and the increase in risks was within the level of uncertainty of the risk estimates. Evaluation of exposure via deposition of PAHs and fluoride onto soils indicated that no risk estimates exceeded the risk-based thresholds. Modeled air concentrations of fluoride and particulate matter (using PM_{10}) were well below health standards.

Ecological Risk Assessment

The ecological risk assessment was undertaken to evaluate risk to plant communities and three wildlife receptors in the vicinity of the Fjardaal smelter under the base case (without seawater scrubbers) and the alternative case (with seawater scrubbers). Risks to plants were evaluated by comparing modeled concentrations of SO_2 and fluoride to conservative toxicity thresholds for sensitive plant communities. The plant communities considered in this assessment included moss/lichen and heather/heath assemblages and lodgepole pine. Risks to vertebrates were evaluated in two steps: 1) derive estimates of long-term concentrations of PAH and fluoride in plants, and 2) derive estimates of dietary intake of PAH and fluoride for herbivorous birds and mammals and comparing this dietary intake to toxicity thresholds developed from the scientific literature.

Modeled exposure of plants to SO_2 indicates that the toxicity threshold value is not exceeded under either scenario. Therefore, exposure of plants to SO_2 from Fjardaal will not result in any appreciable risk of toxicity, although higher concentrations of SO_2 are modeled under the alternative case with seawater scrubbers. Modeled exposure of plants to hydrogen fluoride indicates that the toxicity threshold for the most sensitive species (mosses and lichens) will be exceeded in limited cases under both scenarios:

- For the base case (without seawater scrubbers) the toxicity threshold will be exceeded by a small margin and over a small area (32 ha on an annual basis and 45 ha on a growing season basis) within the dilution zone
- For the alternative case (with seawater scrubbers) the toxicity threshold will be exceeded by a larger margin and over a larger area (155 ha on an annual basis and 559 ha on a growing season basis) than for the base case.

Modeled exposure of lodgepole pine to hydrogen fluoride indicates that the toxicity threshold for this species will not be exceeded under the base case (without seawater scrubbers) but will

be exceeded both within and outside the dilution zone over an area of 217 ha under the alternative case (with seawater scrubbers). The risk estimates for plants are likely to overstate actual risk because the thresholds for fluoride toxicity are based on sublethal effects that are unlikely to result in mortality to plants if the thresholds are exceeded by a small margin and for short periods. Thus, the ecological effect, if any, of the predicted exceedances of fluoride toxicity thresholds for plants is likely to consist of subtle, localized changes in plant communities favoring more tolerant species such as heather/heath and grasses.

Modeled PAH concentrations are not expected to result in the exceedance of toxicity thresholds for terrestrial birds or mammals at any location under either scenario. In some limited cases, modeled hydrogen fluoride concentrations may exceed conservative toxicity thresholds for birds and mammals. If it is assumed that these animals consume 100 percent grasses, modeled exposures will not result in the exceedance of toxicity thresholds for any receptor at any location. However, if it is assumed that the wildlife receptors consume 100 percent heather, modeled toxicity thresholds are exceeded at some points for the rock ptarmigan and wood mouse and the threshold is exceeded by a wider margin and at more points under the alternative (with seawater scrubber) scenario than the base case (without seawater scrubber) scenario. These results are based on several conservative assumptions regarding exposure and toxicity. When life history and population dynamics are taken into account, these exceedances are not likely to result in population-level effects with the possible exception of effects to wood mouse populations in a very small area within the dilution zone (72 ha under the base case without seawater scrubbers) and in an area extending partially outside the dilution zone (440 ha under the alternative case with seawater scrubbers).

To the extent that any adverse effects to plants, mammals, or birds may be manifest, the likelihood of such effects, on average, is lower under the base case without seawater scrubbers, than under the alternative case, with seawater scrubbers.

1 Introduction

1.1 Background

Alcoa is constructing the Fjardaal aluminum smelter at the Hraun industrial area in Reydarfjordur, Fjardabyggd, East Iceland (Figure 1-1). After the smelter becomes operational in 2007, it will produce a maximum of 346,000 metric tonnes per year (tpy) of aluminum. The original concept for an aluminum smelter at this location was developed by Hydro Aluminium AS, Haefi hf., Landsvirkjun, and Reydaral hf., who planned to construct a facility (hereafter referred to as Reydaral) that would produce 240,000–280,000 tpy in Phase 1 with the possibility of increasing capacity to 360,000–420,000 tpy in a Phase 2 expansion. A detailed environmental impact assessment (EIA) was prepared in 2001 for both phases of the proposed plant, to evaluate potential impacts on terrestrial and marine environments during plant construction and operation, and socioeconomic impacts during operation (Reydaral 2001). The EIA was submitted to the Iceland Planning Agency in May 2001 and was approved in August 2001. The main structures of the original aluminum smelter included potrooms, casthouse, anode production facilities, anode rodding plant, fume treatment facilities, power transmission substation, alumina silos, warehouses, and service buildings.

In the spring of 2002 when Hydro Aluminium AS declared that they would be unable to meet the project schedule, Alcoa and Landsvirkjun entered into negotiation with the Icelandic government to construct a facility at the same location. In November 2002, Alcoa purchased Reydaral from Norsk Hydro and Haefi hf. Also in November 2002, Alcoa prepared a report (Honnun et al. 2002) comparing the environmental impacts of a 322,000 tpy Fjardaal plant with the two-phase, 420,000 tpy original Reydaral design. Other than the change in production capacity, currently determined to be a maximum of 346,000 tpy, the main difference between the Fjardaal plant and the original Reydaral design is that Alcoa will not install an anode production plant, using imported anodes in the production process instead. In addition, Alcoa will export spent pot liner instead of placing it in a landfill on the facility.

Honnun et al. (2002) concluded that the Fjardaal facility would be able to meet applicable air emission standards without the need for installing a seawater scrubber system by improving air dispersion using increased buoyancy via a 78 m stack. Thus, the Fjardaal facility will have no process water discharge to the sea.

In 2005, Alcoa retained Exponent to evaluate potential ecological impacts related to effluent from the seawater scrubbers (Exponent 2005). The ecological assessment was focused on aquatic invertebrates, plankton, fish, seabirds, and marine mammals, and considered polycyclic aromatic hydrocarbons (PAHs), fluorides, and sulfur dioxide (SO_2). Data from facilities in Norway and Canada were used to estimate the potential exposure to the chemicals of concern. The assessment determined that impact of the effluent on the marine ecosystem would be insignificant except in the seawater scrubber scenario, where adverse effects could be expected in benthic macroinvertebrates in the immediate vicinity of the effluent outlet. The findings from this prior assessment were also considered here.

This report relied upon the following key documents for background and data:

- Earth Tech. 2006. *Assessment of Air Quality Impacts of Emissions from the Alcoa Aluminum Plant in Reydarfjordur, Iceland* and subsequent Alcoa Iceland CALPUFF modeling results (Earth Tech 2005b) and air dispersion modeling results with deposition (Earth Tech 2005c) and subsequent additional modeling for carcinogenic PAHs (cPAHs) (Scire 2006a,b, pers. comm.)

- Exponent. 2005. *Ecological Risk Assessment for Use of Wet Scrubbers at Alcoa Fjardaal Aluminum Plant in Reydarfjordur, Fjardabyggd, Iceland*.

- Honnun, Alcoa, and VST. 2002. *Aluminum Plant in Reydarfjordur, Fjardabyggd: Comparison of Environmental Impact of the Proposed 322,000 tpy Alcoa-Reydaral Aluminum Plant and the Two Phase 420,000 tpy Reydaral Aluminum Plant*. Honnun Engineering Consultants, Reykjavik, Iceland; Alcoa, Reykjavik, Iceland; and VST Consulting Engineers, Reykjavik, Iceland.

- RETEC. 2005. *External Environmental Monitoring Work Plan Baseline Survey, Fjardaal Smelter Project, Reydarfjordur, IS*.

- Reydaral. 2001. *Aluminum plant in Reydarfjordur Fjardabyggd—Environmental Impact Assessment (EIA) 1st Phase; 240.000-280.000 TPY; 2nd Phase Expansion up to 360.000-420.000 TPY.* Prepared by Honnun hf., VST hf., Hydro Technology and Projects, and Hydro Aluminium. Reydaral hf., Reykjavik, Iceland.

1.2 Objective and Scope

As a part of the current EIA, Exponent has been retained by Alcoa to assess the risks to human health and the regional ecology of air emissions under two scenarios: 1) Fjardaal without seawater scrubbers (the previous design, or "base case"), and 2) Fjardaal with seawater scrubbers to achieve additional SO_2 control (the "alternative case"). The risk estimates presented in this report are based on air dispersion modeling results prepared by Earth Tech under separate contract to Alcoa. Alcoa, Reydaral (2001) and Earth Tech (2005a,b) initially developed emission parameters based on emissions from other aluminum smelters with similar technology and operating procedures. They presented initial results of modeling without deposition wherein estimates of the concentration of particulate matter (PM_{10}), SO_2, hydrogen fluoride, and benzo[a]pyrene (BaP) were compared to the corresponding ambient air standards or air quality guidelines based on Norwegian, Icelandic, or European directives (EC 1999; EU 2004; Reydaral 2001), or U.S. standards (U.S. EPA 2005c). Earth Tech provided Exponent with additional modeling results incorporating deposition modeling in order to support the receptor-specific analysis that is required for risk assessment (Earth Tech 2005c).

The objective of the risk assessment is to determine whether there is a consequential difference in the level of risk to human and ecological receptors from constituents in air emissions because

of the use of seawater scrubbers. This objective is met by developing reasonably conservative exposure estimates for the most highly exposed receptors and comparing those exposure estimates to well-accepted and conservative effects levels. The technical approaches used in this report are consistent with standard practice in risk assessment as documented by U.S. EPA (1989, 1993, 1997a,b, 2005b).

1.3 Document Organization

The remainder of this report is organized into the following sections:

- Section 2, *Conceptual Site Models*—This section first provides an overview of primary aluminum production, including a description of the formation and control of emissions of principal substances of potential concern. Next, an overview of the air dispersion modeling is provided including major assumptions and variables, and a summary of modeling runs. Finally, this section provides conceptual site models (CSMs) characterizing potential exposure of human and ecological receptors to substances of potential concern in air emissions. The CSMs include a description of the demographic and ecological setting in the vicinity of Fjardaal.

- Section 3, *Human Health Risk Assessment*—This section presents all the key components of the human health risk assessment (HHRA). First, chemicals of potential concern (CoPCs) in air emissions and relevant health-based criteria are identified for screening emissions predicted by the air modeling. Second, exposure point concentrations (EPCs) are determined for each receptor identified in the CSM. Third, toxicity of the CoPCs is discussed. Finally, the exposure and toxicity of both carcinogens and noncarcinogens are assessed in the risk characterization. The risk characterization includes identification of major sources of uncertainty and the effect of the uncertainty on conclusions regarding estimated risk.

- Section 4, *Ecological Risk Assessment*—This section presents all the key components of the ecological risk assessment (ERA). First, the problem formulation is developed, which builds on the CSM to identify complete exposure pathways to ecological receptors, assessment and measurement endpoints for evaluating risk, and toxicity profiles for selected constituents in air emissions. Second, EPCs are estimated for key ecological receptors at key locations. This step includes development and presentation of food-web models for dietary exposures to higher trophic level receptors. Third, the exposure and toxicity assessment are combined in a risk characterization for each receptor. Finally, major sources of uncertainty are defined and the effect of the uncertainty on conclusions regarding estimated risk are discussed.

- Section 5 *Conclusions*—This section presents the conclusions of the HHRA and ERA. Conclusions are presented that compare risk estimates under the base case (no seawater scrubbers) with risk estimates under the alternative case (seawater scrubbers for additional SO_2 removal).

- Section 6, *References*—This section presents a list of references cited in the report.

- *Appendices*—Appendix A presents a series of tables containing the results from the calculation of human health cancer risk and noncancer hazard indices for each of the human receptors. Appendix B presents the derivation of soil concentrations used in the HHRA and ERA. Appendix C provides a summary of toxicity test results presented in the scientific literature.

2 Conceptual Site Models

2.1 Overview of the Smelter, the Smelting Process, and Sources of Aerial Emissions

The Fjardaal smelter is designed with two potrooms, each approximately 1,100 m in length. The production capacity of Fjardaal is expected to be 322,000 tpy at startup, increasing to a maximum estimated capacity of 346,000 tpy if necessary power becomes available, and the plant gains more efficient use of the pots. The smelter is expected to be operational in April 2007, and will consist of the following major components (HRV 2005):

- Two potrooms
- Dry scrubber facilities
- Cast house
- Anode rodding shop
- Main substation and rectifier building
- Light fuel oil storage
- Auxiliary services
- Warehouses and storage buildings.

Primary aluminum production, or smelting, is the process by which refined alumina (Al_2O_3) from bauxite ore is electrolytically reduced to aluminum metal by immersing carbon anodes into an electrolytic bath consisting primarily of molten alumina and sodium aluminum fluoride (Na_3AlF_6, or cryolite). The bath is contained in carbon-lined cells, or pots, where the carbon lining acts as the cathode. The electrolytic reduction of alumina by carbon is represented by the following:

$$2Al_2O_3 + 3C \rightarrow 4Al + 3CO_2$$

Atmospheric emissions from the plant will originate primarily from the following activities:

- Captured and treated gases from the potrooms
- Fugitive gases from the potrooms that are vented to the atmosphere
- Material handling.

Other minor sources of emissions include activities such as fluxing of the molten metal in the cast house, cathode lining and delining, and anode assembly in the anode rodding plant.

The principal constituents in gaseous emissions from primary aluminum production are the following (Reydaral 2001):

- **Fluorides**—Fluorides (gaseous hydrogen fluoride and particulate fluorides) are vaporized from the cryolite bath (Na_3AlF_6) and are captured and recycled back to production via the dry gas treatment center (low concentrations escape through the stack).
- **Particulates—**Particulate material consists mainly of alumina (Al_2O_3) and cryolite. Fine particulate material entrained in the flue gas from the pots is captured and recycled back to production via the dry gas treatment center (low concentrations escape through the stack).
- **Sulfur Dioxide—**SO_2 is generated by the oxidation of sulfur in the pitch and coke that is used in the anodes.
- **Carbon Dioxide and Carbon Monoxide—**Carbon dioxide is formed by the reaction of the oxygen in the alumina with the carbon of the anodes. Carbon monoxide is formed by the incomplete reaction of the oxygen in the alumina with the carbon of the anodes.
- **Perfluorocarbons**—Perfluorocarbons such as tetrafluoromethane (CF_4) and hexafluoroethane (C_2F_6) are formed by elevated temperatures during excursions of the electric resistance across the pot (anode effect).
- **Polycyclic Aromatic Hydrocarbons (PAHs)—**PAHs originate either by volatilization from or combustion of the coal-tar pitch used as a binder in anode manufacture and in the cathode lining.

Fjardaal will be operated according to process control procedures and the latest emission control technology employed by modern prebake aluminum plants. Best available technology, as defined in the Integrated Pollution Prevention and Control Directive 96/61/EC, will be used for treatment of aerial emissions. Gaseous emissions are minimized by operational controls and contaminants will be captured and recycled back to production via the dry gas treatment center (low concentrations escape through the stack) at Fjardaal by dry scrubbers. Most of the fluorides, dust, and PAHs (>99 percent) are removed with dry scrubbing. The most important operational controls are implemented in the potrooms where the electrolysis pots are closed and fumes (gases and particulates) generated inside the pots are collected via a special ventilation system and transported to the gas treatment center.

2.2 Air Emission Modeling

Earth Tech, Inc., used current state-of-the-science, comprehensive meteorological and regulatory air dispersion modeling systems to conduct air emission modeling. The modeling approach is described in detail by Earth Tech (2005a, 2006) and is based on the CALMET diagnostic meteorological model (Scire et al. 2000a) and the CALPUFF non-steady-state dispersion model (Scire et al. 2000b).

Figure 2-1 shows a schematic of air emission sources and principal buildings used in the air dispersion modeling. Table 2-1 presents a summary of point source emission parameters with and without seawater scrubbers. With no seawater scrubber system installed, the 78 m fume stack will discharge all captured emissions from the smelting operations following treatment by the dry scrubber system. Alternatively, a seawater scrubber system with four additional stacks, each 40 m tall, would be installed to reduce overall SO_2 emissions. The casthouse furnace emissions are unaffected by installation of seawater scrubbers; thus, this source term is the same for each case.

For the seawater scrubber scenario, emissions were modeled assuming use of coke with 3 percent sulfur content in the anodes (as opposed to 1.8 percent sulfur content for the scenario without seawater scrubbers) to produce conservative (i.e., high) estimates of SO_2 emissions. Under the seawater scrubber scenario, there are no emissions of SO_2 from the fume stack; however, there is a small increase in estimated SO_2 emissions from the potline roof vents and seawater scrubber stacks to account for the potentially higher sulfur content in the anodes. Estimated emissions of fume stack fluoride, PM_{10} and PAHs are also decreased with the use of seawater scrubbers. In order to model growing season emissions of fluoride conservatively, the estimated fluoride emissions for the potline roof vents were increased based on observed summer emission rates from a similar facility, Deschambault smelter in Quebec, Canada.

A total of 99 sets of air modeling results were used in this risk assessment (Table 2-2). The constituents listed in Table 2-2 were selected for modeling because they are the principal components of air emissions from aluminum smelters and the government of Iceland regulates them. Oxides of nitrogen (NO_x) were not modeled because they are expected to be an insignificant component of air emissions. NO_x emissions are generated only from the potline at a rate of roughly 2.4 kg/hour. Because ambient air standards for SO_2 and NO_x are similar and NO_x emissions are less than 1 percent of the SO_2 emission rate, there was no need to model NO_x emissions. $PM_{2.5}$ and total suspended particulates (TSP) were not modeled. There are no Iceland ambient air standards for $PM_{2.5}$ or TSP, and the modeling instead focused on evaluation of compliance with PM_{10} standards. Perfluorocarbons were not modeled because they are expected to be generated sporadically over short time periods and there are no regulatory limits for perfluorocarbons. Furthermore, environmental concern about fluorocarbons stems from their potential role as a greenhouse gas, rather than from potential localized human health or ecological effects.

2.3 Application of Modeling Results

Air dispersion modeling was conducted to accurately simulate complex transport and fate mechanisms that govern the short-term and long-term distribution of gaseous and particulate constituents throughout the project area. Key features and mechanisms simulated by the modeling include the following:

- Point source and line source releases
- Overland and overwater boundary layer effects
- Non-steady-state emissions and meteorological conditions

- Vertical wind shear, plume rise, and building downwash
- Complex terrain
- Dry and wet deposition.

See Earth Tech (2006) for a more complete description of the modeling.

Modeling results are presented for two nested grids of points (termed "receptors" by Earth Tech [2006]) that extend 19 km west, 32 km east, 18 km north, and 13 km south from the Alcoa facility (Figure 2-2). The innermost grid is in the immediate vicinity of the Fjardaal smelter and consists of 4,224 points with a spacing of 100 m between points. The outermost grid consists of 6,560 points with a spacing of 200 m between points. For purposes of evaluating exposure to humans and ecological receptors, the grid was projected geographically using GIS. This risk assessment employs the data generated from Earth Tech (2005c) using emissions modeled with and without deposition. Estimated air concentrations and deposition rates, with or without the effect of seawater scrubbers, were then compared on a point-specific basis or examined statistically or probabilistically to describe the nature of exposure of each constituent to sensitive receptors. The specific procedures used for exposure assessment and risk characterization are described in more detail in Sections 3 and 4, respectively.

2.4 Human Health Conceptual Site Model

The human health CSM identifies the environmental setting, potential sources, transport mechanisms, exposure media, exposure routes, exposure pathways, and human receptors. The conceptual model is used to evaluate potential exposure pathways, which are defined as the course a chemical, takes from a source to an exposed receptor. Exposure pathways consist of the following four elements: 1) a source; 2) a mechanism of release, retention, or transport of a chemical to a given medium (e.g., air, water, soil); 3) a point of human contact with the medium (i.e., exposure point); and 4) a route of exposure at the point of contact (e.g., incidental ingestion, dermal contact). If any of these elements are missing, the pathway is considered incomplete (i.e., it does not present a means of exposure). Only those exposure pathways judged to be potentially complete are of concern for human exposure.

2.4.1 Environmental Setting

The facility site area is relatively remote, located within a sparsely populated area on a major forked fjord on the eastern seaboard of Iceland. The two populated areas nearest the facility are villages located on each fork of the fjord. The facility and Reydarfjordur village (population 632, [Reydaral 2001]), approximately 6 km to the west of the facility, are located on the northern side of the major east-west fork, Reydarfjordur fjord. Eskifjordur village (population 972, [Reydaral 2001] is on the eastern side of the minor fork, Eskifjordur fjord, approximately 5 km northeast of the facility. Figure 2-2 depicts the entire area of the air dispersion modeling and the individual receptor location grid modeled by Earth Tech, and the major human land uses within the modeled area. The modeling analysis evaluated the potential for air impacts related

to emissions of PAHs (total PAH and carcinogenic PAHs [cPAH]), particulate fluoride and hydrogen fluoride (PF/HF), SO_2, and particulate matter (PM_{10}) (Earth Tech 2005a,b,c, Scire 2006a, pers. comm.). The potential for receptors to contact CoPCs in the air or via soil deposition is evaluated in the HHRA. Figure 2-3 is a schematic representation of these potential exposure pathways.

The immediate area surrounding the facility is not inhabited, and future residential use is considered unlikely because of the location and the formal regulatory restrictions on its use. Specifically, a dilution zone (Figure 2-2) has been established in the Environmental Operating Permit according to Icelandic regulations (Reydaral 2001). This area is off limits to residential and agricultural or rangeland use. The dilution zone allows dilution of chemicals to take place and stipulates that chemical concentrations from the facility emissions may exceed environmental limits within the area (Alcoa 2005). The limits used to define these permissible maximum pollution levels are divided into two categories: vegetation protection limits, which are set to protect vegetation, and health protection limits, which are set to protect human health. It is necessary to abide by both limits outside the dilution zone.

The current water source for Reydarfjordur village is located in the river delta of Njorvadalsa, and no chemical contamination of the water was indicated in a study done in the summer of 1990 (Reydaral 2001, p. 37) or in preliminary data for monitoring in the fjord (RETEC 2005). The water is not fluoridated (RETEC 2005; Birgisson 2005, pers. comm.). Residences outside of the village get their water from local groundwater wells or a spring close by. There is no indication that the facility will affect these resources and therefore ingestion of water from these sources is not included in the analysis.

The diet of residents in the region is similar to a western-based diet. Locally available foods include fresh fish, locally raised lamb, potatoes, rhubarb, and wild berries. In all of eastern Iceland, ptarmigan, wild geese, and reindeer are hunted and eaten. Farming, in particular sheep farming, in the area has declined because of decreased agricultural subsidies and deteriorating lamb markets (Nysir 2002, p. 14). The farms closest to the facility at the time of start-up that currently raise sheep are located at Kollaleira on the western edge of Reydarfjordur village, Sletta, south of Kollaleira at the end of the fjord (Figure 2-4), and Pernunes, across the fjord and southeast of the facility. There is some hypothetical potential for exposure to constituents from the facility through deposition onto the soil and plants and subsequent uptake through food crops. This potential for exposure was evaluated through consideration of estimated fluoride and PAH concentrations in soil.

The culture of the region is based on local fisheries and farming (Reydaral 2001). Fishing currently drives the economy of the region. Aerial deposition of fluorides, SO_2 and PAHs in the fjord seawater surrounding the facility will be subject to dispersion and dilution. Deposition of fluoride in waters of the fjord in the ranges predicted by the air modeling is expected to have insignificant effects on ambient fluoride concentrations. Seawater typically contains fluoride concentrations of 1.2–1.5 mg/L (ATSDR 2003). By comparison, the highest predicted deposition rate (annual mean) for total fluoride in the entire modeling grid is 0.0183 $\mu g/m^2/s$, which would result in an annual addition of only 0.06 mg/L over the top 10 m of the water column. Actual addition of fluoride is expected to be much lower. For example, mean annual deposition of fluoride over the entire modeling grid is 0.001 $\mu g/m^2/s$. Long-term accumulation

would not be expected because fluoride in seawater rapidly forms stable complexes with calcium and magnesium, and calcium carbonate would drive the transfer of fluoride in its dissolved form to incorporation into sediment.

Deposition of PAHs in waters of the fjord in the ranges predicted by the air modeling is also expected to have insignificant effects on ambient PAH concentrations. The highest estimated deposition rate (annual mean) for PAHs in the entire modeling grid is 0.000019 $\mu g/m^2/s$, which would result in an annual addition of only 0.00006 $\mu g/L$ over the top 10 m of the water column. PAHs are highly hydrophobic and will rapidly become associated with organic and inorganic particles in the water column, where they would be steadily transferred to the sediment or flushed out of the fjord. The loading of PAHs to the fjord from aerial deposition is insignificant compared to the estimated loading via the use of seawater scrubbers. Exponent (2005) determined that loading of PAHs to the fjord by seawater scrubbers would result in no appreciable ecological effects.

Although fish take up PAHs, concentrations of PAHs are reduced or eliminated by metabolism in fish tissue (ATSDR 1995). Because of this metabolism, PAHs would not be expected to accumulate to any measurable degree in edible fish tissue. Thus consumption of fish by people is not a likely exposure route for human receptors. Food chain uptake does not appear to be a major source of exposure to PAHs for fish. The rapid dilution of fluoride and SO_2 to background concentrations coupled with the movement of fish through the area, further limiting their exposure, suggest that adverse effects would be unlikely at the concentration estimates predicted in the fjord.

2.4.2 Potential Receptors

Given the site characteristics including current use and future use, and considering the dilution zone described above, the most likely potential human receptors include onsite workers and residents or farmers who live near the site. Onsite workers whose responsibilities are primarily outdoors are likely to be the receptor population with the highest exposure potential. Although workers will be protected through adherence to strict occupational health standards, this risk assessment evaluated potential health risks in the absence of such controls as a means to provide information to risk managers. Future onsite residential use of this site is highly unlikely; however, some interest has been expressed in the potential for future residential use of the Fjardaal Team Village (FTV), which is a temporary community for Fjardaal project workers located just west of the facility.[1] Therefore, to provide information regarding this potential use, the risk assessment includes consideration of an FTV hypothetical resident. Potential risks related to facility emissions for seagoing workers along the fjord are also evaluated. Identified residents in the villages and farming areas could potentially be exposed to CoPCs generated from the site. Locations of residences and farms were collected (Agustsdottir 2005, pers. comm.; Birgisson 2005, pers. comm.), mapped, and were considered in the risk assessment process. Potential exposures for visitors in the Holmanes Reserve area were also considered. Thus the following receptors were considered in the risk assessment process:

[1] www.fjardaal.com/documents/en-FactSheetFTV.pdf.

- Onsite outdoor worker
- Seagoing worker
- Hypothetical maximum exposed individual (MEI) residing at the facility fenceline
- Future hypothetical resident at FTV
- Closest resident (former residence at Teigagerdi)
- Residents in Reydarfjordur and Eskifjordur villages
- Closest agricultural farmer
- Visitor to the Holmanes Reserve.

Figure 2-4 shows the locations of these potential receptors. The first step of the HHRA was a screening process that identified potential receptors and chemicals to evaluate further in the quantitative risk assessment. Specifically, concentration estimates for SO_2, fluoride, and particulates in ambient air were considered in comparison with all relevant standards; PAHs were compared with health protective screening values in air and in soil (Figures 2-5 through 2-17). This analysis is described in Section 3.1 and as indicated there, the concentration estimates were well within or below all relevant standards and screening levels for most of the potential receptors. Modeled ambient air concentration estimates for SO_2, fluoride, and particulates did not exceed relevant standards in either scenario, with or without seawater scrubbers. PAH concentrations were well below the risk-based concentration (RBC) for PAHs identified by U.S. EPA and were also below the lower end of the range of BaP standards identified in the EIA. Although the screening indicated that risks would be lower than the 10^{-6} level, risk estimates were calculated to provide further information to risk managers. Risk estimates were derived for the following receptors:

- Onsite outdoor worker—potential inhalation of PAHs in air
- Seagoing worker—potential inhalation of PAHs in air
- Hypothetical MEI residing at the facility fenceline—potential inhalation of PAHs in air.

The key receptor populations are discussed further here.

Future Onsite Outside Worker. A future onsite outdoor worker scenario was evaluated for the facility area, in which adult workers are exposed to PAHs in outdoor air and in surface soil from aerial deposition. Surface soil exposure pathways considered included inhalation, ingestion, and dermal contact.

Seagoing Worker. A future seagoing worker scenario was evaluated for the facility area, in which adult workers are exposed to PAHs in air during outdoor work on the dock, on transport vessels, or fishing within the area of the facility. Most of the fishing inside the fjord is on long

line and is done mainly on the south side, near the mouth of the fjord. This kind of fishing is practiced year round. These fishing boats go out during the day to set and retrieve their lines. The fishing vessels typically sail past the plant on their way in and out the fjord, and do not stay within the dilution zone for any extended period of time. There are some less used fishing grounds off Holmanes Nature Reserve. Gill nets and trawls are also used, but to a lesser extent (as cited in Reydaral 2001). Hook-and-line fishing occurs mainly in the summer and mostly outside the fjord near the island Skrudur. An area of the fjord (Figure 2-4) has been identified to represent the area evaluated in the risk evaluation for seagoing workers.

Maximum Exposed Offsite Individual (Fenceline Receptors). The MEI represents a hypothetical resident (adult or child) who resides at a receptor location within the dilution zone, yet not on facility property. Note that current regulations preclude habitation within the dilution zone, thus this receptor represents a worst-case exposure scenario. Potential exposure pathways evaluated include exposure to PAHs through inhalation of ambient air. In addition, the assessment considered deposition of PAHs onto soil and subsequent exposure through inhalation of, ingestion of, and dermal contact with surface soil. By definition the MEI is intended to represent the worst-case scenario in terms of potential health risks. All of the other receptors, other than the onsite worker, are located further from the facility and their magnitude of exposure, and potential risks, are considered to be lower.

2.5 Ecological Conceptual Site Model

This section presents a CSM that describes pathways and mechanisms by which ecological receptors can be exposed to chemicals released in aerial emissions from the Fjardaal smelter. A CSM is a planning tool useful for identifying chemical sources, complete exposure pathways, and potential receptors on which to focus the risk assessment. The purpose of a CSM is to ensure that all potential pathways are considered, regardless of whether those pathways are complete. The following sections characterize the terrestrial habitats and ecological receptors in the vicinity of the smelter and identify potential pathways by which key receptors may become exposed to aerial emissions.

2.5.1 Environmental Setting

The smelter is being constructed on the Hraun industrial tract in the municipality of Fjardabyggd, on the east coast of Iceland (Figure 1-1). The site is about 5 km east of the village of Reydarfjordur. The site lies on the north shore of the Reydarfjordur fjord, an inlet of the North Atlantic Ocean. The location of the smelter is on relatively flat land that slopes gradually toward the coast, but the fjord is surrounded by mountains up to 1,000 m high.

The land in the immediate vicinity of the smelter site is largely undeveloped and sparsely populated. There is no active agriculture near the site. Tree farming has been started at Teigagerdi. Grazing will be prohibited in a 3 km zone around the smelter once it is operational. The remainder of the land is primarily natural, and includes the Holmanes Nature Reserve to the east of the smelter site.

2.5.2 Terrestrial Vegetation Communities

Vegetation communities in the area surrounding the smelter site have been well characterized through studies conducted by Natturustofa Austurlands (East Iceland Environmental Research Institute) as part of the EIA conducted in advance of smelter construction, and results have been summarized by Jonsdottir (1999). Key findings from the investigation are summarized here.

The area of the smelter consists of a mosaic of predominantly grassland, heather/heath, moss, wetland, and lichen plant communities (Figure 2-18). Heather/heath and grasslands are widely distributed and are the most common vegetation types. Heather/heath generally grows in dry and shallow soil. This is a diverse community consisting of dwarf shrubs, grasses, sedges, and rushes, although most species other than mosses tend to have a low abundance. Dwarf shrub heath is also widely distributed (especially on hill slopes), but is rarely found in large unbroken areas. Natural and cultivated grasslands are scattered throughout the area, predominantly along the coasts and lower mountain slopes, but also interspersed among the moss heath and dwarf shrub heath vegetation. Wetlands are generally found beneath mountain slopes above rock ridges. In areas where farms have been abandoned, grassfields are no longer harvested. Jonsdottir (1999) noted that active grass harvesting still occurred around Holmar and Teigagerdi and at farms west of Reydarfjordur, and horse grazing occurs on grassfields around Flateyri and Hraun.

Mosses are the dominant plant form in many vegetation types, and in some types, such as moss heath, they are the only plant form growing in some areas. In all, 99 moss species, 89 lichen species, and 190 vascular plant species were recorded in the EIA survey. Lichens were less widely distributed and were predominantly seen in dwarf shrub heath habitat. No rare or protected species were found in the area of investigation.

2.5.3 Terrestrial Wildlife

Few terrestrial bird species appear to inhabit the project area. Stefansson and Porisson (1999) conducted a survey of bird species within an area extending from Bjargtangar to Holmanes. The authors recorded a total of 34 species, the majority of which were seabirds, waterfowl, and wading birds that would be more commonly associated with the open waters and shoreline of the fjord than with the terrestrial habitats around the site. Several species that have been confirmed or are likely to breed in east Iceland, such as the common raven (*Corvus corax*), meadow pipit (*Anthus pratensis*), and northern wheatear (*Oenanthe oenanthe*) could occur in heath and grassland habitats in the vicinity of the smelter. The redwing (*Turdus iliacus*) has also been confirmed to breed around Reydarfjordur, although this species prefers birch woods, which suggests a limited distribution around the smelter site because of the limited extent of this habitat relative to other plant community types (Jonsdottir 1999). The ptarmigan (*Lagopus muta*) is a common bird of heath and scrub habitat elsewhere in Iceland (Vuijk 2005), but Stefansson and Porisson (1999) observed only one individual in their survey and classified the species as only possibly breeding in the area.

Terrestrial mammalian species diversity is low in Iceland. Reindeer (*Rangifer tarandus*) occur in the Reydarfjordur area during the winter and small numbers may occasionally pass through the area of the smelter, although their presence in the area, if any, would likely be of a very

limited duration (Reydaral 2001). Arctic fox (*Alopex lagopus*) are widely distributed in Iceland, and likely occur in natural habitats around Reydarfjordur. Most small mammal species found in Iceland, such as house mouse (*Mus musculus*), brown rat (*Rattus norvegicus*), and black rat (*R. rattus*) live commensally with humans and would not be expected to occur away from human habitation. The wood mouse (*Apodemus sylvaticus*) is the only small mammal that likely occurs in natural habitats around the smelter.

2.5.4 Potential Exposure Pathways

An exposure pathway is the course a chemical takes from a source to an exposed receptor. Exposure pathways consist of the following four elements: 1) a source; 2) a mechanism of release, retention, or transport of a chemical to a given medium (e.g., air, soil); 3) a point of contact with the medium (i.e., exposure point); and 4) a route of exposure at the point of contact (e.g., absorption, ingestion). Only those exposure pathways judged to be potentially complete are of concern for ecological receptors. Potential pathways by which ecological receptors may be exposed to chemicals in aerial emissions from Fjardaal are illustrated in Figure 2-19.

Primary exposure pathways are those expected to contribute most to risk estimates, while secondary exposure pathways are not expected to contribute substantially to risk. Primary exposure pathways for plants are absorption by vegetative surfaces of chemicals from aerial emissions and uptake from soil via the roots. Terrestrial organisms, such as soil fauna, can be exposed to chemicals in soil (e.g., fluoride and PAHs) either through direct contact or ingestion of soil. Terrestrial invertebrates can also be exposed to chemicals through ingestion of plant material. Birds and mammals are primarily exposed to chemicals through ingestion of plant material and incidental ingestion of soil. Birds and mammals can also be exposed via inhalation of chemicals in air, although this pathway is considered a secondary exposure route relative to the exposure likely received via the diet.

2.5.5 Potential Ecological Receptors

The ecological receptors with the greatest likelihood of exposure to chemicals in aerial emissions are the vegetative communities that occur in areas around the plant. As discussed above, there is a mosaic of plant communities in the vicinity of the smelter site composed of a fairly diverse assemblage of mosses, lichens, grasses and sedges, and shrubs, with some trees. All these species are potentially exposed to chemicals in air and also to chemicals that deposit on land and become incorporated in soil. Thus, the plant communities that surround the smelter site are a key focus for this screening assessment, in particular moss and lichen species because of their sensitivity to chemicals such as fluoride and SO_2.

Terrestrial bird species are likely to be present and utilize the plant communities within the site, however most are not predominantly herbivorous. For example, the raven is primarily a carrion-eater, the Northern wheatear is insectivorous, and the redwing is primarily insectivorous, but does incorporate plant matter into its diet. However, the ptarmigan is typically considered an herbivorous bird in the Iceland fauna, but appears to be uncommon in the area around Reydarfjordur (Stefansson and Porisson 1999). The reindeer, while strictly herbivorous, is a wide-ranging species that is, at most, transient in the area, and its exposure to

chemicals in plants would be limited to short periods during foraging within the project area. Of all terrestrial receptors, the wood mouse is likely the most highly exposed to chemicals in plants and soil, as it is a resident species with a limited home range, and its diet is primarily vegetarian, although it will consume earthworms and insects in times of food scarcity.

3 Human Health Risk Assessment

The purpose of the HHRA is to evaluate the likelihood that health effects could occur in people who come into contact with the CoPCs associated with Fjardaal air emissions under two potential scenarios. Specifically, the HHRA was conducted to determine whether there are consequential differences in risks with and without seawater scrubbers. The Fjardaal HHRA uses standard procedures developed by the U.S. Environmental Protection Agency (U.S. EPA) adapted where appropriate to the specific conditions of the site (cited below). A prior air modeling analysis conducted by Earth Tech (2005a,b) did not identify any instances where applicable standards would be breached. However, this risk assessment is being conducted to compare risks with and without seawater scrubbers and is another means to evaluate potential risks and potential exposure pathways that may not be considered in the context of the available standards.

Site background information was summarized in Section 1.1, and Section 2.4 provides the human health CSM. Sections 3.1 through 3.4 describe the results of the four steps recommended in U.S. EPA guidance for risk assessment:

- Identification of CoPCs and comparison with standards
- Exposure assessment
- Toxicity assessment
- Risk characterization.

An uncertainty assessment is included in the risk characterization to place potential risks in context. The uncertainty assessment discusses HHRA assumptions that may lead to over- or underestimates of potential site risks.

This risk assessment includes a comparison of estimated concentrations with relevant standards from Iceland, Norway, and U.S. EPA. No Icelandic guidance for risk assessment was identified in this review. The European directives on which the Icelandic standards were based were reviewed and standard values were included in the screening assessment (EC 1999; EU 2004) as well as the standard listed in the original EIA (Reydaral 2001) and air modeling (Earth Tech 2005a) reports. It should be noted, however, that the PAH standard considered in the previous EIA has subsequently been revised, so both values are considered for completeness and consistency with the current EIA evaluation. The risk assessment methods and text here reference risk assessment methods used by U.S. EPA and draw from the following documents:

- *Risk Assessment Guidance for Superfund: Volume 1: Human Health Evaluation Manual* (Part A) (U.S. EPA 1989)
- *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance "Standard Default Exposure*

Factors" Interim Final (OSWER Directive # 9285.6-03) March 1991 (U.S. EPA 1991)

- *Exposure Factors Handbook* (U.S. EPA 1997b)
- U.S. EPA Integrated Risk Information System files (U.S. EPA 2005a)
- U.S. EPA Region 9 preliminary remediation goals table (U.S. EPA 2005b), that provides RBCs.

3.1 Identification of Contaminants of Concern and Relevant Criteria

The air dispersion and deposition modeling data tables generated by Earth Tech for PAHs, SO_2, PF/HF, and PM_{10} with and without seawater scrubbers were used to identify CoPCs (Earth Tech 2005c). The model was processed with deposition in the model to account for any soil to human exposure pathways. Screening of PM_{10}, SO_2, PF/HF, PAHs, and cPAH air concentration estimates against toxicity criteria is described in Section 3.1.1. Screening of soil concentration estimates is described in Section 3.1.2.

The modeling by Earth Tech was described in detail in their prior assessment without deposition (Earth Tech 2005a, 2006). Earth Tech provided data files that included estimated ambient air concentrations and deposition rates for the CoPCs, and are summarized in Table 2-2. As described earlier, these data are separated into a base case representing operating conditions without seawater scrubbers and the alternative case, with seawater scrubbers. As a result of using seawater scrubbers, there was a decrease in estimated SO_2 air emissions from the fume stack. The modeling results were loaded into a database and coded according to receptor location. The following areas of receptors were identified in Figure 2-4: 1) within the facility, 2) outside of the facility but within the dilution zone, 3) the area of the fjord waters from the northern shoreline to the southern one bounded by a north south line 1.5 km to the east and west of the facility, 4) the Holmanes Reserve, 5) the FTV, 6) a 200-m radius around the closest former residence (Teigagerdi), 7) a 200-m radius around the closest farm (Kollaleira), 8) Reydarfjordur village, and 9) Eskifjordur village.

Section 3.1.1 presents a summary of the air modeling data and the methodology for identifying the CoPCs. Section 3.1.2 identifies the estimated deposition rates available from Earth Tech (2005c), a description of the calculations used to model soil concentration estimates, and a screening of the estimated soil concentrations against residential soil RBCs.

3.1.1 Air

In order to identify CoPCs in air, the modeled air concentration values within each receptor location were compared with the human health criteria compiled from Iceland, Norway, Europe, and U.S. EPA (Table 3-1). Figures 2-5 through 2-17 show the statistics for ambient air concentration estimates for each chemical at each receptor location both with and without seawater scrubbers. Air concentration estimates were compared to the relevant standards for

SO_2, hydrogen fluoride, and PM_{10}). Air concentration estimates for PAHs were compared with health-protective screening concentrations (i.e., RBCs derived by U.S. EPA Region 9 [U.S. EPA 2005b]).

Specifically, all concentration estimates for PAHs in air (Figures 2-10 and 2-11, Table 3-2) were compared with U.S. EPA-derived RBCs for residential air. This is a conservative means of evaluating contact with air because these values are based on daily exposure to PAHs in air in a residential scenario, whereas exposures for workers would be shorter in frequency and duration. Consequently, the use of residential screening numbers is expected to provide a conservative (i.e., health protective) means to screen the site data to identify CoPCs for nonresidential uses evaluated in the HHRA. The RBC for BaP is based on a target excess cancer risk of 10^{-6} and the RBC for fluoride is based on a hazard index of one. Each chemical is discussed in the following sections.

3.1.1.1 PAHs

Composition of PAHs—Most of the PAHs in aerial emissions from aluminum smelters originate from the coal tar that is typically used as a binder in anode manufacturing. Raw materials for anode paste consist of high-grade coke (petroleum and pitch coke) and coal tar pitch, mixed with typical ratios of 75:25 to 80:20 coke to pitch. PAHs are released from coal tar pitch when the anode temperature is raised to the required temperature for the electrolytic process to proceed. Generally, dry scrubbers are efficient at removing large- and medium-sized particles and inorganic compounds. Because higher molecular weight PAHs generally have the highest affinity for particles, they are more likely to be removed by the scrubbers than lower molecular weight PAHs. Thus, retention of PAHs by emission control systems is expected to vary more or less in relation to molecular weight.

Figure 3-1 presents graphs of the percent composition for total PAHs (gaseous plus particulate) in potroom roof vent samples and stack samples from the Alcoa Deschambault smelter in Quebec, Canada. The Deschambault smelter is similar in design, operation, and capacity to Fjardaal and is therefore expected to serve as a reasonable representation of aerial emissions from Fjardaal. As is apparent from these data, it is difficult to determine a complete description of the PAH composition of aerial emissions from aluminum smelters because most PAHs cannot be detected in environmental samples. However, it is apparent from these data that PAHs with molecular weights greater than pyrene (molecular weight 202) are generally not detected in aerial emissions. This is consistent with the expectation that dry scrubbers will preferentially remove higher molecular weight PAHs. Emissions from Deschambault potroom roof samples are dominated by phenanthrene, fluorene, and fluoranthene where these PAHs make up more than 94 percent of the total PAH measured (assuming undetected PAHs were present at one-half the detection limit). Emissions from Deschambault stack samples are dominated by phenanthrene, fluorene, acenaphthene, naphthalene, and fluoranthene where these PAHs make up 67 percent of total PAHs (assuming undetected PAHs are present at one-half the detection limit).

In order to calculate risk estimates for PAHs, it is desirable to have data for all of the cPAHs (i.e., benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, BaP, chrysene, dibenz[a,h]anthracene, indeno[1,2,3-cd]pyrene) and for each of the PAHs thought to have

noncarcinogenic effects (i.e., acenaphthylene, acenaphthene, anthracene, benzo[ghi]perylene, fluoranthene, fluorene, naphthalene, phenanthrene, and pyrene). Although the undetected results for cPAHs in the Deschambault data limit the potential to generate precise estimates, these data do provide an indication of the upper-limits of the percent of cPAHs in the PAHs analyzed at that facility. Thus, even though the cPAHs were largely undetected and may not even be present, they were evaluated here through calculations including the undetected values at one-half the detection limit. Data were available for both gaseous and particulate PAHs from both of these sources (i.e., roof particulate, roof gases, stack particulate, stack gases).

Tables 3-3 and 3-4 show the Deschambault data for roof samples and stack samples, respectively, and show the calculation of the percent of cPAHs from those data. These percentages represent the adjusted concentration of all cPAHs. Specifically, concentrations of each of the cPAHs were divided by the total PAH concentration to derive a percentage. Next the cPAH percent concentrations were adjusted to reflect the relative carcinogenic potency as identified by U.S. EPA (2005b). The relative potency estimates were derived by EPA to reflect the potential carcinogenicity of each of the cPAHs relative to the potency of BaP. In order to adjust each cPAH percentages to reflect the potency of individual PAHs relative to BaP, the cPAH percentages were multiplied by the relative potency estimate. Then all of the adjusted percentages were summed to derive four estimates of the percentage of cPAH for the four data sets (i.e., roof particulate, roof gases, stack particulate, stack gases). As indicated in Tables 3-3 and 3-4 the cPAH percentages ranged from 0.011 to 6.5 percent.

Earth Tech (2005a,b,c, 2006) modeled total PAH concentrations in air and in deposition rates. Subsequently, Earth Tech also modeled cPAHs for the purposes of the HHRA (Scire 2006a,b, pers. comm.) using the percentages of cPAH described above in the following application (Scire 2006a, pers. comm.):

- Lines (from roof data): gas/particle split of total line source PAH: 97 percent/3 percent:
  - Percentage of line source gas PAH that is cPAH: 0.011
  - Percentage of line source particle PAH that is cPAH: 2.3
- Points (from stack data): gas/particle split of total point source PAH: 46.7 percent/53.3 percent
  - Percentage of point source gas PAH that is cPAH: 6.5
  - Percentage of point source particle PAH that is cPAH: 5.6.

The model estimates for cPAHs in air resulting from this application are shown in Figure 2-10.

To evaluate the noncarcinogenic effects of PAHs, the total PAH estimate from Earth Tech was adjusted by Exponent using the assumption that the nine noncarcinogenic PAHs (represented by naphthalene, see discussion below), made up 70 percent of total PAH emissions. Because measured emissions of noncarcinogenic PAHs at Deschambault were much lower than 70 percent of the total PAHs, this is a health protective assumption. Modeling with seawater

scrubbers results in slightly higher PAH concentrations at certain locations within the modeling grid as a result of a number of factors, predominantly building downwash and differences in stack height between the fume stack and the four seawater scrubber stacks (Scire 2005, pers. comm.).

Identification of PAH CoPCs—In assessing carcinogenic risk, the model estimates for cPAHs were compared with three values: 1) the lower end of the range of BaP standards (0.0001 $\mu g/m^3$) used in the original EIA (Reydaral 2001) and air modeling reports (Earth Tech 2005a), which represents the most stringent criterion to be found in Europe; 2) the current European Union standard (0.001 $\mu g/m^3$); and 3) the U.S. EPA RBC of 0.00092 $\mu g/m^3$ (U.S. EPA 2005a). No receptor locations exceeded any of these standards or the RBC (Figures 2-10). However, to provide information to risk managers, risks to the onsite worker, the MEI at the fenceline, and the seagoing worker will be assessed further in the risk characterization section. Similarly, for noncarcinogenic risks, there are no receptor locations that have annual ambient air values that exceed the most conservative U.S. EPA inhalation RBC (3.1 $\mu g/m^3$) for naphthalene (U.S.EPA 2005b) (Figure 2-11). Nevertheless, the noncancer hazards were evaluated in the risk assessment to provide a basis for comparison between the base case (without seawaters scrubbers) and the alternative case (with seawater scrubbers).

3.1.1.2 Sulfur Dioxide

Air concentration estimates of SO_2 were all below the health protective standards and much further below levels at which human health effects are observed. Ambient air concentration estimates were screened against the available annual, 24-hour, and 1-hour SO_2 Icelandic standards as well as the annual, 24-hour, and 3-hour U.S. EPA National Ambient Air Quality Standards (NAAQS)[2] (Table 3-1; Figures 2-5 through 2-8). U.S. EPA has set NAAQS for six principal pollutants including SO_2, which are called "criteria" pollutants. The primary NAAQS (annual and 24-hour standards) are set to protect public health by providing a margin of safety to protect the health of sensitive populations such as asthmatics, children, and the elderly. Adverse effects under consideration are lung irritation and aggravation of bronchitis. The secondary NAAQS (3-hour standard) is set to protect public welfare, including protection against visibility impairment, damage to animals, crops, vegetation, and buildings. A statistical summary of SO_2 concentration estimates at receptors with and without seawater scrubbers can be found in Table 3-5.

Annual ambient air concentration estimates were below the Icelandic regulations and U.S. EPA NAAQS for human health effects, modeled both with and without seawater scrubbers (Table 3-5 and Figure 2-5).

The Icelandic 24-hour average SO_2 standard value (50 $\mu g/m^3$) allows up to three exceedances per year and was not surpassed when modeled either with or without the seawater scrubbers (Table 3-5 and Figure 2-6). When modeled without seawater scrubbers, there were more receptor locations that exceeded the value: 1 day at 308 receptors and 2 days at 6 receptors; however, these are within the number of allowed exceedances per year. The U.S. EPA 24-hour NAAQS value was not exceeded.

[2] www.epa.gov/air/criteria.html

The maximum 3-hour average SO_2 concentration estimates were below the U.S. EPA secondary NAAQS (1,300 $\mu g/m^3$) for human health effects, estimated for both with and without seawater scrubbers (Figure 2-7, Table 3-5). No Icelandic regulation regarding a 3-hour period was identified.

The Icelandic 1-hour average SO_2 standard value allows up to 24 exceedances of 350 $\mu g/m^3$ per year (Table 3-1). The maximum number of days that had air concentration estimates that exceeded this value at any location was 1 day when modeled with seawater scrubbers and 5 days when modeled without. When modeled without seawater scrubbers, there were more locations that exceeded the standard value; however, all were within the number of allowed exceedances per year (Table 3-5 and Figures 2-8 and 2-9). There is not a U.S. EPA 1-hour NAAQS value for SO_2.

3.1.1.3 Fluorides

The ambient air concentration estimates for 24-hour hydrogen fluoride were below the Norwegian guidelines for protection of human health for fluorides in air. The distribution for annual, 24-hour, and 1-hour air concentration estimates with and without seawater scrubbers can be found in Figures 2-12 to 2-15. There are no U.S. health criteria for hydrogen fluoride in residential air. See Section 3.3.2 for a further discussion on readily available guidelines. Risk resulting from inhalation at these levels is considered negligible.

3.1.1.4 Particulate Matter

Both PM_{10} annual ambient air concentration estimates and 24-hour ambient air mean concentration estimates fell below the low end of the Icelandic standards and below the U.S. EPA NAAQS (U.S. EPA 2005c) (Figures 2-16 and 2-17). PM_{10} includes all particles with a diameter less than 10 μm. $PM_{2.5}$ was not modeled by Earth Tech (2005a,b,c) and is not a required parameter of monitoring within Iceland. Because levels of PM_{10} are very low, a risk from $PM_{2.5}$ is not expected.

3.1.2 Soil Concentration Estimates for PAH and Fluoride

In order to estimate potential soil concentrations of chemicals that could result from facility operations, Earth Tech (2005c) included wet and dry deposition in their modeling runs with and without seawater scrubbers. The maximum deposition rates ($\mu g/m^2$-s) from within the facility and in the dilution zone were used to calculate the soil concentration estimates for the chemicals that had oral toxicity values that could be used in HHRA (i.e., fluorides and total PAHs). These estimated deposition rates represent the highest rates modeled within the receptor grid and in general decrease with distance from the facility.

3.1.2.1 Method

Two scenarios were modeled in this assessment. The first involves estimated deposition on bare soil. The second involves estimated deposition on generalized grass tissue that was deposited onto soil upon senescence. The accumulation of PAHs and fluoride in soils is governed

predominantly through wet and dry deposition. Under conditions similar to this site, gaseous concentrations of PAHs and hydrogen fluoride do not partition significantly to the terrestrial surface. This is because of the low inherent vapor pressure and comparatively larger surface area between the airborne particles relative to the terrestrial surface. Hence, the predictions of total PAH and fluoride accumulation in the facility soils were determined based exclusively on a pathway of vapor partition to particulate (wet or dry) and deposition of particulate to bare soil. This was done as an equilibrium model assuming constant annual deposition rates. As such, the equilibrium concentration can be defined as the concentration where the rate of deposition equates to the rate of elimination. The rate of deposition on bare soil was determined as follows:

$$k_{in}\left(\mu g \diagup kg\text{ - }day\right) = k_{dep} \times \frac{1}{D} \times M_{soil}$$

where:

k_{in} = rate of PAH or fluoride accumulation in soils (μg/kg-day)

k_{dep} = rate of aerial deposition ($\mu g/m^{-2}$-day)

D = standardized soil depth of the mixing zone (0.1 m)

M_{soil} = density of soil (m^3/kg).

On vegetated soils, the rate of addition of PAH or fluoride input to the soil is equivalent to the concentration of PAH or fluoride in or on the plant and the time weighted rate of conversion from standing biomass to detritus. This calculation was performed only for grasses because grasses will likely be the predominant type of vegetation within the plant boundary and dilution zone. The deposition rate for fluoride and PAH concentrations was determined as follows:

$$k_{dep-p}\left(\mu g \diagup kg\text{ - }day\right) = f([F]_{air}) \times \frac{BM_a}{L} \times \frac{1}{D} \times M_{soil}$$

where:

k_{dep-p} = rate of PAH or fluoride deposition to soil from plants (μg/kg-day)

$f([F]_{air})$ = concentration of PAH or fluoride in or on plant matter for a given aerial air concentration (μg/kg).

BM_a = area-based plant mass (kg/m^2)

L = senescence rate (days)

D = standardized soil depth of the mixing zone (0.1 m)

M_{soil} = density of soil (m^3/kg).

Derivation of the plant concentrations is outlined in Section 4.3.

The elimination of chemicals of concern from the soil was modeled based on two processes. The first is bulk transport with precipitation. This was applied for both fluoride and PAHs. For this elimination mode, the proportion of elimination is proportional to the concentration of PAHs in the soil, the partition coefficient for soil versus water and the daily probability of rain. For PAHs, the organic carbon partition (K_{oc}) for BaP was applied. For fluoride, it was assumed that hydrogen fluoride in runoff/leachate was completely miscible in water and that each rainfall resulted in a uniform removal from 12.5 to 100 percent. The algorithms used were as follows:

$$\text{For total PAHs}: \quad k_{out\,1}\left(\mu g \big/ \text{kg - day}\right) = [COC]_{Soil} \times k_{oc} \times \text{P(Precip)}$$

$$\text{For fluoride}: \quad k_{out\,1}\left(\mu g \big/ \text{kg - day}\right) = [COC]_{Soil} \times e^{\ln\{1\ldots8\}\cdot \text{P(Precip)}}$$

where:

$k_{out\,1}$	=	rate of PAH or fluoride elimination through bulk transport (μg/kg-day)
$[COC]_{Soil}$	=	PAH or fluoride soil concentrations (μg/kg)
k_{oc}	=	organic carbon partition coefficient (μg/μg)
P(Precip)	=	standardized area of deposition (1/day).

The second mode of elimination from the soil is the result of biodegradation. This was applied only to PAHs because fluoride is not biodegradable. Based on modeling in EpiSuite (U.S. EPA 2000), the biological half-life of PAHs (using BaP as a sentinel constituent) in soil is approximately 60 days. Hence, the biological elimination rate was derived as follows:

$$k_{out\,2}\left(\mu g \big/ \text{kg - day}\right) = \frac{\ln(2)}{t_{1/2}}$$

where:

$k_{out\,2}$	=	rate of PAH elimination in soils through biodegradation (μg/kg-day)
$t_{½}$	=	biological half-life assumed for PAHs (days).

Overall soil accumulation was determined as the summation of the input and elimination coefficients that was solved as the limit as $t \rightarrow \infty$ as follows:

PAHs:

$$[PAH]_t = \int_{t=o}^{\infty} k_{in} \times dt - ([PAH]_{(t-dt)} \times k_{out\,1} \times dt + [PAH]_{(t-dt)} e^{k_{out\,2} \cdot dt})$$

$$[PAH]_t = \lim_{t \to \infty} \left(k_{dep} \times A \times L \times \Delta t - [PAH]_{(t-1)} \times \left[k_{oc} \times P(\mathrm{Precip}) \times \Delta t + e^{\frac{\ln(2)}{t_{1/2}} \times \Delta t} \right] \right)$$

Fluoride:

$$[F]_t = \int_{t=o}^{\infty} k_{in} \times dt - [F]_{(t-dt)} e^{k_{out\,2} \times dt}$$

$$[F]_t = \lim_{t \to \infty} \left(k_{dep} \times A \times L \times \Delta t - [F]_{(t-dt)} e^{\frac{\ln(2)}{t_{1/2}} \times \Delta t} \right)$$

Parameterization and results are provided in Tables B-4 and B-5 (Appendix B) for fluoride and PAHs, respectively.

3.1.2.2 Screening of PAH and Fluoride Concentrations in Soil

Figures 3-2 and 3-3 identify the soil concentration estimates for total PAH and hydrogen fluoride worst-case scenario, respectively, in comparison with the RBC for BaP and for fluoride in soil. These RBCs were derived by U.S. EPA Region 9 and incorporate health protective assumptions regarding the potential for exposure to chemicals in residential soils as a result of ingestion, dermal contact, and inhalation of particulates. The RBC for BaP is based on a target 10^{-6} risk and the RBC for fluoride is based on a hazard index of one. As can be seen in Figures 3-2 and 3-3, the soil concentration estimates are well below the RBCs. PAH concentrations were modeled as total PAHs and consequently could be considered to be at least an additional order of magnitude lower for BaP. In consideration of this factor, and given the relatively minimal soil concentration estimates, risks related to direct contact are expected to be well within acceptable limits.

The potential for the food-chain to be affected by PAHs and hydrogen fluoride is greatly reduced or eliminated by the relatively low contribution, the location of the higher deposition within industrial areas, and the degree to which PAHs are metabolized in tissues. Information on hydrogen fluoride concentrations in soil is somewhat limited but suggests that the contribution related to facility operations would be well below background levels. RETEC (2005) is undertaking baseline monitoring of soils and plants in the project area; however, the results of this sampling were not available at the time of this writing. Fluoride tends to transport and concentrate into the inorganic portions of soil. Average U.S. eastern and western fluoride concentrations were 340 and 410 mg/kg, respectively (ATSDR 2003). No data were found on the fluoride content of soils in Iceland during the production of this report. However, because of the volcanic activity and known emissions of fluoride in Iceland (Stone 2004), it is presumed that fluoride levels are similar if not higher than in U.S. soils. The maximum estimated

concentration that would result from the contribution of the planned facility is near 43 μg/kg (Figure 3-3), much lower than background concentrations in the United States.

3.2 Exposure Assessment

In an HHRA, exposure assessment is the process of identifying human populations that could potentially contact site-related CoPCs and estimating the magnitude, frequency, duration, and route(s) of potential exposures. An exposure pathway describes a chemical's transport from its source to a potentially exposed individual and must include a source, transport mechanism, receptor, and point of entry into the body. Only when each of these elements is present can an exposure pathway be complete, and only complete exposure pathways have the potential to result in health risk. Potential exposures associated with the CoPCs identified at the site are evaluated by identifying current and potential future uses of the property, those populations that could be exposed to the chemicals (i.e., the receptors), and the manner in which they may be exposed (i.e., the exposure pathway). The relevant potential exposure pathways are described in the CSM above and shown in Figure 2-3. This section describes the methodology used to quantify exposure pathways identified in the CSM. Consistent with guidance from U.S. EPA, reasonable maximum exposure (RME) estimates were applied for all complete exposure pathways. Exposure and risk estimates were derived using deterministic methodology and health protective assumptions identified by U.S. EPA.

3.2.1 Exposure Assumptions

As described previously, three exposure scenarios were evaluated: an onsite outdoor worker; a seagoing worker; and hypothetical residents at a location immediately adjacent to the site (adult and child scenarios). Exposure to PAHs in air was evaluated using the following general equation identified by U.S. EPA (1989):

$$\frac{C_{air} \times InhR_{air} \times EF \times ED}{BW \times LT}$$

where:

C_{air} = PAH (BaP or naphthalene concentration estimate in air [mg/m^3])

$InhR_{air}$ = inhalation rate (m^3/day)

EF = exposure frequency (days/year)

ED = exposure duration (years)

BW = body weight (kg) (70 kg for adult scenarios and 15 kg for child resident)

AT = averaging time (70 years × 365 days/year = 25,550 days for carcinogenic effects of BaP and exposure duration x 365 days for noncarcinogenic effects of naphthalene).

Table 3-6 provides all exposure assumptions applied in the risk assessment and Table 3-2 provides the air concentration estimates for PAHs.

Exposure frequency is the number of days of exposure per year assumed in the risk estimates. For the outdoor worker, 250 days per year was assumed, consistent with a work pattern of 5 days a week for 50 weeks per year and with exposure estimates for workers recommended by U.S. EPA (1989, 1997b). The seagoing worker was assumed to have a lower frequency of exposure of 100 days per year, consistent with time spent out of port. Workers who may spend their entire time outdoors at the docks would be covered under the exposure estimates for the facility outdoor workers. The exposure frequency for residents was assumed to be 350 days per year consistent with year-round exposure outdoors with two weeks per year away from home (U.S. EPA 1997b).

Exposure duration is the number of years that exposure is assumed to continue. For the worker scenario, a 25-year duration was applied (U.S. EPA 1997b). The residential exposure duration estimate of 30 years is consistent with upper-end estimates of the amount of time individuals in the U.S. typically live at one residence (U.S. EPA 1997b). The exposure duration estimate for a child was 6 years, based on 6 years of childhood.

Inhalation rates were based on health protective assumptions about exposure as identified by U.S. EPA Region 9 including 20 m^3 per day for an adult resident and 10 m^3 per day for a child (U.S. EPA 2005b). For the facility outdoor worker and seagoing worker, it was assumed that the entire work day was outdoors, which is likely an overestimate for most, if not all employees. The inhalation rate for these workers was derived assuming 2 hours of heavy activity at an inhalation rate of 2.5 m^3/hour and 6 hours of an average activity level with an inhalation rate of 1.3 m^3/hour (U.S. EPA 1997b, Table 5-23).

3.2.2 Exposure Concentration Estimates

Exposure concentration estimates were derived from air concentration estimates and are intended to represent concentrations experienced by the receptor over the exposure period (U.S. EPA 1989). In evaluating the RME exposure scenario, U.S. EPA guidance specifies the use of the 95 percent upper confidence limit (UCL) on the mean concentration. As recommended in *Supplemental Guidance to RAGS: Calculating the Concentration Term* (OSWER Publication 9285.7-081 [U.S. EPA 1992]), the concentration term is an estimate of the arithmetic average concentration for a contaminant based on a set of site sampling results. Because of the uncertainty associated with estimating a true average concentration at a site, the 95 percent UCL of the arithmetic mean should be used for this variable. The 95 percent UCL provides reasonable confidence that the true site average will not be underestimated.

Air concentration estimates from each of the areas under consideration were summarized separately. Summary statistics, UCL values, and the EPCs used in the HHRA are presented in Table 3-2, along with the distribution type and UCL calculation method used. U.S. EPA's ProUCL 3.0 software was used to calculate the summary statistics and UCL values. The UCL reported is the recommended method from the software, which is in accordance with the Risk Assessment Guidance for Superfund (RAGS) guidance.

3.3 Toxicity Assessment

The purpose of a toxicity assessment is to evaluate the potential for CoPCs to cause adverse health effects in exposed persons and to thoroughly define the relationship between the extent of exposure to a hazardous chemical and the likelihood and severity of any adverse health effects. The standard procedure for a toxicity assessment is to identify toxicity values for carcinogenic and noncarcinogenic effects and to summarize other relevant toxicity information. This section describes the methods used to evaluate toxicity that could result following oral, dermal, or inhalation exposure to CoPCs, and provides a brief toxicity profile for PAHs, PF/HF, PM_{10}, and SO_2. Section 3.5, *Uncertainty Assessment*, also discusses uncertainties in the toxicity values.

U.S. EPA-derived toxicity values used in risk assessments are termed cancer slope factors (CSFs), reference doses (RfDs), and reference concentrations (RfCs). CSFs are used to estimate the incremental lifetime risk of developing cancer corresponding to chronic daily intakes (CDIs) calculated in the exposure assessment. The RME potential for noncarcinogenic health effects is evaluated by comparing estimated daily intakes to the RfD and RfCs, which represent daily intakes at which no adverse effects are expected to occur over a lifetime of exposure. Both CSF and RfD/RfCs are specific to the route of exposure (e.g., inhalation exposure). For inhalation, unit risk factors for carcinogens and RfCs for noncarcinogenic effects were applied. In addition to the risk estimates, the basis for the standards for PM_{10}, SO_2, and hydrogen fluoride are briefly described here.

U.S. EPA sets NAAQS as required by the Clean Air Act as last amended in 1990[3] for pollutants considered harmful to public health and the environment (U.S. EPA 2005c). *Primary standards*, used in this screening risk assessment, set limits to protect public health, including the health of sensitive populations such as asthmatics, children, and the elderly.

Secondary standards set limits to protect public welfare, including protection against decreased visibility, damage to animals, crops, vegetation, and buildings. The U.S. EPA Office of Air Quality Planning and Standards set NAAQS for six principal pollutants, which include SO_2 and PM_{10}.

3.3.1 PAHs

The PAH compounds are a class of organic chemicals that share common structural features (two or more joined aromatic rings) and similar toxicological, physical, and chemical properties. They are formed during incomplete burning of coal, oil, gas, garbage, or other organic substances, and are present as the main constituents of creosote (creosote is produced from the high temperature treatment of coal, certain woods, and plants). PAHs do not typically occur alone but rather in mixtures of two or more compounds. In general, these compounds are absorbed into the body quickly and easily through inhalation, ingestion, and skin contact. Once in the body, PAHs are distributed to fatty tissue and are mostly stored in the kidneys, liver, and fat. Smaller amounts of PAHs may be stored in the spleen, adrenal glands, and ovaries. Animal

[3] www.epa.gov/oar/caa/

studies indicate that PAHs are not stored in the body for an extended period of time, but are excreted quite rapidly (ATSDR 1995).

As described above, both total PAH and cPAH concentration estimates (as BaP equivalents) were modeled. These data were used to derive health-protective estimates of risk for both carcinogenic and the noncarcinogenic effects of PAHs. There are seven established cPAHs (benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, BaP, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene). To evaluate the carcinogenic risks associated with PAH exposures, modeling data for combined cPAHs (expressed as BaP equivalent) were used in the risk assessment (See Section 3.1.1.1, above). To estimate noncancer effects associated with PAHs, total PAH estimates were adjusted using an assumption that 70 percent of the total PAHs were naphthalene, which represents the most conservative of the noncarcinogenic PAHs (acenaphthylene, acenaphthene, anthracene, benzo[ghi]perylene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene).

The current European Union ambient air target value for the protection of human health for BaP, 0.001 $\mu g/m^3$, is derived from a European Communities directive (Sigfusson 2005, pers. comm.; EU 2004). The lower end of the range of former ambient air standards used in the original EIA (Reydaral 2001) and air modeling (Earth Tech 2005a) reports (i.e., 0.001 $\mu g/m^3$ to 0.0001 $\mu g/m^3$) includes the most stringent criteria to be found in Europe (0.0001 $\mu g/m^3$). The U.S. EPA Region 9 RBC (U.S. EPA 2005b) for BaP residential soil is 0.062 mg/kg for soil and 0.00092 $\mu g/m^3$ in ambient air. cPAH estimates derived by Earth Tech (Section 3.1.1.1 above) were compared with the current standard, the former standard, and the U.S. EPA Region 9 RBC. For naphthalene, the RBC for residential soil is 56 mg/kg and 3.1 $\mu g/m^3$ in ambient air. The carcinogenic slope factor for BaP is 7.3 $(mg/kg\text{-}day)^{-1}$ and was derived by U.S. EPA based on data from mice dosed orally with BaP. They were found to develop excess cancers of the forestomach, squamous cell papillomas, and carcinomas (U.S EPA 2005a). Although there is no inhalation-based CSF for BaP at this time, inhalation risks were evaluated using route-to-route extrapolation as identified by U.S. EPA Region 9 (U.S. EPA 2005b). This approach assumes that the carcinogenic effects of a chemical or mixture operate independently of the route of exposure. Although there are some uncertainties related to this approach, and it may overestimate risks because some of the cancers used to derive the CSF were at the site of application, cPAHs are capable of causing cancers at sites remote from the site of application. Therefore, in order to provide information needed for risk management, it was conservatively assumed that the oral CSF also represents the carcinogenic potential of BaP by inhalation.

The RfC for naphthalene of 0.003 mg/m^3 based on a lowest-observed-adverse-effect level (LOAEL) of 9.3 mg/m^3 (human equivalent concentration), observed in mice that developed hyperplasia and metaplasia in respiratory and olfactory epithelium, respectively, following chronic inhalation of naphthalene in air, was used to evaluate the noncancer effects of PAHs. U.S. EPA derived the RfC through application of a 3,000-fold uncertainty factor, which accounted for extrapolation from mice to humans (factor of 10), and protection of sensitive human subpopulations (factor of 10), extrapolation from a LOAEL to a no-observed-adverse-effect level (NOAEL) (factor of 10). To account for database deficiencies, including the lack of a 2-generation reproductive toxicity study and chronic inhalation data for other animal species, an additional factor of 3 was added, to arrive at a chronic RfC for naphthalene of 0.003 mg/m^3.

3.3.2 Fluoride

Hydrogen fluoride is a colorless, corrosive, irritating gas or liquid that is made up of a hydrogen atom and a fluorine atom. Most exposure to fluoride is through consumption of water, beverages, food, and use of dental products. Small amounts of fluoride are added to toothpaste or drinking water to help prevent dental decay. Eating, drinking, or breathing large amounts of fluorides after long-term exposures can cause skeletal fluorosis, though it is extremely rare (ATSDR 2003). Food contains varying levels of fluoride. Animals that ingest fluoride-exposed plants accumulate fluoride; however, fluoride accumulation primarily occurs in the skeletal system and is unlikely to biomagnify up the food chain. There are no identified U.S. health criteria for hydrogen fluoride in residential air; the Norwegian guidelines used in the original EIA for protection of human health for fluorides in ambient air are 25 $\mu g/m^3$ annual average, and 10 $\mu g/m^3$ for the growing season average (Reydaral 2001; Sigfusson 2005, pers. comm.). There are no NAAQS for fluoride. The U.S. EPA Region 9 RBC (U.S. EPA 2005b) for residential soil is 3,666 mg/kg. This RBC is based on a reference dose for fluorine (as soluble flouride) derived from data in a population of children who developed objectionable dental mottling following an exposure to fluorine at water levels of 2–10 ppm in water (U.S. EPA 2006).

3.3.3 Sulfur Dioxide

SO_2 is a colorless, water-soluble, irritant gas. At sufficient concentrations, SO_2 is primarily an upper airway and eye irritant. In the airways, it produces bronchoconstriction and mucous secretion (ATSDR 1998). U.S. EPA measures short-term SO_2 exposure in annual, 24-hour, and 3-hour averaging periods. The primary NAAQS for SO_2 levels are 0.3 ppm (80 $\mu g/m^3$) for the annual averaging period and 0.14 ppm (365 $\mu g/m^3$) for the 24-hour averaging period, not to be exceeded more than once per year (U.S. EPA 2005c). The primary NAAQS (annual and 24-hour standards) are set to protect public health by providing a margin of safety to protect the health of sensitive populations such as asthmatics, children, and the elderly from respiratory effects including lung irritation and aggravation of bronchitis. The secondary NAAQS for SO_2 for the 3-hour averaging period is 0.5 ppm (1,300 $\mu g/m^3$). The secondary NAAQS (3-hour standard) is set to protect public welfare, including protection against visibility impairment, damage to animals, crops, vegetation, and buildings. The Icelandic limit for the protection of human health for SO_2 is derived from a European Communities directive (Sigfusson 2005, pers. comm.; EC 1999). Over a 1-hour averaging period, it requires that 350 $\mu g/m^3$ not be exceeded more than 24 times in a calendar year. Over a 24-hour averaging period, a maximum of 125 (high end) to 50 $\mu g/m^3$ (low end) is not to be exceeded more than three times in a calendar year. Over a calendar year, air concentrations are not to surpass 20 $\mu g/m^3$.

There are no definitive data in humans or animals that indicate a carcinogenic potential for SO_2 (ATSDR 1998; IARC 1997). Long-term exposure to persistent levels (0.4 to 3.0 ppm) of SO_2 for 20 years or more can cause changes in lung function.

3.3.4 Particulate Matter

Particulate matter, or PM, is a mixture of solid and liquid droplet particles in the air. PM_{10} includes coarse, inhalable particles ranging in diameter from 2.5 μm to 10 μm and can penetrate

the upper region of the body's respiratory defense mechanisms. Fine particles, or $PM_{2.5}$, include particles with diameters less than or equal to 2.5 μm. The Icelandic limits for the protection of human health for PM_{10} are derived from a European Union directive (Sigfusson 2005, pers. comm.; EC 1999). Over a 24-hour averaging period, exposures are not to exceed 50 $\mu g/m^3$ more than 7 (low end) or 35 times (high end) in a calendar year. Over a calendar year, air concentrations are not to surpass 20 $\mu g/m^3$ (low end) to 40 $\mu g/m^3$ (high end). U.S. EPA (2005c) has a current annual NAAQS for PM_{10} of 50 $\mu g/m^3$ and a 24-hour standard of 150 $\mu g/m^3$. The annual $PM_{2.5}$ standard is 15 $\mu g/m^3$ and the 24-hour standard is 65 $\mu g/m^3$.

3.4 Risk Characterization

In risk characterization, quantitative exposure estimates and toxicity factors are combined to calculate numerical estimates of potential health risk. In this section, potential cancer and noncancer health risks are estimated assuming long-term exposure to CoPCs as modeled by Earth Tech (2005a,b,c). As described in Section 3.2, *Exposure Assessment*, potential risks are estimated for the future worker and residential or farmer scenarios to provide a health protective means of considering possible future uses. The risk characterization methods described in RAGS (U.S. EPA 1989) are used to calculate potential RME excess lifetime cancer risks for carcinogens, and hazard indices for CoPCs with noncancer health effects. These methods and the results of the risk characterization are described below. Table 3-7 shows excess cancer risk estimates for the RME, while Table 3-8 presents RME hazard indices. In addition, tables in Appendix A present detailed results of the risk calculations for each exposure pathway, including EPCs and CDIs calculated for the RME, toxicity values used in risk estimates, and potential risk estimates for each CoPC in each exposure pathway.

3.4.1 Carcinogens

3.4.1.1 Methods

Quantifying total excess cancer risk requires calculating risks associated with exposure to individual carcinogens and aggregating risks associated with simultaneous exposure to multiple carcinogenic CoPCs. A cancer risk estimate for a single carcinogen is calculated by multiplying the carcinogenic CDI of the CoPC by its slope factor. A 1×10^{-6} cancer risk represents a one-in-one-million additional probability that an individual may develop cancer over a 70-year lifetime as a result of the exposure conditions evaluated. Because cancer risks are assumed to be additive, risks associated with simultaneous exposure to more than one carcinogen in a given medium are aggregated to determine a total cancer risk for each exposure pathway. Total cancer risks for each pathway are then summed for reasonable combinations of exposure pathways, to determine the total cancer risk for the population of concern.

U.S. EPA's *Guidelines for Cancer Risk Assessment* states, "…the linearized multistage procedure (typically used to calculate CSFs) leads to a plausible upper limit to the risk that is consistent with proposed mechanisms of carcinogenesis…The true value of the risk is unknown, and may be as low as zero" (51 Fed. Reg. 185:33992, 33998 [1986]). Because of uncertainties in methods available to assess risk, it is also possible that risks are underestimated.

Although the determination of an acceptable risk level is ultimately a decision to be made by risk managers, the findings presented here are compared with the range of acceptable risk levels cited in U.S. EPA's National Contingency Plan (NCP) (40 CFR Part 300), which U.S. EPA describes as the "blueprint for the Superfund law." The NCP states that risk levels in the range of 10^{-4} to 10^{-6} and lower are considered to be within the range of acceptable risks for Superfund sites. Consequently, where risks are lower than 10^{-6}, they are typically considered to be well within acceptable levels by regulatory agencies. Moreover, the peer-reviewed process for conducting risk assessment and the health protective toxicity criteria used through out this risk assessment provide a protective means to evaluate any potential hazards at the Fjardaal site.

3.4.1.2 Quantification of Carcinogenic Risks

Carcinogenic risk estimates were calculated for exposure to BaP in air for the following receptors: the onsite outdoor worker, the seagoing worker, and hypothetical residents at a location immediately adjacent to the site (adult and child scenarios). As indicated above, cPAH concentrations (as BaP equivalents) were derived using the conservative assumption that undetected cPAHs were present at half of the detection limit. Appendix A, Tables A-9 through A-16 RME provide carcinogenic risk estimates for the RME scenario. These findings are summarized more briefly in Table 3-7. As is indicated there, no cancer risk estimates were greater than 10^{-6}, which is the lower end of the acceptable risk range identified by U.S. EPA. The estimated risks were extremely low. The highest risk was 8×10^{-9}, or an additional excess cancer risk estimate of 8 in 1,000,000,000 exposed under the assumed conditions. Risk estimates were slightly higher for model estimates assuming a seawater scrubber was in place, but these differences were minimal and all risk estimates were well within the range considered to be acceptable by regulatory agencies.

3.4.2 Noncarcinogens

3.4.2.1 Methods

Unlike carcinogenic effects, other potentially adverse health effects are not expressed as a probability. Instead, these effects are expressed as the ratio of the estimated exposure over a specified period to the RfC derived for a similar exposure period (e.g., CDI:chronic RfD). This ratio is termed a hazard quotient. If the CDI exceeds the RfC (i.e., hazard quotient greater than 1), there may be concern for noncancer adverse health effects. Exposures resulting in a hazard quotient less than or equal to 1 are very unlikely to result in noncancer adverse health effects. Because U.S. EPA states that the range of possible values around RfCs is "perhaps an order of magnitude" (U.S. EPA 2005a), the significance of intakes exceeding the RfC by one-half an order of magnitude or less (i.e., hazard quotients ranging from 0.5 to 5) must be considered carefully.

3.4.2.2 Quantification of Noncarcinogenic Risks

Hazard indices calculated here incorporate the health protective assumption that 70 percent of the total PAH concentration estimate was present as naphthalene. Naphthalene was selected as

the basis for evaluating the noncarcinogenic effects of PAHs because it is the only PAH with an RfC. Appendix A, Tables A-1 to A-8 provide hazard indices for exposure to naphthalene in air for the following receptors: the onsite outdoor worker, the seagoing worker, and hypothetical residents at a location immediately adjacent to the site (adult and child scenarios). Table 3-8 shows a brief summary of hazard indices. As indicated there, no hazard indices were greater than the threshold of 1.0. In fact, the highest hazard index of 0.0028, was far below 1.0, indicating that hazards, if any, are well within acceptable levels related to this exposure.

3.5 Uncertainty Assessment

Because risk characterization serves as a bridge between risk assessment and risk management, it is important that major assumptions, scientific judgments, and estimates of uncertainties be described in the assessment. Risk assessment methods are designed to be conservative to address the uncertainties associated with each step in the risk assessment process.

Key factors in risk assessment methods that are likely to result in underestimates or overestimates of potential site risks include the following:

- Air dispersion modeling applied numerous upper-end assumptions that may tend to overestimate risks.
- Calculation of assumed PAH composition also incorporated health-protective assumptions (i.e., the calculation of cPAH estimates assuming undetected PAHs were present at one-half the detection limit or that 70 percent of the total PAH is naphthalene) that likely overestimate exposures.
- Scenarios regarding future site use are estimates and may reflect higher or lower exposures than actual use patterns.
  - It is assumed that the outdoor worker works outside every day all year, whereas actual outdoor exposure is probably reduced in the winter.
  - Precise work areas were not known and the area used to derive exposure estimates (i.e., UCL) may over- or underestimate risks somewhat. However, because the estimates were selected to be close in to the facility, an overestimate is more likely than an underestimate.
- Use of U.S. EPA's CSFs for carcinogens, which are based on the assumption that any exposure to a carcinogen is associated with some risk of cancer, is likely to overestimate risks.

The RfC for naphthalene applied here includes a 3,000-fold uncertainty factor. Thus the finding of a hazard index well below one indicates that there is a margin of safety in the hazard estimate of many orders of magnitude.

4 Ecological Risk Assessment

4.1 Problem Formulation

As discussed in the introduction, this risk assessment was undertaken to determine if there is an appreciable difference in the human health and/or ecological risks from emissions from the Fjardaal smelter with and without seawater scrubbers. This problem formulation section outlines the complete exposure pathways, selection of assessment endpoints, selection of measurement endpoints, and the ecological receptors. The information presented in this section is used to conduct the risk characterization (Section 4.5).

4.1.1 Complete Exposure Pathways

As discussed in Section 2.5, exposure pathways are likely to exist for plants in the project area and for wildlife that are resident in Reydarfjordur or that incorporate part of the area around the smelter site within their foraging area, either spatially or seasonally. These potential wildlife receptors include terrestrial mammals and birds that forage in the aerial deposition zone. Birds and mammals may be exposed to chemicals through the ingestion of food, and possibly through the incidental ingestion of soil. The consumption of plants could represent an important pathway for exposure to fluoride and PAHs. None of the chemicals evaluated here exhibit strong tendencies to bioaccumulate in food chains; therefore a screening assessment that focuses on primary consumers of plant material will provide risk estimates that are protective of other wildlife species.

4.1.2 Selection of Assessment Endpoints

Risk assessments should use site-specific assessment endpoints that address chemical-specific potential adverse effects to local populations and communities of plants and animals (U.S. EPA 1999). Consistent with this guidance, assessment endpoints were selected, taking into account their biological significance, their susceptibility to potential contact through direct or indirect exposure to CoPCs, and the availability of pertinent assessment models and toxicological information in the literature.

Assessment endpoints are selected to be protective of various ecological communities because they are expected to experience maximum integrated exposures in the terrestrial food webs. The goal of this assessment is to use site-specific assessment endpoints to address chemical-specific potential adverse effects to local populations and communities of plants and animals both within and outside the dilution zone. Two assessment endpoints were selected for this study:

- **Assessment endpoint 1:** Protection and maintenance of local populations and communities of plants.
- **Assessment endpoint 2:** Protection and maintenance of local populations and communities of birds and mammals.

These assessment endpoints were selected because they are ecologically relevant, they may be associated with potential stressors (aerial emissions), and they are relevant to management goals, including protection of endemic populations. Representative receptors were chosen to evaluate the assessment endpoints outlined above. Assessing risk for each representative receptor is expected to be protective of most other species that use the study area, given the habitat types and foraging material present. Selection of the representative receptor species exposure models, and all of the input parameters are described in Section 4.1.4.

For all of the selected receptors, the focus is on the stability and viability of exposed populations in the study area. Ecological risk questions for the ERA are based on the assessment endpoints, and they provide a basis for evaluating risk under the base case (without seawater scrubbers) and the alternative case (with seawater scrubbers). The risk questions for the study area are:

- **Risk Question 1—**Do SO_2 and fluoride levels that will be present in ambient air, with or without seawater scrubbers, have the potential to adversely affect sensitive plant communities?
- **Risk Question 2—**Do PAH and fluoride that will be present in ambient air, soil, and plants (with and without seawater scrubbers) have the potential to adversely affect birds or mammals via dietary ingestion?

4.1.3 Selection of Measurement Endpoints

Measurement endpoints provide the actual measurements used to evaluate each of the assessment endpoints and are the basis for evaluating risk. Several exposure scenarios are evaluated for the base case (without seawater scrubbers) and the alternative case (with seawater scrubbers) to portray reasonable worst case conditions, thus ensuring an environmentally protective analysis. For this risk assessment, the measurement endpoints are as follows:

- **Measurement Endpoint 1:** To estimate risk to local communities of plants, modeled air concentrations of SO_2 and hydrogen fluoride are compared to appropriate toxicity threshold criteria for sensitive plant communities.
- **Measurement Endpoint 2:** To evaluate risk to bird and mammal communities, estimated dietary doses of PAHs and fluoride are compared to toxicity reference values (TRVs) to designate benchmark concentrations in air that could potentially cause risk to the receptors.

4.1.4 Ecological Receptors

Risk to the plant communities in the project area is evaluated to be protective of the most sensitive species. In particular, potential effects are evaluated for mosses, lichens, and lodgepole pine (*Pinus contorta*). Mosses and lichens are known to represent the plant species that are most sensitive to SO_2 and fluoride in air. Lodgepole pine is specifically selected for evaluation because of its importance to the local community and because of the commercial silviculture operation at Teigagerdi.

Three vertebrate species were selected as wildlife receptors. The two mammalian receptors, including the wood mouse (*Apodemus sylvaticus*) and the Icelandic sheep (*Ovis aries*), and the one avian receptor, the rock ptarmigan (*Lagopus muta*), are described below. All three receptors were selected because they are present in Iceland, they are all herbivorous, and the food ingestion pathway was limited strictly to plant material. The selected receptor species serve primarily as recognizable surrogate models for other species that may be exposed. Vertebrate receptors are described below.

4.1.4.1 Rock Ptarmigan (*Lagopus muta*)

The rock ptarmigan (*Lagopus muta*) is the only member of the grouse family that occurs in Iceland (Magnusson et al. 2004). Rock ptarmigan are small grouse with varying winter and summer plumages (Vuijk 2005). The ptarmigan exist in truly arctic environments, including well-drained, hummocky arctic and alpine tundra with rocky ridges or outcrops and mixed vegetation, including lichen, heather, sedges, and *Salix* or *Betula* communities. These communities are mostly sparse in the most arid regions and denser and shrubbier at lower elevations (Holder and Montgomerie 1993).

Most ptarmigan populations spend winters on their breeding habitat, while others winter in shrubby areas at or above the snowline. Ptarmigan are residential ground dwelling birds that can be found in all vegetated lands (scrubs etc.). More than 99 percent of their diet is plant material, in all seasons. Ptarmigans feed on berries, buds, germinating seeds, and to a much lesser degree, insects when available (Holder and Montgomerie 1993). Crowberries, and to a lesser degree, blueberries, are plentiful within the study area (Jonsdottir 1999) and could provide a food source for ptarmigans, among other plant matter found throughout the heath and grasslands. Rock ptarmigan are likely to be an important food source for predators like the gyrfalcon and the arctic fox.

4.1.4.2 Wood Mouse (*Apodemus sylvaticus*)

Widespread and common throughout continental Europe and Britain, the wood mouse (*Apodemus sylvaticus*) is a common rodent in Iceland, introduced approximately 1,100 years ago (Hersteinsson 2005). The wood mouse is similar in appearance to the common house mouse (*Mus musculus*) but differs by its larger size and lighter fur color (Schlitter and Van der Straeten 2004). Wood mice are characterized by having an elongated body (7–15 cm), a tail as long as the body, large ears, and large eyes. The hind legs are more developed than the forelegs, so that it moves through habitats by jumping. Females mate year round, producing three to four litters per year, each with 4–5 young.

The wood mouse is nocturnal and frequents various terrestrial biomes, including arable land, pastureland, and rural gardens (Schlitter and Van der Straeten 2004), but does not prefer overly wet habitats (Wildscreen 2004). The wood mouse consumes mostly seeds, and to a lesser extent buds, fungi, and fruits and tubers (Schlitter and Van der Straeten 2004). The wood mouse would likely occur in all habitats within the study area including moss heath, wild and cultivated grassland, and dwarf shrub heath cover types.

4.1.4.3 Icelandic Sheep (*Ovis aries*)

Although the study area is not currently grazed by sheep (Jonsdottir 1999), the Icelandic domestic sheep was evaluated to assess risk should the area be used for grazing again in the future. Sheep are also considered a surrogate receptor for reindeer that occasionally forage in the area, especially in the winter (Jonsdottir 1999). Icelandic sheep graze on brush and grasses (OSU 1996) and could use the grassland cover types found within the study area.

4.2 Characterization of Exposure

4.2.1 Plant Exposure

Plants are exposed to the phytotoxic constituents SO_2 and fluoride by direct contact with the constituents in air. Plants are also exposed to PAHs and fluoride, which they can incorporate into their tissues. The accumulation of PAHs on or in plant material occurs predominantly through wet and dry deposition. Movement from soils to plants may result in PAH accumulation within the root cortex, but PAHs are not translocated into the above ground vegetation (Slaski et al. 2000). Under certain special conditions, plants have been shown to accumulate gaseous PAHs through both stomatal uptake and partitioning into cuticular waxes. However, under direct industrial output conditions as considered in this assessment, gaseous PAH concentrations are so low as to represent an insignificant source, compared to wet and dry particulate deposition rates. This is because gaseous PAHs readily partition to atmospheric particles as a result of their inherently low vapor pressures, and they partition preferentially to particles as opposed to plant surfaces, because airborne particulate matter has a much higher surface area to volume ratio than terrestrial plants (Bakker et al. 2000). Fluoride can accumulate in plants from root uptake from soil, wet and dry aerial deposition, and gaseous stomatal uptake (Weinstein and Davison 2004).

4.2.2 Wildlife Exposure

To evaluate exposure and risk to terrestrial receptors that feed on plants in the Reydarfjordur study area, a food-web exposure model was developed to determine benchmark concentrations for birds and mammals that might be exposed to PAHs and fluoride while feeding in the vicinity. As described above, two mammalian receptors and one avian receptor were evaluated using food-web models. After the food-web models were developed, the models were used to determine benchmark concentrations that would be protective of the selected receptors that use

the area. The food-web models were developed using scientifically defensible, conservative input parameters selected from the scientific literature.

4.2.2.1 Food-web Modeling

The food-web modeling approach that was used is a standard approach consistent with U.S. EPA's wildlife exposure guidance (U.S. EPA 1993; 61 Fed. Reg. 47552). The food-web model estimates dietary exposure as a body-weight-normalized total daily dose for each receptor species. The structure of the food-web exposure model is described by the following equation:

$$IR_{chemical} = \frac{\sum_i \left(C_i \times M_i \times F_i\right)}{W}$$

where:

$IR_{chemical}$ = total ingestion rate of chemical from all dietary components (mg/kg body weight-day)

C_i = concentration of the chemical in a given dietary component or environmental medium (mg/kg)

M_i = rate of ingestion of dietary component or medium (kg/day)

F_i = fraction of the daily intake of a given dietary component or medium derived from the study area (unitless area-use factor)

W = body weight of receptor species (kg).

The term $IR_{chemical}$ can be expanded to specify each ingestion medium, which includes vegetation as the primary food item:

$$IR_{chemical} = [\Sigma\ (C_{veg} \times M_{veg} \times F_{veg}) / W$$

This model provides an estimated total dietary exposure for PAHs and fluoride resulting entirely from the consumption of food (plants) on a mg/kg body weight-day basis. For both the mammalian and avian species, the exposure calculation assumes that the entire diet for all receptors comes from the Reydarfjordur study area ($F_i = 1$) and that 100 percent of the chemical in ingested food is absorbed.

The diet of the wood mouse, sheep, and ptarmigan was conservatively assumed to be 100 percent plant matter obtained from the Reydarfjordur study area. In the case of this assessment, the plant matter was assumed to be either heather/heath or grasses. Although the wood mouse and ptarmigan are known to consume berries, seeds, and buds, it was necessary to assume that they consumed the vegetative structures of the heather/heath and grasses because the information needed to model concentrations from the fruiting parts of the plants, such as berries and seeds, to the air concentrations, was not available.

Assuming that the receptors consume the vegetative parts of the plant is likely to be more conservative than assuming they are consuming the fruiting parts. For example, PAH

concentrations in plants are almost entirely the result of deposition from the air (Slaski et al. 2000). Therefore, for PAHs, the same concentration present in berries or seeds would also be present in heathers and grasses, with the exception that berries and seeds are likely to be exposed to air for a much shorter period of time than vegetative matter, given their ephemeral presence on the plants. Fluoride, on the other hand, is incorporated into plants through gaseous uptake (Less et al. 1975). Therefore, the vegetative portions of the plants where photosynthesis occurs, and where gaseous exchange occurs, would be expected to have higher concentrations of fluoride than the reproductive portions of the plant, which are not photosynthetic.

These conservative assumptions regarding ecological receptors' exposure likely overestimate the potential for exposure, but the use of such conservative assumptions is appropriate in a predictive screening assessment such as this to reduce the possibility of drawing false negative conclusions regarding ecological risk. Exposure to PAHs and fluoride in water and soil via incidental ingestion was assumed to be negligible as compared to consumption of food. Incidental soil ingestion for the wood mouse and rock ptarmigan is likely to represent a small portion of their dietary exposure—approximately 2 percent and 9 percent of the diet, respectively (Beyer and Fries 2003; Table 4-1). Water ingestion rates for both the wood mouse and rock ptarmigan are likely to be minimal as well. Typically for mice, non-seed plant materials provide a significant proportion of the mouse's daily water requirements (MacMillen and Garland 1989). Water ingestion rates for the ptarmigan are generally not reported in foraging studies (Williams et al. 1980, Irving et al. 1987, Wren et al. 1994). West and Meng (1966) suggest that ptarmigan do not consume frost-laden branches until later in the day when the sun or wind dissipates frost.

The exposure parameters for mammalian and avian receptors are provided in Table 4-1. The mean adult female body weight was used in the exposure calculations. Because male individuals generally have greater body weights than females of the same species, using the body weight for females is a conservative (precautionary) assumption. Food ingestion rates were calculated using allometric equations by Nagy (2001) (Table 4-1). It was assumed that animals are residents of the Reydarfjordur study area for the entire year. This assumption is also conservative, especially for animals such as reindeer that are likely to forage over large areas and spend only a relatively short period in the Fjardaal assessment area. This assumption is also likely to be conservative for ptarmigan because these birds generally move seasonally in response to the snow line.

4.2.2.2 Derivation of Toxicity Reference Values

The food-web models described above were not used to estimate exposure for the receptors but instead were developed to determine benchmark concentrations that would be protective of receptors that utilize the area. To calculate the benchmark values, it was first necessary to determine what contaminant dose is needed to pose risk to the receptors.

A TRV is a contaminant dose or body burden that is compared to site-specific doses or body burdens to assess the potential risk to an ecological receptor. A TRV can be based on results from laboratory or field studies. Exposure estimates from the food-web models are compared to the benchmark TRVs for each receptor to develop quantitative estimates of risk.

TRVs are derived from the primary toxicology literature, such as that summarized in the toxicity profiles. TRVs are expressed as a daily dietary dose, and are calculated from dietary exposure endpoints according to the following general formula:

$$TRV = ([diet] \times IR)/BW$$

where:

TRV	=	toxicity reference value for NOAEL or LOAEL endpoints (mg/kg body weight per day)
[diet]	=	dietary concentration (mg/kg food or mg/L drinking water) associated with a given endpoint
IR	=	daily ingestion rate (kg food/day or L drinking water/day)
BW	=	body weight (kg).

Avian and mammalian TRVs that were selected to determine benchmark concentrations using the food-web models are described below and outlined in Table 4-1. For a detailed review of the ecological effects of PAHs and hydrogen fluoride in birds and mammals, please see Section 4.4, *Characterization of Ecological Effects*.

Avian PAH TRV—Studies on the toxicity of PAHs in birds, particularly with regard to impacts on reproduction, are rare. No appropriate feeding studies exist by which to develop a TRV. The only applicable study found was that reported by Hough et al. (1993) that examined the effects of BaP on pigeons. Three- to six-month old pigeons were administered a dose of 10 mg/kg BaP (intramuscular) weekly for a period of 5 months. The treatment birds were reported to have suffered complete reproductive failure and an associated gross alteration in ovarian structure. This dose, which corresponds to a daily exposure of 1.43 mg/kg-day, was considered representative of a LOAEL for birds. To estimate the no-effects TRV, a 10-fold level of uncertainty was applied to the LOAEL TRV to derive an estimate of 0.143 mg/kg-day.

Mammalian PAH TRV—The evaluation of PAH toxicity to mammals focused on a study by Mackenzie and Angevine (1981) that examined the reproductive effects of BaP on mice. Female CD-1 mice were exposed to BaP ranging from 10 to 160 mg/kg-day through daily intubation. Treatment commenced on day 7 after the best estimated time of conception and continued through day 16 of gestation. Mean pup weight was observed to be significantly reduced in the 10 mg/kg-day treatment group. This treatment was therefore considered to be applicable as a LOAEL TRV. The estimation of the no-effects TRV was based on the application of a 10-fold level of uncertainty to the toxicity estimate to derive a value of 1 mg/kg-day.

Avian Fluoride TRV—The avian fluoride TRV was selected from a study completed on domestic turkeys by Nahorniak et al. (1983). Although studies on starlings and other wild birds were available, the study by Nahorniak et al. (1983) was selected for TRV derivation primarily because of the similarity of ptarmigan and turkey dietary preferences, and the relatively long duration of study. A wild turkey diet consists of seeds, nuts, fruits, plant leaves, and occasionally insects, similar to the rock ptarmigan.

Newly hatched turkeys were dosed with various concentrations of sodium fluoride added to their diet, a mix of soybean and corn, for up to 18 weeks. At four and eight weeks of age, the young turkeys in the 400 ppm dose group exhibited significantly reduced body weights. At 8 weeks of age, reduced growth, leg deformities, and mortality occurred in the group dosed at 800 ppm. Using reduced body weights as a conservative endpoint (as compared to mortality), a LOAEL was calculated using the 400 ppm fluoride diet. The NOAEL was calculated using the 200 ppm fluoride dosage, because mortality and body weight were not significantly different from the other dose groups after 18 weeks.

To calculate the LOAEL, the minimum average 18-week body weight (105 grams) for male turkeys was selected from the Nahorniak et al. (1983) non-dosed study group, and the food ingestion rate (246 g DW/day) was calculated using an allometric equation based on body weight for Galliformes birds (Nagy 2001). Use of the male body weight in this case is expected to be conservative because the average weight of an 18-week old male is less than the average weight of an adult female. Additionally, the 400 ppm fluoride diet was assumed to be wet weight and to consist of approximately 20 percent moisture, resulting in a dry weight dose equal to 500 mg/kg fluoride. The resultant LOAEL is 26.5 mg/kg BW/day.

To calculate the NOAEL, the assumptions stated above remained constant, except the 200 ppm dose was converted to dry weight (assuming 20 percent moisture), yielding a dry weight dose equal to 250 mg/kg fluoride. The resultant NOAEL is 13.2 mg/kg BW/day.

Mammalian Fluoride TRV—A study of mice conducted by NTP (1990) was chosen to derive the mammalian fluoride TRV. In this study, mice received various concentrations of sodium fluoride in water for a period of six months. The 300 ppm dose group experienced decreases in weight gain and death prior to the end of the study, whereas the 200 ppm dose group did not. Based on the endpoints of reduced weight gain and death, the 300 ppm dose was selected as the LOAEL group and the 200 ppm group serves as the NOAEL. Assuming the mouse body weight of 30.7 g, and a water ingestion rate of 0.004 L/day, the NOAEL TRV for mammals equals 24.5 mg/kg/d, and the LOAEL TRV equals 42.1 mg/kg-day.

The study by NTP (1990) was appropriate because it was of longer duration than other studies and it was conducted on mice, therefore most closely matching the herbivorous diet of the wood mouse and Icelandic sheep.

Other studies were reviewed but were short-term, or did not offer relevant information regarding food concentrations. For example, Aulerich et al. (1987) presented data on reproductive effects in mink exposed to dietary concentrations of 33 to 350 ppm. However, this study is not appropriate because mink are piscivorous/carnivorous and are a poor surrogate for the receptors in this risk assessment.

4.2.2.3 Calculating Benchmark Concentrations

To calculate the benchmark concentration, the food-web models were compared to the threshold, or benchmark, values (TRVs) for each receptor to develop quantitative risk estimates or hazard quotients.

Hazard quotients are the ratio of the measured or predicted exposure concentrations to the TRV:

$$HQ = \frac{IR_{chemical}}{TRV}$$

where:

HQ	=	hazard quotient (unitless)
$IR_{chemical}$	=	ingestion rate of the chemical (mg/kg body weight-day)
TRV	=	toxicity reference value.

Hazard quotients less than 1.0 indicate that the chemical is unlikely to cause adverse ecological effects. Hazard quotients above 1.0 indicate some potential for adverse ecological effects; however, hazard quotients above 1.0 do not necessarily signify unacceptable risk. Other pieces of information, such as sources of uncertainty and site-specific exposure information, are weighted in the risk evaluation and interpretation of hazard quotients to determine the significance of hazard quotients greater than 1.0.

Food-web models were developed for the selected receptors to predict exposure from food. "Benchmark" plant concentrations were then calculated for PAHs and fluoride that are equivalent to concentrations in plants consumed by the representative receptors that would result in exposures equal to the NOAELs and would thus correspond to negligible ecological risk.

The benchmark plant concentrations were then converted to benchmark air concentrations, using the methods outlined in Section 4.3, below. This was done in a probabilistic manner, based on the statistical distribution of the air modeling output and assumed distributions for other variables to account for variability and uncertainty in the concentration and deposition estimates.

4.3 Prediction of Fluoride and PAH Concentrations in Plants

Concentrations of fluoride and PAHs in plants were modeled based on predicted atmospheric concentrations provided by Earth Tech (2005a,b,c). Two types of models were used in this analysis: 1) a one-compartment additive deposition model was used to estimate the accumulation of PAHs and fluoride in soils and to estimate the accumulation of PAHs on plants, and 2) a one-compartment kinetic accumulation model was used to estimate accumulation of fluoride in plants. The two plant models were developed for potential forage sources of ecological receptors: 1) generalized grasses and 2) heather and heather-like shrubs. This was because in the four major biotopes identified in and around the Reydarfjordur fjord, these two plant types represent the predominant sources of available forage material and are likely to best represent dietary exposure to herbivorous wildlife receptors. The two plant species were modeled separately because of several differences in factors affecting accumulation. For example, grasses and heathers exhibit widely different senescence rates which will affect long-term availability of PAHs and fluoride to receptors.

4.3.1 PAH Deposition on Plants

The accumulation of PAHs on or in plant material occurs predominantly through wet and dry deposition. Movement from soils to plants may result in PAH accumulation within the root cortex, but is not translocated into the above ground vegetation (Slaski et al. 2000). Under certain special conditions, plants have been shown to accumulate gaseous PAHs through both stomatal uptake and partitioning into cuticular waxes. However, under direct industrial output conditions as considered in this assessment, gaseous PAH concentrations are so low as to represent an insignificant source, compared to wet and dry particulate deposition rates. This is because gaseous PAHs readily partition to atmospheric particles as a result of their inherently low vapor pressures, and they partition preferentially to particles as opposed to plant surfaces, because airborne particulate matter has a much higher surface area to volume ratio than terrestrial plants (Bakker et al. 2000).

Plant accumulation of PAHs through aerial deposition was modeled based on the following algorithm:

$$[\text{PAH}]\left(\text{mg}/\text{kg DW}\right) = \frac{k_{dep} \times L \times AF}{BM_a}$$

where:

[PAH]	=	concentration of PAHs in vegetative plant material (mg/kg DW)
k_{dep}	=	rate of combined wet and dry PAH deposition ($\mu g/m^2$-s)
L	=	senescence rate of vegetative material (s)
AF	=	adhesion factor (unitless)
BM_a	=	area plant biomass (kg/m^2 DW).

All source terms are independent so no correlation correction was required. Because of uncertainty associated with the prediction of the adhesion factor, a constant equal to 1.0 was assumed for both grasses and heather. It is estimated that this assumption will result in over-prediction of actual PAH concentrations in plant material. Senescent material was assumed to exit the plant compartment and enter the soil compartment. Input parameter distributions are summarized in Table B-1.

4.3.2 Fluoride Accumulation in Soil

Unlike PAHs, fluoride can accumulate in plants from root uptake from soil, wet and dry aerial deposition, and gaseous stomatal uptake (Weinstein and Davison 2004). Because of the complexity associated with the multiple and interdependent uptake routes, overall rate models were used to predict fluoride accumulation in grasses and heather. These were based on observational studies from which multiple regression relations between aerial fluoride concentrations could be correlated with observed fluoride concentrations in the specific types of plants. Details on the development of the specific regression models are discussed below.

4.3.2.1 Fluoride Uptake in Heather

Uptake of fluoride in heather was modeled based on the results of Horntvedt (1997). In this study, air concentrations of fluoride from five aluminum smelters were monitored along with fluoride concentrations in plants in and around the monitoring stations. Horntvedt developed a linear model for an accumulation rate of fluoride in *S. aucuparia* (rowan or mountain ash) leaves. Comparisons of fluoride concentrations in other species provided relative conversion factors. To develop a relative concentration for heather-like shrubs, a union of the conversion factors for *Calluna vulgaris* and *Vaccinium myrtillus L.* were applied as follows:

$$[F]_{Heather}\left(\mathrm{mg}/\mathrm{kg\,DW}\right) = \left(b + m \times [F]_{air}\right) \times L \times f\left(\text{Rowan} \rightarrow \text{Heather}\right)$$

where:

$[F]_{Rowan}$ = fluoride concentration in *S. aucuparia* (mg/kg DW)

$[F]_{air}$ = fluoride concentration in air ($\mu g/m^3/day$)

L = senescence rate of vegetative material (s)

f(Rowan→Heather) = conversion factor from rowan to heather (unitless).

Parameter distributions are provided in Table B-2 (Appendix B).

4.3.2.2 Fluoride Uptake in Grasses

Uptake of fluoride in grasses was modeled based on the results of Less et al. (1975). In this greenhouse study, the uptake of atmospheric fluoride by *Loloium perenne L.* (perennial ryegrass) was monitored over various periods throughout the year. The authors developed an uptake model (S) based on the difference between observed fluoride concentrations and natural fluoride concentrations (5 mg/kg DW) over the atmospheric fluoride concentration. This was converted into a daily uptake rate as follows:

$$k_{air} = \frac{S}{\Delta t} = \frac{[F]_{plant} - 5}{[F]_{air} \times \Delta t}$$

where:

k_{air} = uptake rate of fluoride by grasses $\left(\frac{mg/kg}{\mu g/m^3/day}\right)$

S = observed accumulation coefficient $\left(\frac{mg/kg}{\mu g/m^3}\right)$

Δt = time of study observation (days)

$[F]_{plant}$ = fluoride concentration in grass (mg/kg DW)

$[F]_{air}$ = fluoride concentration in air ($\mu g/m^3$)

5 $mg \cdot kg^{-1}$ = the assumed background concentration by Less et al. (1975).

The study reported significant uptake rates both in the presence and absence of precipitation. To correct for this, the value of k_{air} was selected based on the probability of precipitation as reported by WMO (2005) as follows:

$$k_{air} = \begin{Bmatrix} P(rain) & k_{air-precip} \\ 1-P(rain) & k_{air-no\ precip} \end{Bmatrix}$$

where:

k_{air} = overall uptake rate of fluoride by grasses $\left(\frac{mg/kg}{\mu g/m^3/day}\right)$

$k_{air\text{-}precip}$ = uptake rate of fluoride by grasses with precipitation $\left(\frac{mg/kg}{\mu g/m^3/day}\right)$

$k_{air\text{- }no\ precip}$ = uptake rate of fluoride by grasses with no precipitation $\left(\frac{mg/kg}{\mu g/m^3/day}\right)$

P(rain) = probability of rain occurring on any given day (unitless; WMO 2005).

Less et al. (1975) also reported that fluoride in grasses equilibrates with fluoride in the atmosphere within the first 10 days of exposure, with little uptake over prolonged exposure. For this assessment, the change in uptake rate (M) over time was modeled as an exponential regression based on the observations at 10 and 20 days as follows:

$$[F]_{plant}\left(mg/_{kg\ DW}\right) = S_{Corr} \times [HF]_{Air} + 5$$

where:

$$S_{Corr}\left(\frac{mg/kg}{\mu g/m^3}\right) = G \times e^{M}$$

and:

$$M = \ln(k_{air})_{10\ days} + \frac{\ln(k_{air})_{20\ days} - \ln(k_{air})_{10\ days}}{10} \times G$$

where:

$[F]_{plant}$ = fluoride concentration in grass (mg/μg DW)

S_{Corr} = time-corrected accumulation coefficient $\left(\frac{mg/kg}{\mu g/m^3}\right)$

G = duration of vegetative growth (days)

k_{air} = precipitation-adjusted uptake rate $\left(\frac{mg/kg}{\mu g/m^3/day}\right)$.

Because of the limited resolution in the data, the time-corrected accumulation coefficient (S_{Corr}) for days 1 through 10 was assumed equal to S as reported for day 10. Parameters used in the execution of the model are provided in Table B-3 (Appendix B).

4.4 Characterization of Ecological Effects

Summaries of PAH, hydrogen fluoride, and SO_2 toxicities to plant, avian, and mammalian receptors are presented below.

4.4.1 Adverse Effects of Sulfur Dioxide

4.4.1.1 Sulfur Dioxide Toxicity in Plants

Sulfur is a plant nutrient, mostly taken up from the soil as sulfate, but also absorbed from the air as SO_2 via stomata. The majority of SO_2 toxicity literature is based on studies that have evaluated lichens as bioindicators of air pollution, as lichens are known to be very sensitive to phytotoxic effects of SO_2. Lichens (especially when moist) can become a large sink for SO_2 because of the compound's high solubility in water. A study by Nash and Gries (2002) found that approximately 70 percent of the absorbed SO_2 can be oxidized to sulfate and leached from lichens, which acts as a detoxifying mechanism. However, the retained SO_2 can be converted to bisulfite, and can be toxic when accumulated at high levels because of acidification and necrosis of plant tissue. Toxicity effects on lichens usually manifest as decreases in photosynthesis and respiration, leaching of electrolytes, spore generation, and increased mortality.

Studies on decreased photosynthesis effects include tests on lichen exposed to 170 $\mu g/m^3$ to 2,500 $\mu g/m^3$ SO_2 in air (Richardson and Nieboer 1983). Decreases in spore germination and spore germination inhibition were found in lichen that were exposed to aqueous SO_2 at concentrations of 0.032 mg/L (Belandria et al. 1989). In laboratory experiments, Grace (1980, as cited in Richardson and Nieboer 1983) found that lichen exposed to 14,600 $\mu g/m^3$ SO_2 in air resulted in potassium leaching. Potassium efflux is interpreted as an increase in cell permeability. McCune (1988) observed that lichen community parameters (species composition, species richness for example) were correlated with 3-year mean annual SO_2 levels ranging from 23 to 40 $\mu g/m^3$ in Indiana.

Liblik and Pensa (2001) summarized critical levels for SO_2 to range from 10 to 30 $\mu g/m^3$ for general vegetation, but for sensitive lichen and *Sphagnum* mosses, a critical limit of 3–9 $\mu g/m^3$ was mentioned. Kashulina et al. (2003) summarized that the critical levels of SO_2 emissions (in annual mean averages) are 15 $\mu g/m^3$ in air for trees growing in cold climates, and 10 $\mu g/m^3$ in air for the most sensitive plants, including moss and lichens. The authors, however, recommended that, based on moss damage in the Kola Peninsula of Arctic Russia, the critical level of SO_2 in air for mosses and lichens should be set lower than previously proposed, to 5 $\mu g/m^3$.

SO_2 toxicity to grasses was demonstrated in a study by Vermehren et al. (1994). Seeds from grasses originating in less polluted areas exhibited an increase in peroxidase activity, but grasses from more polluted areas had a higher tolerance to SO_2. Similarly, Ayazloo and Bell (1981, as cited in Palmason and Magnusson 1998) reported greater tolerance in grasses exposed up to 500 $\mu g/m^3$. Palmason and Magnusson (1998) found that *Lolium perenne* had no adverse effects when exposed to 37 ng/L SO_2 and *Agrostis capillaries* and *Nardus stricta* were stimulated by SO_2. Variations of sensitivity to SO_2 occur in different grass species. For example, *Festuca rubra* has a decreasing tolerance to SO_2 with distance, but *Dactylis glomerata* had no apparent pattern of tolerance (Wilson and Bell 1986).

SO_2 toxicity has also been studied in trees. Toxic effects include foliage necrosis, photosynthesis inhibition, electrolyte leaching, and decreases in respiration. Studies with Scots pine exposed to 70 $\mu g/m^3$ of SO_2 in air resulted in photosynthesis inhibition (Freer-Smith and Taylor 1992). Scots pine exposed to 92 $\mu g/m^3$ SO_2 in air exhibited leakage of electrolytes from the shoots, and concentrations of <10 $\mu g/m^3$ in ambient air resulted in necrosis in the foliage. Shaw et al. (1993) summarized that Scots pine (*Pinus sylvestris*) is one of the most SO_2 sensitive pines. The period shortly after budburst is the most sensitive period for pines (Shaw et al. 1993). During the critical period of needle expansion, the threshold SO_2 concentration for injury was <10 $\mu g/m^3$ (mean concentration during needle expansion; Shaw et al. 1993). Ozolincius et al. (2005) examined trees (*Pinus sylvestris*, *Picea abies*, *Betula* spp., *Fraxinus excelsior*, *Alnus* spp., *Populus tremula*, and *Quercus robur*) in Lithuania, where SO_2 concentrations ranged from 2 to 50 $\mu g/m^3$. Liblik and Pensa (2001) studied coniferous trees in northeastern part of Estonia, and the first signs of damages occurred at less than 15 $\mu g/m^3$ on an annual average basis, with significant changes at 10–30 $\mu g/m^3$ (annual average basis). Complete degradation of *sphagnum* moss occurred at 3–9 $\mu g/m^3$ (annual average basis).

Palmason and Magnusson (1998) report that the plants most sensitive to SO_2 are lichen, mosses, and conifers. Reindeer moss (*Cladina rangifera*) has a predicted threshold concentration of 20–30 $\mu g/m^3$ over 24 hours (Grace et al. 1985, as cited in Palmason and Magnusson 1998). The lowest threshold of SO_2 sensitivity is 5–10$\mu g/m^3$ in lichen (Salemaa 2004). The threshold for conifers is greater, at 280 $\mu g/m^3$ for 8 hours (Palmason and Magnusson 1998). Areas that are exposed to chronic SO_2 will be affected by changes in diversity and richness of species, which will be visible with changes in the vegetation community along gradients of SO_2 concentrations (U.S. EPA 1982).

There is a potential that subtoxic concentrations of sulfur dioxide may differentially impart competitive advantages to some plant species, possibly leading to a shift in the composition of the communities in the vicinity of the smelter and thereby altering succession. Plants are

capable of using airborne sulfur dioxide as a metabolic sulfur source (Veenranjaneyulu et al. 1991). Chloroplasts possess the capability to reduce sulfur dioxide to bisulfite and sulfide that can be used directly in sulfur amino acid anabolism (Ghisi et al. 1989). This capacity varies among plant species. Under conditions of sulfur limitations, such as in Iceland (UNCSD 1997), an increased capacity to utilize airborne sulfur could impart a competitive advantage. However, field studies on the impact of sulfur dioxide found no increase in plant growth or yield at concentrations lower than approximately 20 $\mu g/m^3$ (WHO 2000). The maximum sulfur dioxide concentrations projected for the smelter were less than 3 $\mu g/m^3$, either with or without seawater scrubbers. Therefore, while it is possible to differentially stimulate plant growth by increasing atmospheric sulfur dioxide concentrations, the magnitude of increases in this case is not likely to affect the composition of the plant communities.

4.4.1.2 Sulfur Dioxide Toxicity in Birds and Mammals

Little information about SO_2 toxicity is available for avian and mammalian receptors. Available studies focus mainly on ambient air pollution or emissions from coal-fired power plants. Toxic effects of airborne SO_2 include changes in pulmonary function, irritation in respiratory airways, lipid peroxidation in some tissues and decreased particle clearance from the lungs (Oehme et al. 1996). SO_2 emissions can be converted to sulfate, sulfuric acid, ammonium sulfate, and ammonium bisulfate by atmospheric processes, which in turn can be inhaled and exhibit the properties of particulates (Oehme et al. 1996). Studies described by Oehme et al. (1996) have documented an increase in calf deaths and general decrease in animal health with increased air pollution. Donkeys exposed to more than 786,000 $\mu g/m^3$ SO_2 for 30 minutes had a decreased ability to clear particles from their lungs (Spiegelman et al. 1968, as cited in Oehme et al. 1996). SO_2 from smelter operations have resulted in increases in hematocrit and hemoglobin concentration in wood mice (Gorriz et al. 1996). Changes in plasma parameters may be an early indicator of diseases and infection and are often coincidental with a reduction in food and water consumption, dehydration, hypoxia, and other toxicity effects (Gorriz et al. 1996).

Researchers found that SO_2 is removed from the upper respiratory tract. Studies with guinea pigs found that scrubbing of SO_2 is more efficient at higher concentrations. For example, at 2,620 $\mu g/m^3$, only 5 percent of the SO_2 was removed, in comparison to 90 percent removal when the exposure concentration was 1,834,000 $\mu g/m^3$ (Amdur et al. 1991, as cited in Oehme et al. 1996). The chronic no effect concentration of SO_2 to dogs exposed for 16 hours/day for 18 months is 1,310 $\mu g/m^3$ **(**Spiegelman et al. 1968, as cited on Oehme et al. 1996).

Existing information on SO_2 toxicity to mammals and birds indicates that toxicity thresholds are several orders of magnitude greater than the highest expected SO_2 concentration from Fjardaal. Therefore, risk to mammals and birds from SO_2 exposure is negligible and is not discussed further in this report.

4.4.2 Adverse Effects of PAHs

Adverse effects of PAHs to biota have been reported in many types of organisms, including mammals, birds, invertebrates, plants, amphibians, and fish. However, the effects of PAHs are

varied. Generalizations cannot be readily made, but may include adverse effects on reproduction, development, and immunity (ATSDR 1993).

4.4.2.1 PAH Toxicity in Plants

Kipopoulou et al. (1999) studied PAH exposure in vegetables in Greece and determined that air deposition, as opposed to soil exposure, is the principal pathway for the accumulation of PAHs in vegetable plants. This is consistent with findings reported by Eisler (1987), who documented that the concentrations of PAHs in plants are substantially lower than those in soil, and concentrations in the two media are poorly correlated because deposition and absorption of atmospheric PAHs is the primary exposure pathway (Eisler 1987).

Phytotoxicity data related to PAHs in soils are sparse. Wittig et al. (2003) exposed poplar cuttings to PAH mixtures in sand and nutrient solutions ranging from 0.1 to 200 mg/kg substrate, and determined that PAH exposure results in a drop in the amount of biomass produced and in the transpiration rate, accompanied by a reduction in the uptake of nutrient solution. However, these studies were not performed with soil mixtures of PAHs, so the physico-chemical properties of the test substrate may not be representative of soils in which trees grow in the wild. Leyval and Binet (1998) performed tests using soils containing a mixture of ten PAHs. They found that at 100 mg/kg DW in soil, growth of ryegrass was stimulated, especially in plants that had mycorrhizae associated with the roots. The results of Leyval and Binet (1998) suggest that terrestrial plants are generally tolerant to PAH contamination in soils, and the results of the review by Kapustka (2004) are similar to earlier reports summarized by Eisler (1987).

4.4.2.2 PAH Toxicity in Birds

Studies on the toxicity of PAHs to birds, particularly with regard to impacts on reproduction, are rare. Hough et al. (1993) dosed pigeons with BaP intramuscularly, at concentrations of 10 mg/kg weekly for 5 months. The researchers found that the treatment birds suffered from complete reproductive failure and associated gross alteration in ovarian structure. Studies that relate PAHs in soil or air to avian effects are non-existent at this time.

4.4.2.3 PAH Toxicity in Mammals

Mammals can absorb PAHs by inhalation, dermal contact, and to a lesser extent, by ingestion (Eisler 1987). PAHs are readily metabolized by vertebrates and they generally do not accumulate in animal tissues to any appreciable extent.

Naphthalene toxicity studies have been conducted on mice via oral gavage. Reproductive effects were not noted at dosages of 133 mg/kg body weight, but effects on ingestion, gastric lesions, and body weights were noted. Kapustka (2004) also summarized studies on mice by Rigdon and Neal (1965) and Neal and Rigdon (1967). The researchers fed mice 250–100 mg/kg BaP throughout the reproductive period, and for 6 days prior, and noticed no weight gain or reproductive effects. However, Mackenzie and Angevine (1981) examined reproductive effects of BaP ranging from 10 to 160 mg/kg-day through daily intubation.

Treatment commenced on day 7 after the best estimated time of conception and continued through day 16 of gestation. Mean pup weight was observed to be significantly reduced in the 10 mg/kg day treatment group.

4.4.3 Adverse Effects of Fluoride

4.4.3.1 Fluoride Toxicity in Plants

Hydrogen fluoride is one of the most phytotoxic of all air pollutants because it is carried in the transpiration stream, leading to accumulation in the apex and margins of leaves (Weinstein and Davison 2004). Fluoride elicits various effects in plants, including growth retardation, loss of germination, reduction in quality of harvested plants, and changes in plant metabolism. Sensitivity to fluoride varies between plant species, which subsequently leads to changes in the community structure of the plants. Grass species have been categorized by Davison and Weinstein (1998) as being:

- Sensitive to intermediate (e.g., maize and sorghum)
- Intermediate (e.g. *Poa* sp., and rye);
- Intermediate to tolerant (e.g., perennial ryegrass, oats, barley, wheat, and rice)
- Tolerant (e.g., bahia grass).

NAS (1971) categorized other plants by sensitivity to airborne fluoride:

- Sensitive (e.g., lichens, mosses, young pine needles, and young barley plants)
- Medium sensitive (e.g., oats, mature barley, and willow)
- Less sensitive to insensitive (e.g., birch, currants).

Airborne fluoride causes necrosis and chlorosis in plants such as grass and clover. Guderian et al. (1969) found chlorosis on white clover and necrosis on grass after fumigation with 2.6 $\mu g/m^3$. Exposure to hydrogen fluoride at a concentration of 2.8 $\mu g/m^3$ for 48 days resulted in mortality of leaf tips in various grasses (Guderian et al. 1969). Research on oats exposed to hydrogen fluoride in air (5.1 $\mu g/m^3$) resulted in a decrease in yield over 15 days (Guderian et al. 1969). Weinstein and Davidson (2003) suggested that injury to the most sensitive vegetation by hydrogen fluoride occurs at concentrations below 0.8 $\mu g/m^3$, for a 1–3 day period, with a long-term threshold concentration of 0.25 to 0.3 $\mu g/m^3$.

Palmason and Skye (1999) conducted an evaluation of effects on plants of fluoride emissions from a proposed smelter at Reydarfjordur, and concluded that moss heath vegetation would be the most sensitive to effects because of the presence of sensitive species, such as mosses, lichens, and bog whortleberries (*Vaccinium uliginosum*). These authors postulated a lower

likelihood of effects associated with heath land, and that effects to the grassland habitat were unlikely.

WHO (1984) established criteria suggesting that long-term exposure to hydrogen fluoride should not exceed 0.2 $\mu g/m^3$. These criteria were based on McCune (1969a,b) who produced dose-response curves for a number of species. The responses for plants are non-linear and there are negative relationships between concentration and length of exposure necessary to cause an effect, so air quality criteria must be stated in terms of time-related concentration.

In the past, the Norwegian Pollution Control Authority recommended air quality criteria for hydrogen fluoride in vegetation ranging from 1 $\mu g/m^3$ over the averaging period of 1 day, to 0.3 $\mu g/m^3$ for 6 months (Ongstad et al. 1994).

McCune (1969a,b) also studied fluoride injuries in plants, and his data were re-examined by Davison (2005), resulting in a composition curve. Based on the curve, maximum long term fluoride concentrations should be less than 0.3 $\mu g/m^3$ to protect the most sensitive plant species studied by McCune (1969a,b).

Pine species vary considerably in sensitivity, but some are almost as sensitive as the most sensitive monocots. Pine needles are only sensitive during the period of expansion so timing of exposure is important (Weinstein and Davison 2004). An extensive literature search was conducted to find an appropriate toxicity threshold for fluoride that would protect lodgepole pine. The search focused on *Pinus* species, given the known susceptibility of this species to fluoride (Doley 1988; Davison 2005) and its importance to the community in the assessment area, but included other plant species as well. Most of the papers that were discovered did not offer the information needed to determine an appropriate toxicity threshold. For example, some papers did not report air concentrations resulting in effects (e.g., Carlson et al. 1979), or time periods of exposure were short-term as opposed to long-term. Doley (1988) studied the inhibition of photosynthesis in four taxa of pine trees grown for 125 days in gaseous hydrogen fluoride at 0, 1.2, 1.8, and 4.3 $\mu g/m^3$. Results indicated that at concentrations of 1.2 $\mu g/m^3$, photosynthesis rates were significantly greater than at the other concentrations. Doley (1988) concluded that at less than approximately 1 $\mu g/m^3$, ambient fluoride concentrations are not likely to have deleterious short-term effects on chlorophyll concentrations and concentrations in *Pinus* spp.

4.4.3.2 Fluoride Toxicity in Birds

Toxicity of fluoride to birds has been studied in numerous papers that have documented the accumulation of fluoride in bones and eggshells. The existing literature has not clearly related tissue concentrations of fluoride in birds to biological effects. Fleming (1996) stated that adverse effects to wild birds or bird populations are rarely attributed to fluoride toxicity. In one field study by Andreasen and Stroud (1987, as cited in Fleming 1996) snow geese (*Chen caerulescens*) that had died from apparent fluoride toxicity had elevated tissue concentrations (32–129 mg/kg in the liver and 6.8–25 mg/kg in the brain) relative to geese in reference areas (<1 mg/kg in the liver and brain). In a study by Vikoren and Stuve (1996), no differences in egg volume, shell thickness, egg fertility, or bone morphology were found between female herring gulls (*Larus argentatus*) and common gulls (*Larus canus*) exposed to fluoride emissions

(concentrations were not stated) in Norway than in reference site gulls, although the exposed gulls had higher fluoride concentrations in femurs and eggshells.

Available laboratory studies of avian fluoride toxicity relate dietary fluoride exposure to potential effects on survival, growth, and reproductive performance. For example, day-old Japanese quail (*Coturnix japonica*) exposed to 750 mg/kg fluoride experienced more than two-fold higher mortality than control birds (Chan et al. 1973). A growth study by Nahorniak et al. (1983) found that male turkeys fed corn-soybean diets (containing 3 to 5 mg/kg fluoride) supplemented with 400 mg/kg fluoride had significantly lower body weights than turkeys fed diets supplemented with 0 to 200 mg/kg fluoride. A reproduction study by Pattee et al. (1988) determined that screech owls (*Otus asio*) produced fewer fertile eggs and young per clutch when exposed to 232 mg/kg fluoride in their diet for six months.

4.4.3.3 Fluoride Toxicity in Mammals

Fluoride accumulation in vegetation by direct deposition from the atmosphere, uptake through plant leaves, or uptake through roots from soil can be substantial enough to cause toxic effects (e.g., fluorosis) in herbivores. Fluoride intake in mammals can lead to acute poisoning if the doses are large, while chronic toxicity in the form of fluorosis can occur from prolonged exposure at lower doses (Cooke et al. 1996). Fluorosis is caused by excessive intake of fluoride, and causes dental and skeletal lesions, hyperostosis, osteopetrosis, and bone deformities. Fluorosis studies have mainly concentrated on cattle because of their economic importance. Krook and Maylin (1979, as cited in Stratus 2000) and Stevens et al. (2000) reported that 40 mg/kg fluoride in diet of cattle can cause osteosclerosis, osteonecrosis, failure of proper bone growth, discoloration and mottling of teeth, tooth attrition, and other effects.

Chronic fluoride toxicity to small mammals has been documented as damage to teeth. Tooth lesions can affect normal operation of teeth, leading to reduced food intake, and subsequently, starvation (Cooke et al. 1996). Field voles from contaminated sites were found to have dental fluorosis when concentrations of fluoride in femur bone were 189 mg/kg (Boulton 1992, as cited in Cooke et al. 1996).

There are currently no regulatory criteria regarding the adverse effects of fluoride on mammals. However, the published literature suggests various thresholds for body burden and dietary intake of fluoride. For example, body/tissue burden threshold of 10 mg fluoride/L blood plasma, is a reasonable threshold below which adverse effects would not be expected to occur in small mammals (Cooke et al. 1996). However, concentrations of 2,000 mg/kg dry weight in teeth and 2,500 mg/kg dry weight in femur or whole skeleton are indicative of sublethal effects and a shortened life span (Cooke et al. 1996).

Dietary thresholds of fluoride in grass were reported by Shupe et al. (1987). A critical fluoride concentration of 40 mg/kg is expected to prevent chronic toxicity in herbivorous small mammals. However, a concentration of 100 mg/kg in grass causes marked dental fluorosis and death after two to three months of dietary exposure in experimental field voles.

4.4.4 Derivation of Protective Plant Criteria Thresholds

4.4.4.1 Sulfur Dioxide Plant Criteria

A number of studies were reviewed to determine the appropriate criteria to use to examine whether air concentrations of SO_2 under the base case (without seawater scrubbers) and alternative case (with seawater scrubbers) would be associated with potential effects on various sensitive plant communities.

Protection of Mosses and Lichens—For the purposes of this risk assessment, the SO_2 air concentration of 10 $\mu g/m^3$ in air was selected as the threshold criteria for mosses and lichens. This value corresponds with the United Nations Economic Commission for Europe SO_2 criterion of 10 $\mu g/m^3$ as the annual mean for SO_2 in air. In the UK, the ambient air SO_2 criterion of 20 $\mu g/m^3$ (annual mean and winter) has been adopted, as stated in Council Directive 1999/30/EC, in the Official Journal of the European Communities (April 1999; EC 1999). Although some lower levels were summarized above, the establishment of a direct causal connection between plant damage and atmospheric SO_2 concentrations is complicated by the emission and dispersion of many other potential pollutants present in the Arctic. Additionally, the value of 10 $\mu g/m^3$ was established to protect sensitive lichens, given their important ecological role and abundance in harsh arctic ecosystems, such as in the study site.

Protection of Heather/Heath Plant Communities—A separate threshold criterion equal to 30 $\mu g/m^3$ was selected to assess whether SO_2 concentrations were present at potentially harmful values, as discussed above. This threshold value was compared to SO_2 concentrations associated with both the base case and the alternative case.

4.4.4.2 Hydrogen Fluoride Plant Criteria

Given the general consistency of criteria values suggested in the above fluoride toxicity section, two separate criteria are used to evaluate effects from hydrogen fluoride.

Protection of Sensitive Plant Communities—A toxicity threshold for fluoride of 0.2 $\mu g/m^3$ is selected to protect sensitive plants (including lichens and mosses) throughout the year from effects of hydrogen fluoride (McCune 1969a,b; Davison 2005).

Protection of Pine Communities—For pine, a toxicity threshold of 0.3 $\mu g/m^3$ during the months of June, July, and August is selected because this represents the most sensitive time of the year for pine, which corresponds with the period of needle expansion in Iceland.

4.5 Risk Characterization

Risk characterization combines the results of the effects characterization with the exposure characterization to quantify whether the representative receptors have the potential to be adversely affected by the chemicals of concern. In this section the modeled air concentrations for the site are compared to the benchmark air concentrations developed in Section 4.2. This risk characterization specifically answers the risk questions that were posed in Section 4.1.2.

4.5.1 Evaluation of Assessment Endpoint 1: Protection and Maintenance of Local Populations and Communities of Plants

In this section, SO_2 and hydrogen fluoride air concentrations predicted for the base case (without seawater scrubbers) and alternative case (with seawater scrubbers) scenarios are compared to air quality criteria and toxicity thresholds. The purpose of this analysis is to answer Risk Question 1: Do SO_2 and hydrogen fluoride levels that will be present in ambient air, under the base case (without seawater scrubbers) or the alternative case (with seawater scrubbers), have the potential to adversely affect sensitive plant communities?

4.5.1.1 Sulfur Dioxide Concentrations and Moss/Lichen Communities

Figure 4-1 illustrates that SO_2 concentrations in air do not exceed the threshold concentration of 10 $\mu g/m^3$, and therefore, no effects to mosses or lichens or similarly sensitive plants or bryophytes are expected either with or without seawater scrubbers.

4.5.1.2 Sulfur Dioxide Concentrations and Heather Plant Communities

The SO_2 air concentrations do not exceed the heather/heath threshold of 30 $\mu g/m^3$ (Figure 4-1), and therefore, no effects to heather plant communities, or similarly sensitive plant communities, are expected under either scenario, either with or without seawater scrubbers.

4.5.1.3 Hydrogen Fluoride Concentrations and Sensitive Plant Communities

Figure 4-2 shows the modeled hydrogen fluoride air concentrations for both the base case (without seawater scrubbers) and alternative case (with seawater scrubbers) scenarios. When compared to the 0.2 $\mu g/m^3$ criteria, the majority of the annual mean of concentrations in the study area fall below the critical level. More air concentration data tend to exceed the criteria in the alternative case (with seawater scrubbers) than in the base case (without seawater scrubbers). Without seawater scrubbers, there are no concentrations outside the dilution zone that exceed the criteria, and within the dilution zone, 96 percent of the data are distributed below the criteria, indicating minimal exceedances.

Figure 4-3 similarly shows the distribution of fluoride air concentrations for both the base case and alternative case, except that the concentrations are calculated using the growing season emission rates. Therefore, this time period represents a sensitive time of year for growing plants. The median air concentrations within the dilution zone for the alternative case (with seawater scrubbers) exceed the criteria, but for the base case (without seawater scrubbers) only 9 percent of the data exceed the criteria. Outside the dilution zone, none of the concentrations exceed the criteria under the base case.

Figures 4-4 and 4-5 illustrate where exceedances occur in the study area, in the base case (without seawater scrubbers) and in the alternative case (with seawater scrubbers). Fluoride exceeds the sensitive plant community criterion outside the dilution zone only under the alternative case (with seawater scrubbers) during the April–September growing season period. However, the areal extent of potential risk drastically declines without seawater scrubbers, for both time periods.

To summarize, fluoride air concentrations exceed critical toxicity thresholds for plants at fewer locations without seawater scrubbers than with seawater scrubbers, regardless of time of year. Outside the dilution zone, none of the air concentrations associated without seawater scrubbers exceed the WHO criterion of 0.2 $\mu g/m^3$, and therefore hydrogen fluoride concentrations are not expected to pose risks to sensitive plant communities. However, inside the dilution zone, there are some exceedances for data distributed above the 75th percentile for the base case (without seawater scrubbers). If seawater scrubbers are employed, effects thresholds for plants may be exceeded beyond the dilution zone.

4.5.1.4 Hydrogen Fluoride Concentrations and Pine Communities

The most sensitive time of the year for *Pinus* spp. is during the period of needle expansion from June through August. When compared to the 0.3 $\mu g/m^3$ lodgepole pine threshold criterion, the distribution of fluoride air concentrations for the alternative case (with seawater scrubbers) exceeded the criterion within and outside the dilution zone, but data associated with the base case (without seawater scrubbers) exceeded the criterion only inside the dilution zone and only for a few data points (i.e., < 5 percent) (Figure 4-6).

This pattern is illustrated in Figure 4-7. The area of potential risk does not extend into the Teigagerdi forestry area. Additionally, without seawater scrubbers, there would be very few exceedances expected. This indicates that there would be no appreciable risk to existing or future pine communities without seawater scrubbers, but the potential for risk to future pine communities increases if seawater scrubbers are installed.

4.5.2 Evaluation of Assessment Endpoint 2: Protection and Maintenance of Local Populations and Communities of Birds and Mammals

The primary line of evidence for evaluating the bird and mammal assessment endpoints is estimation of dietary exposure for the ecological receptor species. In this section, the PAH and fluoride air concentrations present in the study area under both the base case (without seawater scrubbers) and alternative case (with seawater scrubbers) are compared to the benchmark air concentrations that were developed using the food-web models for ptarmigan, mice, and Icelandic sheep. The TRVs were used to determine threshold dietary concentrations below which effects are unlikely (e.g., risk is negligible).

The purpose of this section is to answer Risk Question 2: Do PAH and fluoride emissions that will be present in the ambient air, and hence plants, under the base case (without seawater scrubbers) or the alternative case (with seawater scrubbers) have the potential to adversely affect birds or mammals via dietary ingestion?

The benchmark air concentrations were compared to modeled air concentrations expected in the study area. Because many conservative assumptions were used in developing the benchmarks, exceedance of the benchmark air concentrations does not mean that adverse effects are necessarily expected. Exceedance of a benchmark simply indicates the potential for adverse effects in sensitive species and signals the need for further evaluation of site-specific risks. If

the modeled air concentrations of the chemicals do not exceed the benchmark concentrations for mammalian and avian diet, the chemicals are unlikely to cause adverse ecological effects.

4.5.2.1 Potential Risk from PAHs

The three wildlife receptors evaluated in this risk assessment that could potentially use the study area for foraging included the ptarmigan, wood mouse, and Icelandic sheep. Figure 4-8 displays the distributions for modeled PAH deposition (with and without seawater scrubbers) and distributions for PAH deposition that would result in exposures equivalent to the NOAEL TRVs for each receptor assuming two different diets, either 100 percent heather or 100 percent grasses. The modeled PAH deposition rates associated with both the base case (without scrubbers) and alternative case (with scrubbers) are well below the PAH deposition rates that would result in excessive exposure to ptarmigan, mouse, and sheep. Therefore, PAHs do not pose risk to any of the wildlife receptors under either the base case or alternative case scenarios.

4.5.2.2 Potential Risk from Hydrogen Fluoride

Figure 4-9 displays the distributions for modeled hydrogen fluoride concentrations in air (with and without seawater scrubbers) and distributions for hydrogen fluoride that would result in exposures equivalent to the NOAEL TRVs for each receptor, assuming that receptors consumed either 100 percent heather or 100 percent grasses. The fluoride concentrations in air associated with both the base case (without scrubbers) and alternative case (with scrubbers) scenarios are below hydrogen fluoride air concentrations that would result in excess exposure for all three receptors consuming 100 percent grasses, and for sheep consuming either 100 percent heather or 100 percent grasses. However, if ptarmigan and mice are assumed to consume 100 percent heather, both receptors could potentially experience risk because of their exceedance of the dietary based NOAEL TRV benchmark. That is, there is some overlap in the distributions of modeled air concentrations and RBCs.

Ptarmigan—Assuming ptarmigan consume 100 percent heather, the highest potential for risk to this receptor exists for the alternative case (with seawater scrubbers) scenario, and would encompass the facility, with potential risk gradually declining with increasing distance from the facility (Table 4-2). There is less than a 50 percent chance that in the environs surrounding the facility, ptarmigan that consume 100 percent heather would be exposed to levels of fluoride exceeding the NOAEL TRV benchmark. This represents only 0.11 percent of the study area, or 21 ha (Table 4-2). The corresponding area for the base case (without seawater scrubbers) encompasses only 0.07 percent of the study site, or 14 ha.

The LOAEL TRV benchmark is not exceeded in either case for ptarmigan consuming 100 percent heather (Figure 4-10). In summary, ptarmigan that hypothetically consume 100 percent heather are exposed to greater risk from hydrogen fluoride with seawater scrubbers than without seawater scrubbers. Whereas a few hydrogen fluoride concentrations slightly exceed the NOAEL TRV benchmark under the baseline (without seawater scrubbers) scenario, none of the concentrations exceed the LOAEL TRV benchmark. Furthermore, if it is assumed that ptarmigan consume 100 percent grasses, toxicity thresholds are not exceeded under either scenario.

Wood Mouse—A similar pattern exists for mice as for ptarmigan. Probability of exposure in excess of the TRV is greatest near the facility, such that there is more than a 50 percent chance for exposure exceeding the NOAEL TRV for mice consuming 100 percent heather. The portion of the study area associated with this level of exposure is an area of 7 ha or 0.03 percent of the study area. The probability of risk is reduced under the baseline scenario (without seawater scrubbers), where again the probability of exposure above the TRV is greatest near the facility, and decreases as one moves away from the facility (Table 4-2). The portion of the study area associated with a greater than 50 percent chance of exposure in excess of the TRV is 4.5 ha or 0.02 percent of the study area (Table 4-2).

When compared to the LOAEL TRV, the probability of exposure in excess of the TRV does not exceed 50 percent in any case. Under the base case (without seawater scrubbers), there is an area of 40 ha (0.21 percent) of the study area with a probability of TRV exceedance between 20 and 50 percent. The corresponding area for the alternative case (with seawater scrubbers) is 160 ha (0.83 percent) of the study area.

4.6 Population Impact Modeling

In order to determine the potential for population effects at varying levels of exposure, the relationship between the stability of local subpopulations of rock ptarmigan and wood mouse and exposure to PAHs and fluoride was modeled. For modeling purposes, a population is deemed to be stable if it is able to maintain its numbers at time t through the next reproductive cycle (t+1), given assumed rates for mortality and reproduction. If the capacity to replace individuals lost either through natural or induced mortality is greater than or equal to the mortality rate, then the population is deemed to be stable. If, however, the reproductive capacity of the subpopulation declines below the projected mortality rate, then the subpopulation is deemed unstable and will need to be supported by outside immigration (or an increase in reproduction or decrease in mortality) to avoid a local extinction event. The objective of this modeling is to determine the exposure level for PAHs and fluoride at which receptor populations might become unstable as a result of decreases in reproduction.

4.6.1 Population Modeling for the Rock Ptarmigan

The population model used for the rock ptarmigan is based on the study by Magnusson et al. (2004) that reports a population for this species based on demographic data collected in northeastern Iceland between 1981 and 2004. The model used in this case was as follows:

$$N^t = e^{-Z_2^t}\left\{e^{-Z_{X,W}^t}\left(\gamma N^{t-1} + \lambda e^{-Z_2^{t-1}} N^{t-2}\right) + N^{t-1}\right\}$$

The best fit for the data was found using the assumptions of $\lambda=0$ and excluding the outliers observed in 1988 and 2000. Using these assumptions, the model simplifies to the following:

$$N^t = e^{-Z_2^t}\left\{e^{-Z_{X,W}^t} \cdot \gamma N^{t-1} + N^{t-1}\right\}$$

where:

N^t = total population at time t (individuals)

Z^t_1 = annual mortality for reproductive hens 1 year of age (per year)

Z^t_2 = annual mortality for reproductive hens greater than 1 year of age (per year)

Z^t_{XW} = mortality for non-reproductive juveniles from late summer to following spring (per year)

γ = ratio of reproductive capacity of 1-year-old birds over the survival of adult birds between spring and late summer (3.92, SE 0.495).

The toxicological endpoints for the exposure of ptarmigans to both PAHs and fluoride are diminished reproductive capacity (please see Section 4.2.2.2 for a discussion of TRVs). For PAHs, the TRV represents 100 percent reproductive failure and for hydrogen fluoride, the TRV represents a significant reduction in growth. For purposes of simplicity, the modeling presented here assumes that exposure of a ptarmigan hen to a dose of PAHs and/or fluoride at concentrations greater than the TRV would result in total reproductive failure. This assumption is likely to result in an overestimate of the probability of population instability.

If a ptarmigan hen realizes complete reproductive failure, then the γ value for that individual is zero. Natural variation in reproductive success is factored over all individuals to derive the projected value for γ listed above. In order to integrate the reproductive impact resulting from exposure to the PAH and fluoride, the affected proportion (p) of the subpopulation N^{t-1} was segregated from the unaffected proportion (1-p) based on the projected level of impact (see Section 4.5.2). As such, the population model is modified to the following:

$$N^t = e^{-Z_2^t}\left\{e^{-Z_{X,W}^t}\cdot\left(\gamma(1-p)N^{t-1}+(0)pN^{t-1}\right)+N^{t-1}\right\}$$
$$= e^{-Z_2^t}\left\{e^{-Z_{X,W}^t}\cdot\gamma(1-p)N^{t-1}+N^{t-1}\right\}$$

The model results reported by Magnusson et al. (2004) indicated increasing adult mortality rates (Z^t_1, Z^t_2) over the 23 years of observation. Extrapolation of this prediction results in an already unstable population dynamic. Rather than rely on this prediction, the model was run over iterations of the observed 23-year cycle at discrete values of p. This eliminated the requirement to discern the carrying capacity because the implied limitations are inherent in the observed mortality rates. The models were started with a seed value of 100 and allowed to run over 4 cycles (92 iterations) in order to equilibrate. Within the fifth cycle, the change in the annual growth capacity (G) was determined as follows:

$$G = \frac{N^t - N^{t-1}}{N^{t-1}}$$

The overall prediction of growth capacity was taken as the average annual value of G over the total of the 23 iterations within the fifth cycle. This permitted the model to achieve equilibrium so that initial seed values used did not affect the final outcome.

Results of the model are illustrated in Figure 4-11. At p=0, the predicted growth capacity, G, for the ptarmigan subpopulation is projected to be between 7.55 and 16.56 percent (5th and 95th percentiles) with a 50th percentile probability of 12.0 percent. If it is assumed that all hens are exposed to concentrations of PAHs or fluoride exceeding the TRV (i.e., p=1), G is projected to have a value of –59.1 percent. The negative value is indicative of a projected annual population decline. The predictions of the 50th percentile values were represented as the following linear function:

$$G = G_0 + ap$$

Parameter	5% Confidence	50% Confidence	95% Confidence
G_0	0.0834	0.120	0.1575
a	–0.673	–0.712	–0.748
Stability Limit p at G=0	0.134 (≅1/7)	0.169 (≅1/6)	0.211 (≅1/5)

4.6.2 Population Modeling for the Wood Mouse

The wood mouse is a small rodent with a rapid reproductive cycle and short life span. This species reaches reproductive maturity by 4 months of age, and rarely lives past 12 months (Wilson et al. 1993). Typically, a mature wood mouse will produce 4 litters during a breeding season, which in Iceland extends from early April to the end of October/early November (Bengtson et al. 1989). Typical litters range from 4 to as many as 9 pups (Montgomery 1989).

The population stability of the wood mouse is more difficult to interpret than that of the ptarmigan. This is because the risk assessment looked at two distinct endpoints for mammalian sentinels. First, the risk associated with exposure to PAHs was based on potential reduced fecundity because of significantly reduced birth weights as the result of exposure to PAHs during gestation. Second, the risk associated with injection of hydrogen fluoride is an increase in mortality based on a duration of exposure of 6 months.

In order to accommodate the potential for population effects resulting from either increased mortality or reduced fecundity, the indigenous mice were modeled in a population matrix with a resolution of 1 month and duration of 100 years. Baseline conditions were determined as follows:

$$n_0^t = \begin{cases} \text{Apr} - \text{Oct} & \sum_{c=4}^{12} n_c^t \cdot \frac{4 \cdot r_c}{7} \\ \text{Nov} - \text{Mar} & 0 \end{cases}$$

$$n_c^t = \begin{cases} \text{Apr} - \text{Aug} & sa_{c-1}^{t-1} \\ \text{Sept} - \text{Nov} & sb_{c-1}^{t-1} \\ \text{Dec} - \text{Mar} & sc_{c-1}^{t-1} \end{cases} \cdot n_{c-1}^{t-1} \quad c \in \{1 \ldots 12\}$$

$$N^t = \sum_{c=0}^{12} n_c^t$$

where:

t	=	generation time (years)
n^t_0	-	number of neonates (cohort 0) at time t
n^t_c	=	number of individuals in the monthly cohort c at time t
N^t	=	total number of individuals in the population at time t
r_c	=	litter number for monthly cohort c (individuals/birthing)
sx^t_c	=	monthly survival for the c monthly cohort c during season x (x=a, spring/summer; x=b, summer/fall; x=c, winter).

Distribution parameters are provided in Table 4-3.

The carrying capacity of a region is defined as the number of animals a given area can support before induced increases in mortality and/or reductions in fecundity reduce the population growth to zero. In typical heather-dominated landscapes in Iceland, the principal limit on growth is the availability of fodder. The estimated minimum carrying capacity (K), based on trapping results derived from regions around Vik, Iceland, is 41.7 ± 8.03 individuals per hectare (Bengtson et al. 1989). Bengston also reported that the sex ratio of the wood mouse is, for the most part, not significantly different from unity. Therefore, 1/2 K can be used as the female carrying capacity.

The introduction and evaluation of the impact associated with exposure to fluoride and PAHs was accomplished by partitioning the mortality and fecundity, respectively, between the baseline population response, and that portion of the population forecasted to be affected by exposure to the substances of concern. For purposes of simplicity in this modeling, it is assumed that exposure to PAHs in excess of the TRV results in complete reproductive failure. This assumption is likely to result in an overestimate of the likelihood of population instability from exposure to PAH since the measured endpoint used to derive the TRV was only a significant reduction in pup birth weight. Application of this endpoint in this case assumes that reduced birth weight equates to 100 percent neonate mortality. Similarly, it is assumed that the observed 82 percent increase in female mortality resulting from a 6-month exposure to hydrogen fluoride at 300 ppm was equivalent to 100 percent mouse mortality at or above the TRV, which was based on an exposure of 200 ppm. The partition of the impact was allocated as follows:

$$r_c = p \cdot 0 + (1-p) \cdot r_{c0}$$
$$sx_c = p \cdot 0 + (1-p) \cdot sx_{c0}$$

Where p is the proportion of the population deemed affected based on the results of the risk assessment.

Because the population densities affect reproduction and mortality rates as the population approaches the carrying capacity, K, background mortality and reproduction estimates are only accurate so long as the population is significantly below this level. Therefore, in order to test the magnitude of the induced impact, the initial female population is artificially set at ¼K. This would simulate a significant mortality event independent of the chemicals of concern. The more vital the population, the faster the female population will rebound to ½K as follows:

$$t_\psi = f\left(N^t = \sum_{c=0}^{12} n_c^t = K \right) \quad \text{and } N^{t_1} = \frac{1}{2}K$$

where t_ψ it the year that the population returns to the carrying capacity K. The value of N^t was evaluated only for the month of March. Because March is the month prior to the start of the breeding season, this represents the minimum population for the year.

The greater the assessed impact on the population, the slower the rate of rebound. This will continue until the impacts are so great that the female population is unable to return to ½K and declines toward local extinction. It is the time of this rebound under various levels of impact that is used as the endpoint to evaluate the status of the population. Illustrations of this relationship at the 5th, 50th and 95th percentiles are provided in Figures 4-12 through 4-14.

4.6.3 Integration of Population Models with Risk Assessment Results

The population models discussed above describe the response of the ptarmigan and wood mouse populations to various levels of exposure to PAHs and fluoride. The risk assessment was evaluated with regard to the proportion of the population likely to receive a level of exposure equal to or greater than the TRV dose. Therefore, the models can be combined based on the proportion of individuals affected and the resulting effect on the stability of the populations.

For the ptarmigan, it is assumed that exposure to either fluoride or PAHs in excess of the TRV will result in reproductive failure (a conservative assumption). Therefore, the population dynamics can be characterized by an equilibrium model that defines a level of impact from either chemical as the point at which population growth is at or below 0. This can be illustrated as a contour at the 50th percentile and the results indicate that the exposure of the local ptarmigan population to hydrogen fluoride and PAHs will not result in an induced negative population growth outside of the plant boundary (Figure 4-15). This conclusion holds for both the base case (without seawater scrubbers) and the alternative case (with seawater scrubbers). Therefore, it can be concluded that the proportional population impacts resulting from exposure to these two chemicals of concern will have no population impact on the ptarmigan in the project area.

For the wood mouse, population stability is evaluated based on the time it takes for the population to rebound from a carrying capacity of ½K to K. This is the result of both the impact on growth from PAHs and assumed mortality from exposure to fluoride. If the rebound is determined to take more than 10 years, but less than 100 years, a population decline is inferred. If the rebound is determined to take more than 100 years, local extinction is inferred. These are illustrated at the 50th percentile in Figure 4-15. The results indicate that nowhere around the aluminum smelter is the proportional exposure high enough to result in the wood mouse populations realizing negative growth. Under the base case (without seawater scrubbers), there is an area of 72 ha (0.37 percent of the study area) that is wholly within the dilution zone where the average wood mouse population would be somewhere between the carrying capacity, and one-half the carrying capacity (i.e., the assumed population growth rate precludes the population from reaching K within a period of 10 years). Under the alternative case (with seawater scrubbers) the corresponding area is 440 ha (2.3 percent of the study area) and extends beyond the dilution zone boundary. Therefore, it can be concluded that the impact of the aluminum smelter would be very minor (reduction in carrying capacity) with regard to the indigenous wood mouse populations.

4.7 Uncertainty Analysis

There are several sources of uncertainty associated with the assumptions in this risk assessment. These sources of uncertainty are common to the risk assessment process in general. There is uncertainty associated with the assumptions made in the selection of exposure parameters, accumulation factors, and TRVs, as well as with data gaps; these are discussed below. Uncertainties inherent in the exposure assumptions specifically include the variability and/or lack of information regarding ingestion rates, the dietary composition of receptors, the contaminant concentrations ingested by the receptors, the portion of the diet obtained from the project area, and the portion of ingested contaminants that is absorbed (i.e., bioavailable). The magnitude of uncertainty differs among sources and can vary with CoPC and receptor.

4.7.1 Uncertainties Associated with Food-Web and Population Model Assumptions

This risk assessment was conducted using diet assumptions that maximized exposure to the receptors from terrestrial sources (i.e., assumed that all fractions of diet were from the site and that each receptor consumed 100 percent aboveground vegetation derived from the site). As mentioned above, the wood mouse consumes mostly seeds, and to a lesser extent buds, fungi, and fruits and tubers (Schlitter and Van der Straeten 2004). Similarly, the ptarmigan consumes berries, buds, germinating seeds, and to a much lesser degree, insects when available. In the food-web model, the receptors were assumed to consume either 100 percent heather or 100 percent grasses. PAHs are deposited onto plants, while fluorides are assimilated by photosynthetic parts of the plants. It is unlikely that the seeds and berries of plants assimilate fluoride to the same extent as the portions of the plant that are photosynthetic, because those portions of the plants are not photosynthetic. Therefore, considering the complete diet composition of the mouse and the ptarmigan, this risk assessment overestimates total exposure to fluoride from food ingestion, because it assumes that the mouse and ptarmigan receptors

consume the entire plant, as opposed to the berries and seeds. The berries and seeds are exposed to air for a much shorter period of time than the entire vegetative plant, therefore, PAH concentrations are overestimated when one assumes the receptors consume the entire plant, not just the reproductive portions.

Area use and temporal use factors are sources of uncertainty that vary with receptor, but not chemical. Benchmarks were developed assuming that all foraging time is spent in the project area. The home range of the wood mouse is undoubtedly small enough to be encompassed by the study area; however, the Icelandic sheep and other grazing animals may or may not use portions of the area for grazing year round. The ptarmigan are nomadic in the winter, moving from one sheltered slope or patch of food to another (Holder and Montgomerie 1993). Some ptarmigan populations winter on their breeding habitat, while others may move to areas near the snowline. It is uncertain how much time the rock ptarmigan and Icelandic sheep would spend on the site, so assuming they use the area 100 percent of the time is a conservative assumption that is likely to overestimate exposure in the food-web models, resulting in conservative risk estimates. Similarly, it is unknown what portion of the study area provides adequate foraging habitat for each receptor, so assuming that 100 percent of the study site is used by the receptors also results in conservative risk estimates.

Therefore, although the ptarmigan and wood mouse could experience potential risk from exposure to fluoride if examined under the NOAEL benchmark scenario, conservative dietary assumptions and the area use assumptions were used to develop the TRV-based benchmarks. Under the LOAEL TRV benchmark scenario, potential risk does not exist for the ptarmigan from fluoride or PAHs, and only slight risk exists for the wood mouse from fluoride, assuming the diet consists of 100 percent heather. If the diet is assumed to consist of 100 percent grass, there is no potential risk to any of the receptors, suggesting that the model is sensitive to the type of food the receptors are assumed to be consuming as well. The assumption that the receptors consume 100 percent heather for their diet overestimates total exposure, resulting in conservative estimates of risk.

In most circumstances, the major source of uncertainty associated with population modeling involves the application of population dynamics from one ecotype to another. In this case, population survey and population characterization for both the ptarmigan and the wood mouse in Iceland were available from the Icelandic Institute of Natural History; thus, ecotype extrapolation is not considered an important source of uncertainty in the population modeling. Other sources of uncertainty include anthropological influences, natural population cycles, and toxicological response. These are described below.

Anthropological Influences—The primary limiting factor in Iceland for both ptarmigan and wood mouse populations is food availability. This is because much of the wilds where the populations were characterized were associated with mature heather and shrub lands. These habitats are generally low in productivity. Some agriculture exists in the Fjardaal area. This introduces early successional habitat, which is high in productivity relative to the reference habitat and therefore would be expected to provide a more abundant food base than heather and shrub. Thus, agricultural fields would likely possess larger carrying capacities and therefore larger populations than heather and shrub habitat. This higher potential carrying capacity is not considered in this analysis because of lack of characteristic data. However, incorporation of this

variable into the analysis would result in an overall increase in the stability of the populations, greater than that predicted by the models. Therefore, the results using a model that assumes no ecotype differences are conservative (i.e., environmentally protective).

Natural Population Cycles—Natural populations are not constant, but typically experience irregular periods of growth and decline. While predicting the precise timing of these fluctuations is very difficult, the probability of a population decline or increase of given magnitude can be inferred because a) the larger the decline, the less likely it is to occur; and b) the lower the population relative to the carrying capacity, the greater the population growth rate. The ptarmigan was modeled base on a 20-year cycle using the observations of Magnusson et al. (2004). During this period the population increased. In the model, it was assumed that the population underwent a major 1-year decline at the end of the cycle relative to the start. This assumed decline was intended to simulate the effects of growth pressure on the population; however, such a decline is not likely to be experienced to the same degree in a natural population and therefore represents an artificial limitation on population growth. Inclusion of this assumption in the population models for the risk assessment results in an overprediction of population level effects resulting from exposure to emissions from the Fjardaal plant.

The wood mouse model is not intended to predict population levels, but rather the potential for population growth. This was projected based on an implied, instantaneous, and catastrophic reduction in the population to half the carrying capacity, which would occur on a 10-year cycle. The artificial constraint placed a reasonable worst-case growth pressure on wood mouse population such that the prediction of overall population stability in the risk assessment can be safely considered to be conservative.

Toxicological Response—The toxicological endpoints used in the derivation of the toxicity reference doses represent the lowest published adverse response for a class of organism to long-term exposure to a CoPC. In this case, these are point estimates because available data precluded the establishment of a dose-response curve. In the population modeling, absolute responses must be used—that is, an individual either dies or survives, and a female either reproduces or fails to reproduce following any given event (e.g., exposure to air emissions). In this assessment, probability of mortality and probability of complete reproductive failure were used as the growth pressures on the ptarmigan and wood mouse. This approach is conservative for the following reasons:

- **PAHs in Birds—**The LOAEL endpoint for exposure to PAHs was complete reproductive failure based on the response to BaP. This PAH is one of the most toxic, while constituting only a small fraction of the total PAHs. This assumption, because of a lack of PAH speciation and differential toxicity data, is likely to overpredict risk and is therefore considered conservative.

- **PAHs in Mammals—**The LOAEL endpoint for exposure to PAHs was low pup birth weight based on the response to BaP. As noted above, this PAH is one of the most toxic, while constituting only a small fraction of the total PAHs. In addition, the population models equated this endpoint with total reproductive failure. This assumption is likely to overpredict risk and is therefore considered conservative.

- **Fluoride in Birds—**The LOAEL endpoint for exposure to hydrogen fluoride was a significant reduction in progeny growth. The population models equated this endpoint with total reproductive failure. This assumption is likely to overpredict risk and is therefore considered conservative.

- **Fluoride in Mammals—**The LOAEL endpoint for exposure to hydrogen fluoride was an 18 percent increase in mortality. The population models equated this endpoint to an LD_{100}. This assumption is likely to overpredict risk and is therefore considered conservative.

In conclusion, uncertainty associated with key assumptions in the population models result in conservative risk predictions regarding the potential for population impacts associated with air emissions from the Fjardaal plant.

4.7.2 Uncertainties Associated with TRVs and Toxicity Thresholds for Plants

TRVs are significant sources of uncertainty in ERAs because laboratory test conditions may not accurately mimic natural exposure, the relative sensitivity of the receptor compared to the test species may be unknown, and the relative identification of the no-effect threshold is imprecise and dependent on dose intervals used in laboratory tests.

In addition to the generalizations regarding the sensitivity of TRVs mentioned above, the avian fluoride TRV selected for the food-web model is clearly conservative. This stems from the fact that the TRV is based on a study of young growing turkeys that had not reached maximum body weight before the study was completed. Given the fact that the food-web models are based on the minimum mean adult female body weight, the avian fluoride TRV is likely quite conservative. It was difficult to locate studies that examined fluoride effects in birds, therefore the Nahorniak et al. (1983) study was used.

Comparison of modeled SO_2 concentrations to conservative toxicity thresholds indicate no potential for risk to plant communities surrounding the facility. SO_2 concentrations in both heather/heath habitat and moss habitats were examined. For heather/heath, the modeled SO_2 concentrations in air in both the base case and alternative case scenarios are well below the threshold concentration of 30 $\mu g/m^3$. Modeled SO_2 concentrations were also compared to a moss threshold of 10 $\mu g/m^3$. Again, modeled air concentrations, under both scenarios, are well below the threshold concentration, indicating no risk to moss communities. Because moss and heather/heath plant communities are considered sensitive to the effects of SO_2, and neither of these communities is expected to be exposed to levels where potential risk could start to be observed, other plant communities in the assessment area can be assumed to be protected as well.

There is a potential that subtoxic concentrations of sulfur dioxide may differentially impart competitive advantages to some plant species, possibly leading to a shift in the composition of the communities in the vicinity of the smelter and thereby altering succession. Plants are capable of using airborne sulfur dioxide as a metabolic sulfur source (Veenranjaneyulu et al.

1991). Chloroplasts possess the capability to reduce sulfur dioxide to bisulfite and sulfide that can be used directly in sulfur amino acid anabolism (Ghisi et al. 1989). This capacity varies among plant species. Under conditions of sulfur limitations, such as in Iceland (UNCSD 1997), an increased capacity to utilize airborne sulfur could impart a competitive advantage. However, field studies on the impact of sulfur dioxide found no increase in plant growth or yield at concentrations lower than approximately 20 $\mu g/m^3$ (WHO 2000). The maximum sulfur dioxide concentrations projected for the smelter were less than 3 $\mu g/m^3$, either with or without seawater scrubbers. Therefore, while it is possible to differentially stimulate plant growth by increasing atmospheric sulfur dioxide concentrations, the magnitude of increases in this case is not likely to affect the composition of the plant communities.

4.7.2.1 Hydrogen Fluoride

Conservative toxicity thresholds were also used to determine if hydrogen fluoride would pose potential risk to sensitive plant communities. Most thresholds evaluated were in the range of 0.2–0.8 $\mu g/m^3$, thus the level of uncertainty about this assumption is not expected to affect the conclusions of the assessment in a meaningful way.

4.7.3 Uncertainties Associated with Modeled Plant Concentrations

Because this assessment is intended to predict situations that currently do not exist, there is no way to validate the plant uptake models against the real situation. In the probabilistic analysis of fluoride and PAH accumulation in plant tissue, every effort was made to illustrate both the uncertainty and variability associated with the predictions. This was accomplished by allowing distributions to represent the predicted parameters necessary to associate available aerial concentration and deposition rates with potential accumulation concentrations in foodstuffs. Many of these parameters were inferred from other locations, other species, or other environmental conditions. Hence, where possible, the distributions were constructed to ensure a conservative assessment, while remaining reasonable with regard to the probability of a given situation occurring. Examples of this were as follows:

- Age distributions for the plant tissue were represented as skewed triangular distributions to favor longer-lived vegetative growth and thereby higher plant accumulation concentrations.
- The period of potential standing biomass was selected to favor overprediction of accumulation concentrations.
- Adhesion factors for depositional materials were assumed to equal unity such that all potential contact equated to an accumulation event.
- Because of considerable uncertainty associated with prediction of elimination of fluoride or PAHs from plant material, no loss of accumulated contaminants from vegetative material was considered.
- Unless otherwise supported by literature results, accumulation of PAHs and fluoride were assumed to be constant right up to the point of senescence.

- Predictions of area-based plant biomass values for heather were conservatively selected based on observations of high altitude stands, thereby conservatively biasing the predictions of per mass accumulation concentrations.
- Fluoride uptake increases with precipitation frequency. Because there was no way to quantify the change in uptakes with the amount of precipitation, every rain event was assumed to represent the optimal conditions for maximum fluoride accumulation rates.

5 Conclusions

5.1 Human Health Risk Assessment Conclusions

In the HHRA, health protective assumptions were applied to estimate exposures to constituents identified as potentially released from the facility with and without seawater scrubbers. Model estimates were then mapped onto the facility footprint and the surrounding land and waterway to determine whether all applicable standards would be met. The finding of this evaluation indicated that there were no instances in which the model estimates predicted that a standard would not be met. The seawater scrubbers do decrease average SO_2 air concentration estimates in the short term (i.e., 1-hour, 3-hour and 24-hour); however, annual estimates are lower without seawater scrubbers. In the short term, both with and without seawater scrubbers, the number of exceedances predicted per year are well below the maximum number of allowed exceedances per year.

The risk assessment also evaluated the chemicals for which toxicity values were available (i.e., PAHs and fluoride). In this screening, estimated air concentrations for PAHs were compared with 1) RBCs derived by U.S. EPA Region 9 using health protective risk assessment assumptions, and 2) more conservative standards identified in some European countries. Although none of the air estimates exceeded any of the available standards or the risk based concentrations, risk estimates were calculated to provide additional information to risk managers.

In this assessment, estimated concentrations of BaP and naphthalene in air were evaluated in a risk assessment for the following receptors: outdoor facility workers; hypothetical residents adjacent to the facility; seagoing workers. The risk assessment applied many health protective assumptions regarding exposure and toxicity as identified by U.S. EPA.

Despite the application of these protective assumptions, all carcinogenic risk estimates were lower than the 10^{-6} risk level identified by many regulatory agencies as the lower end of the acceptable risk range of 10^{-6} to 10^{-4} and all hazard indices were well below the threshold of 1.0, with the highest estimate being 0.00028. These findings suggest that risks are well within acceptable levels under both cases. Risk estimates for PAHs were slightly higher for the alternative case (with seawater scrubbers) than the base case (without seawater scrubbers), but were still well within acceptable levels and the increase in risks was within the level of uncertainty within the estimate.

Potential pathways related to deposition of PAH onto soils were evaluated through derivation of soil concentration estimates for PAHs and fluoride. The estimated soil concentrations were compared with risk-based RBCs for soil and none of these estimates exceeded the risk-based thresholds. Therefore, soil pathways were not evaluated further. Modeled fluoride and particulate matter (using PM_{10}) estimates were well below health standards.

5.2 Ecological Risk Assessment Conclusions

The ERA was undertaken to evaluate risk to plant communities and three wildlife receptors in the vicinity of the Fjardaal smelter under the base case (without seawater scrubbers) and the alternative case (with seawater scrubbers). Modeled exposure concentrations for SO_2 and PAHs do not result in the exceedance of threshold toxicity values at any location under either scenario. Although higher concentrations of SO_2 are modeled under the alternative case with seawater scrubbers, this constituent is not expected to adversely impact mosses, lichens, heather/heath, or other sensitive plant communities under either scenario. Similarly, modeled PAH concentrations are not expected to result in the exceedance of toxicity thresholds for terrestrial mammals or birds at any location under either scenario.

In some limited cases, modeled hydrogen fluoride concentrations may exceed conservative toxicity thresholds. Modeled exposure of plants to hydrogen fluoride indicates that the toxicity threshold for the most sensitive species (mosses and lichens) will be exceeded in limited cases under both scenarios:

- For the base case (without seawater scrubbers) the toxicity threshold will be exceeded by a small margin and over a small area (32 ha on an annual basis and 45 ha on a growing season basis) within the dilution zone
- For the alternative case (with seawater scrubbers) the toxicity threshold will be exceeded by a larger margin and over a larger area (155 ha on an annual basis and 559 ha on a growing season basis) than for the base case.

Modeled exposure of lodgepole pine to hydrogen fluoride indicates that the toxicity threshold for this species will not be exceeded under the base case (without seawater scrubbers) but will be exceeded both within and outside the dilution zone over an area of 217 ha under the alternative case (with seawater scrubbers).

Potential risks from hydrogen fluoride air concentrations were also examined in relation to the herbivorous diet of the wildlife receptors, including the rock ptarmigan, wood mouse, and Icelandic sheep. If it is assumed that these animals consume 100 percent grasses, modeled exposures will not result in the exceedance of toxicity thresholds for any receptor at any location. However, if it is assumed that the wildlife receptors consume 100 percent heather, modeled toxicity thresholds are exceeded at some points for the rock ptarmigan and wood mouse; these thresholds are exceeded at more points under the alternative scenario than the base case scenario.

The foregoing conclusion is true if modeled hydrogen fluoride air concentrations are compared to the NOAEL benchmark. However, if the LOAEL benchmark is used instead, there are no exceedances of toxicity thresholds for the rock ptarmigan without seawater scrubbers. Wood mice may be exposed to concentrations above risk thresholds if it is assumed that they consume 100 percent heather, but this is not true if it is assumed that they consume 100 percent grasses. This conclusion holds for both base case and alternative case when compared to the NOAEL TRV. When compared to the LOAEL TRV, potential risks are greatly reduced, especially in the base case. An examination of population models for these two species indicated no potential for

population impacts to the rock ptarmigan outside the facility boundary and potential reduction in carrying capacity for the wood mouse. Potential impacts to wood mouse extend over a larger area under the alternative case (with seawater scrubbers) than under the base case (without seawater scrubbers). Under the base case, potential impacts to wood mouse are not expected to extend over more than about 0.37 percent of the entire project area, and wholly within the dilution zone. By contrast, these impacts may extend outside the dilution zone for the alternative case.

All risk estimates are likely to overstate actual risk as a result of several conservative assumptions regarding exposure and toxicity. For example, the thresholds for fluoride toxicity to plants are based on sublethal effects that are unlikely to result in mortality to plants if the thresholds are exceeded by a small margin and for short periods. Similarly, the risk estimates for exposure to mammalian and avian receptors assume that individual organisms are exposed throughout their home range and throughout the year. Actual exposures are likely to be much lower. To the extent that any adverse effects to plants, mammals, or birds may be manifest, the likelihood of such effects, on average, is lower under the base case without seawater scrubbers, than under the alternative case, with seawater scrubbers.

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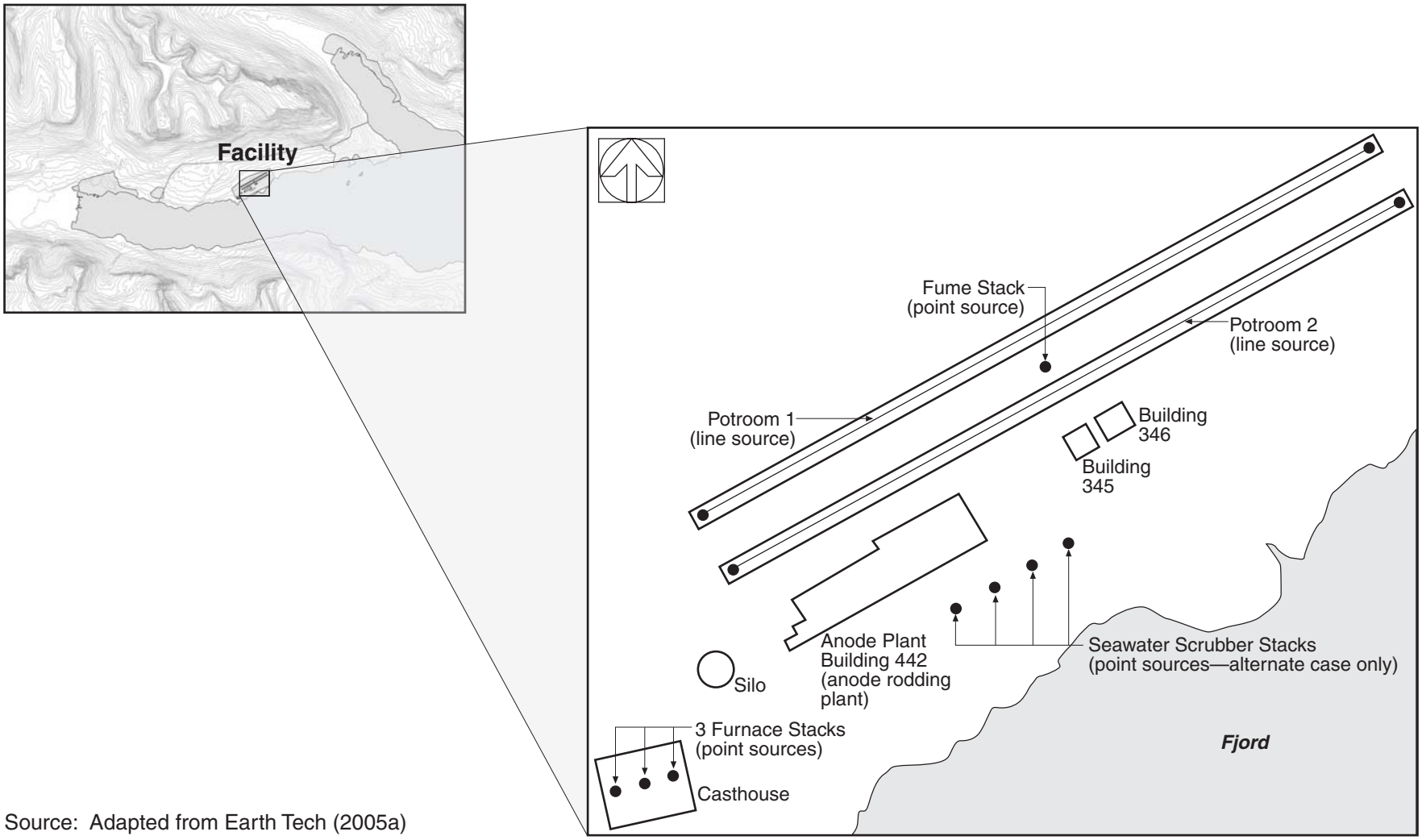
Figures

Figure 1-1. Facility location



Source: Adapted from Earth Tech (2005a)

Figure 2-1. Schematic of air emission sources used in dispersion modeling

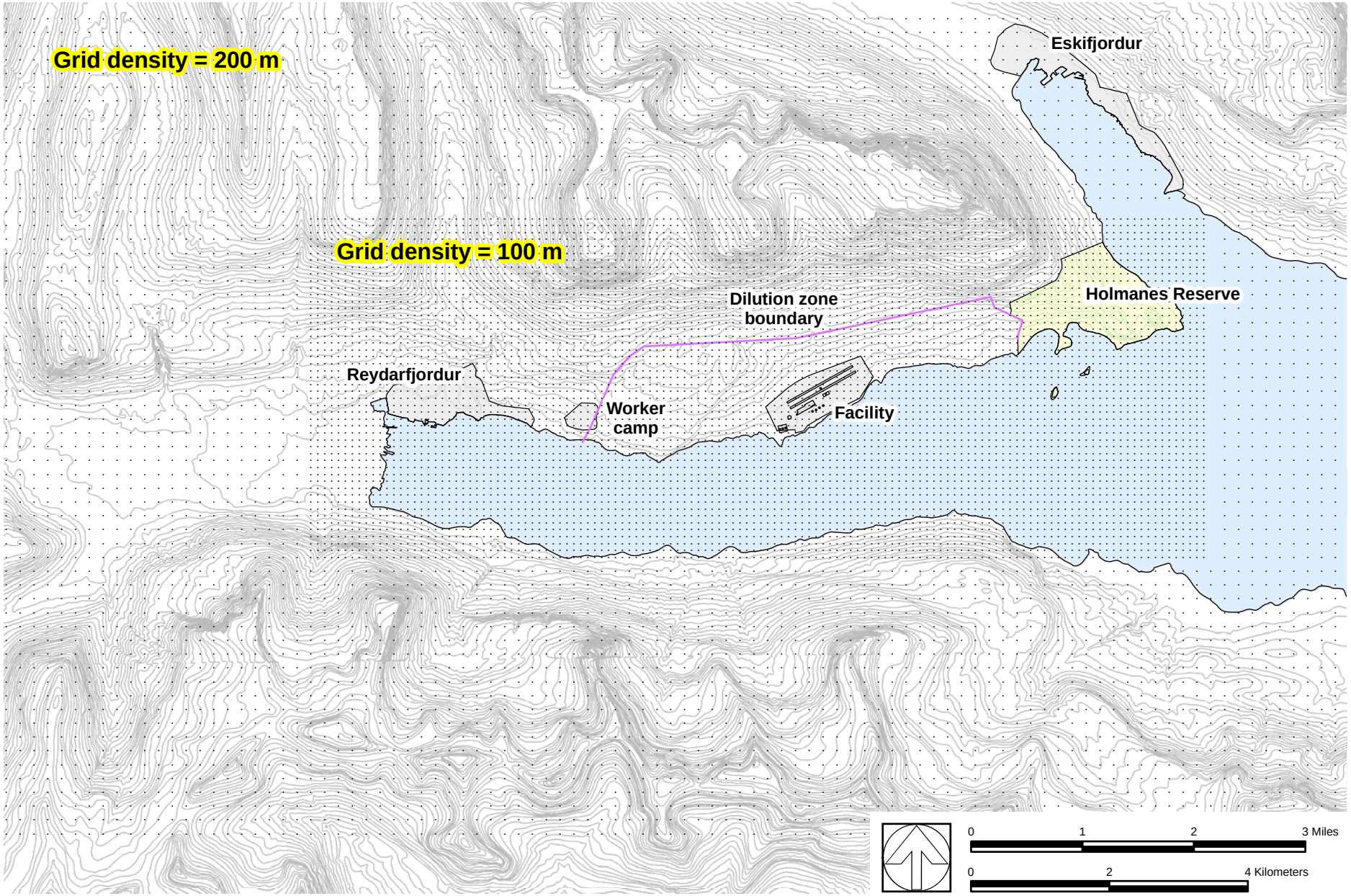


Figure 2-2. Schematic of air dispersion modeling grid relative to key site features

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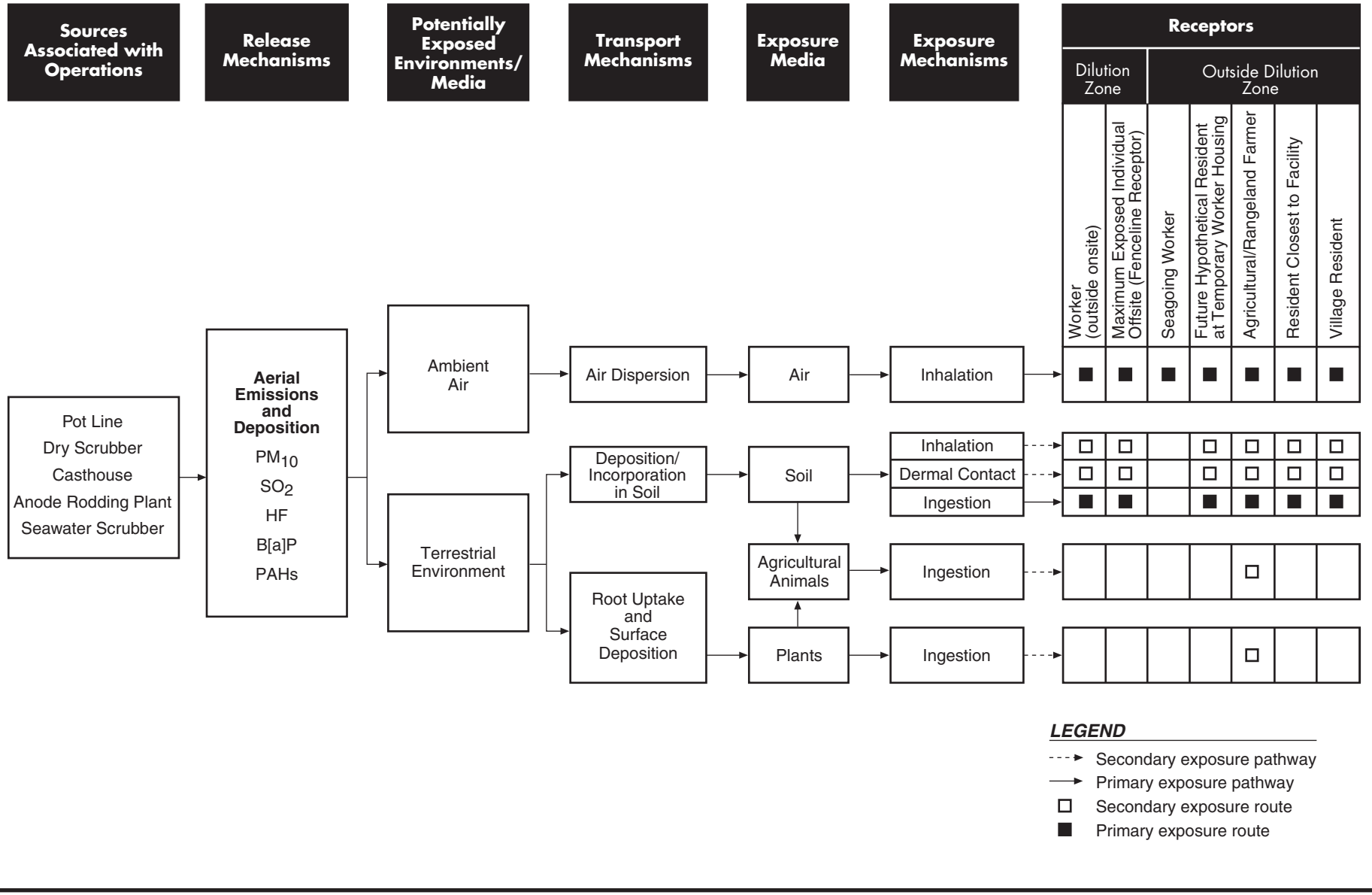

Figure 2-3. Conceptual site model for the Fjardaal human health screening risk assessment

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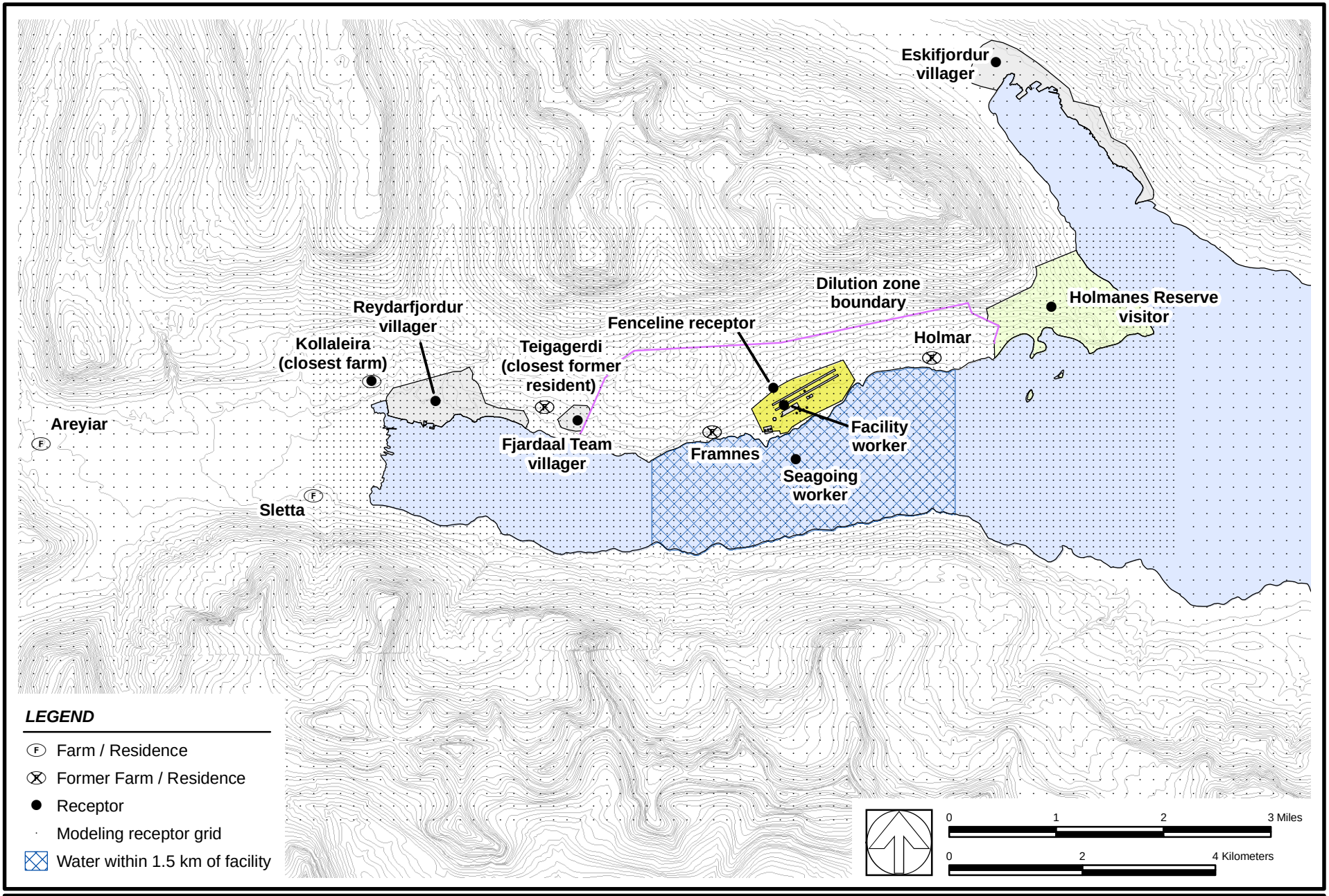

Figure 2-4. Identification of locations used in assessment of human health risk

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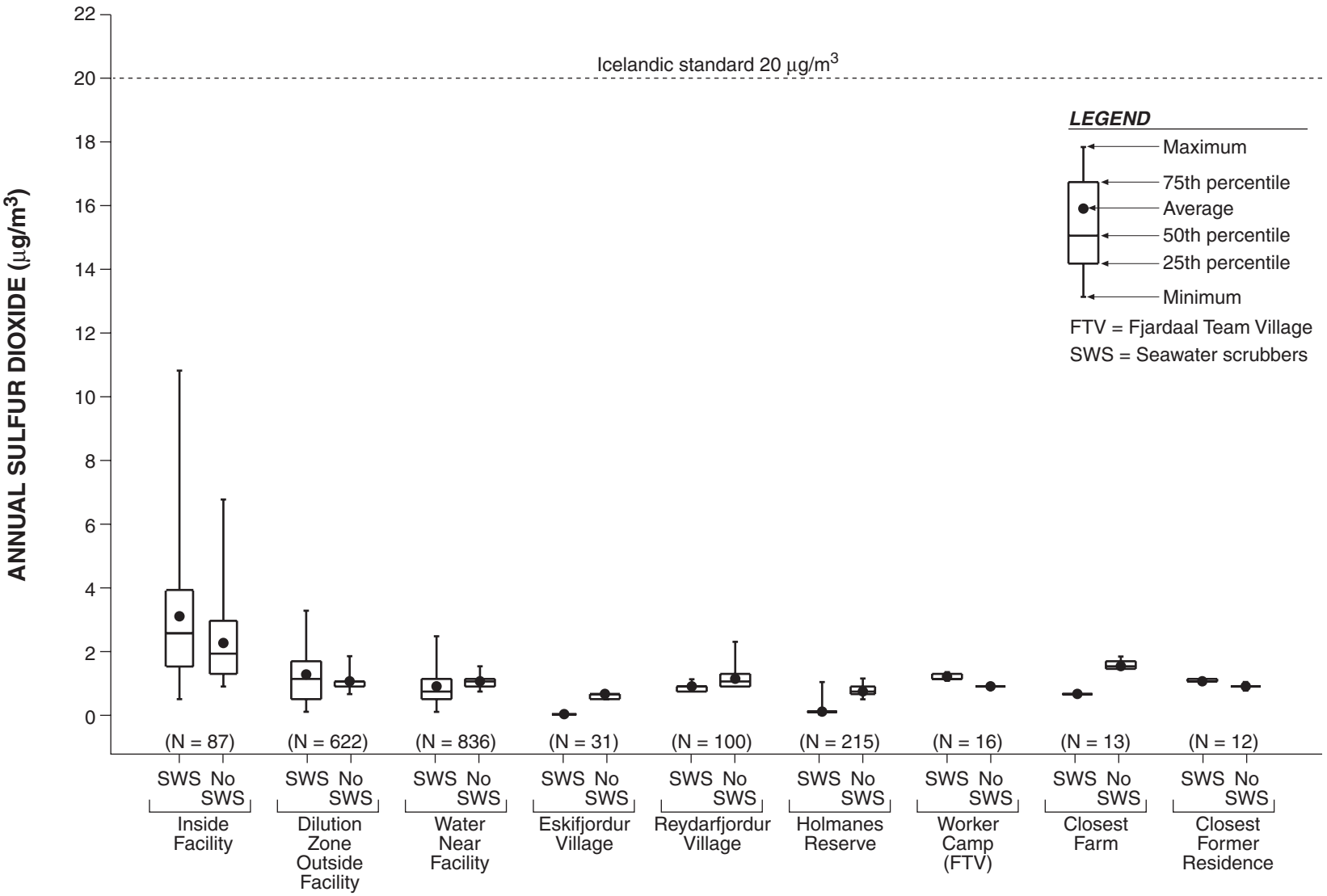

Figure 2-5. Boxplots showing the distribution of annual average ambient SO_2 air concentration estimates ($\mu g/m^3$) at individual human health receptor locations

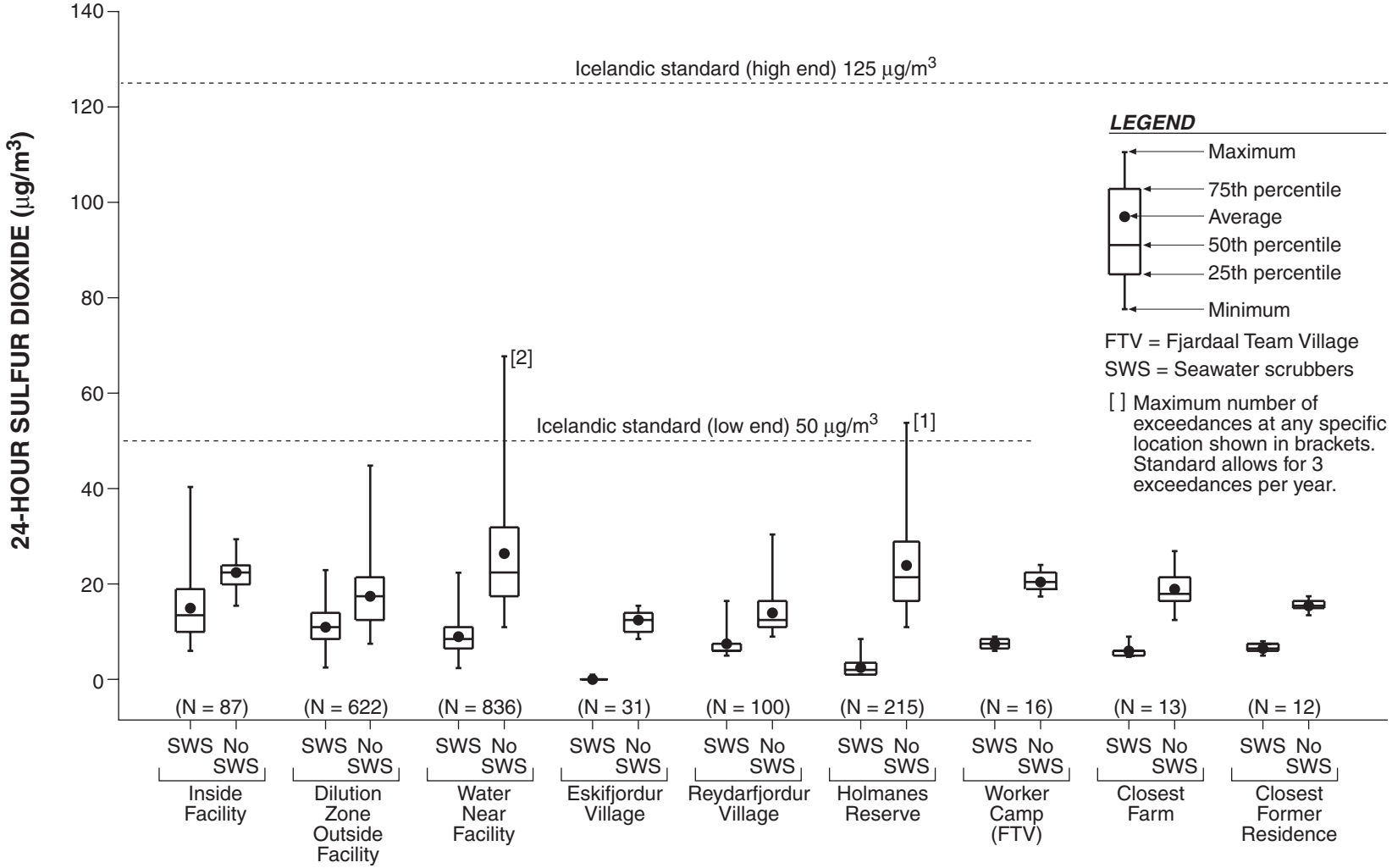

Figure 2-6. Boxplots showing the distribution of maximum 24-hour average SO_2 air concentration estimates (μg/m3) at individual human health receptor locations

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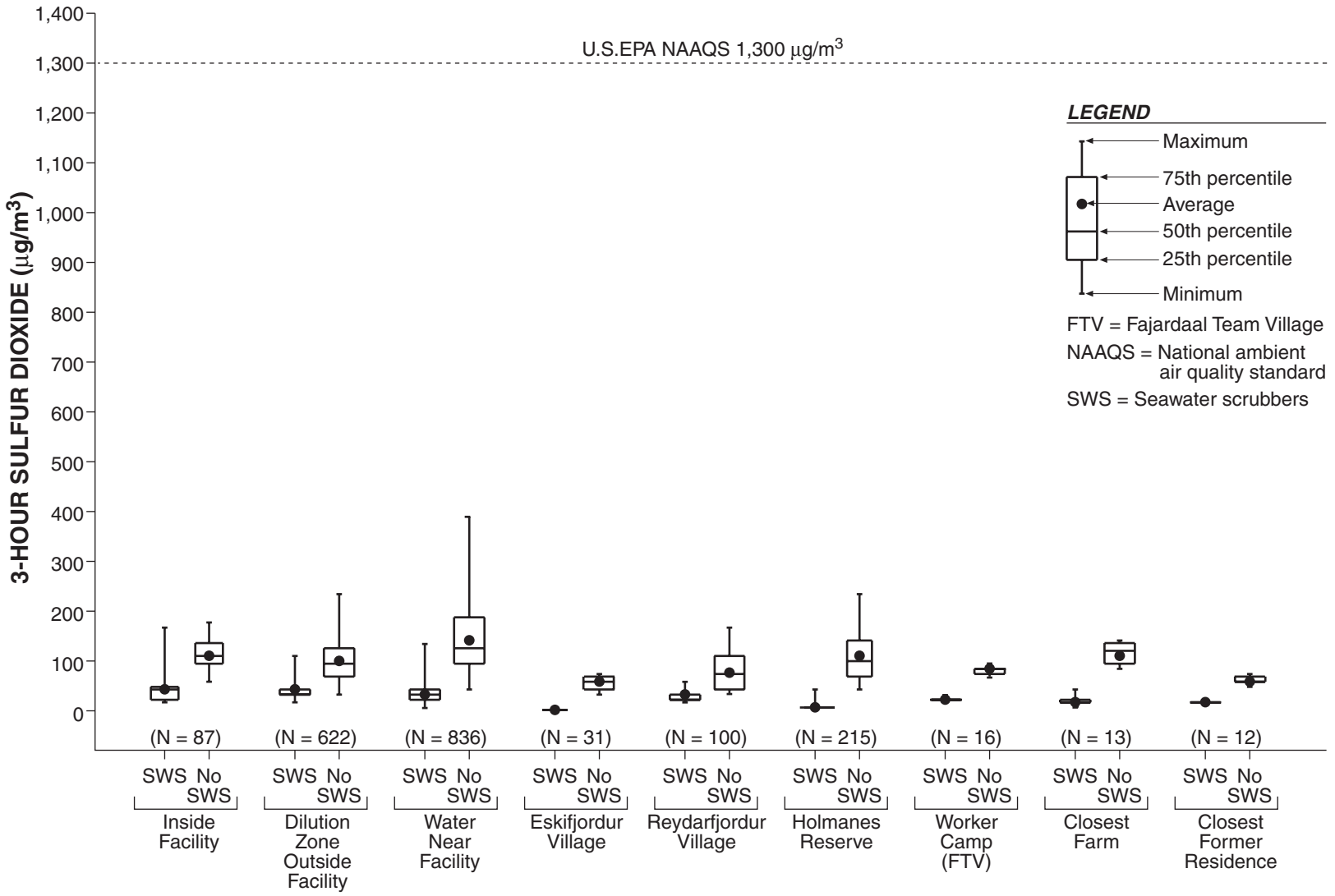

Figure 2-7. Boxplots showing the distribution of maximum 3-hour average SO_2 air concentration estimates (µg/m3) at individual human health receptor locations

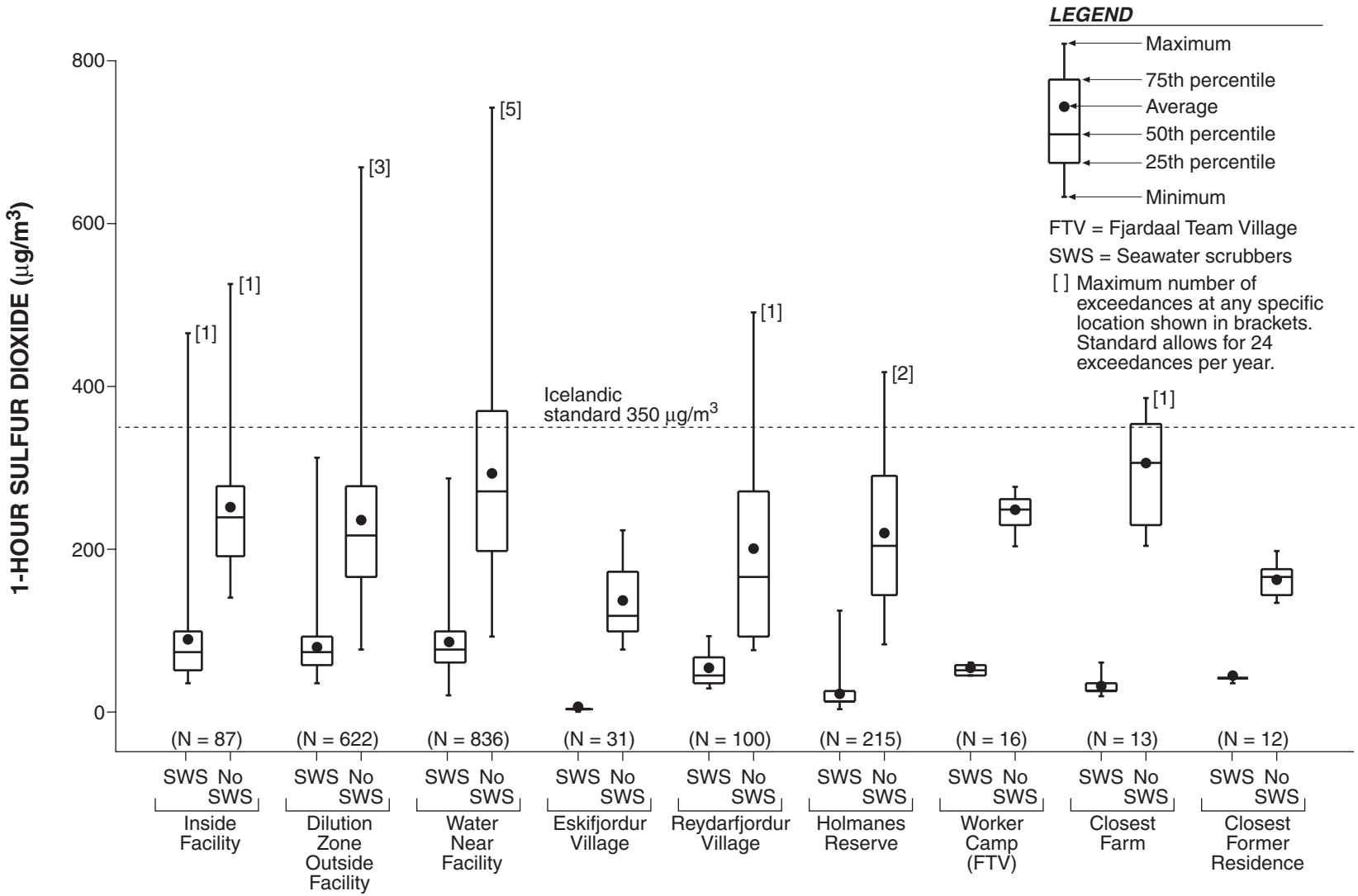

Figure 2-8. Boxplots showing the distribution of maximum 1-hour average SO_2 air concentration estimates ($\mu g/m^3$) at individual human health receptor locations

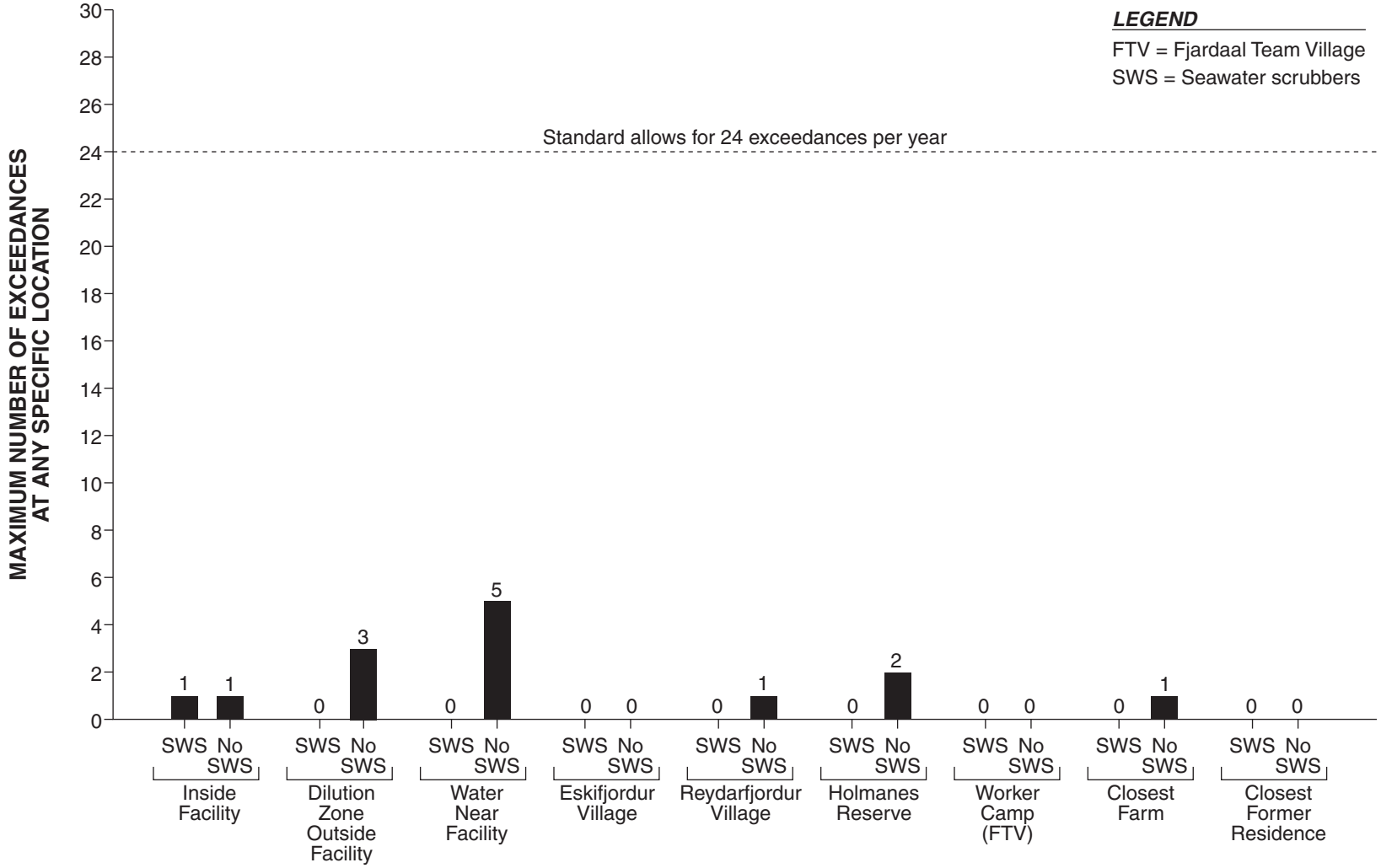

Figure 2-9. Maximum number of exceedances of the Icelandic 1-hour average SO_2 standard (350 $\mu g/m^3$) at any specific location

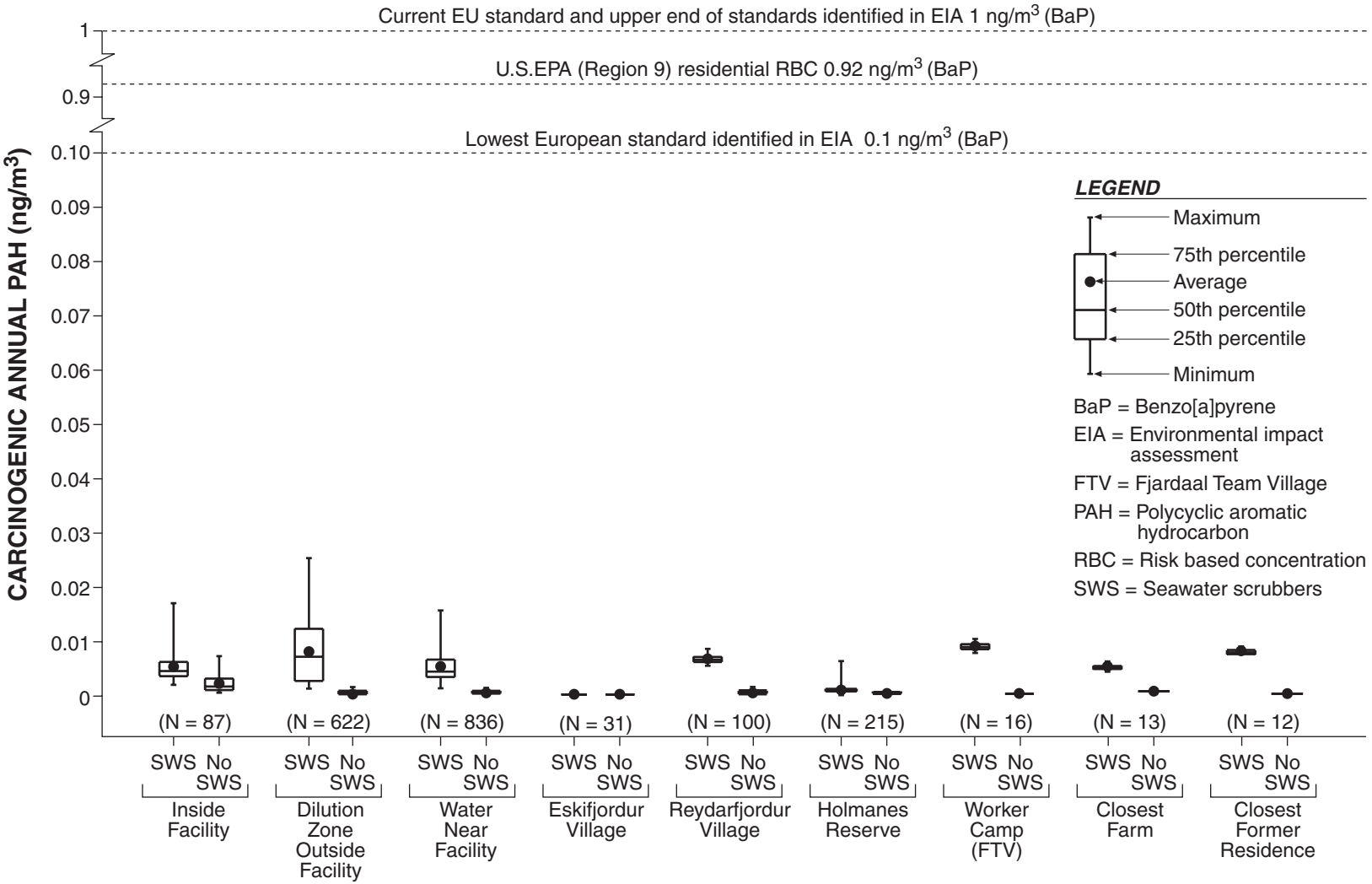

Figure 2-10. Boxplots showing the distribution of carcinogenic PAHs annual average air concentration estimates (ng/m^3) at individual human health receptor locations

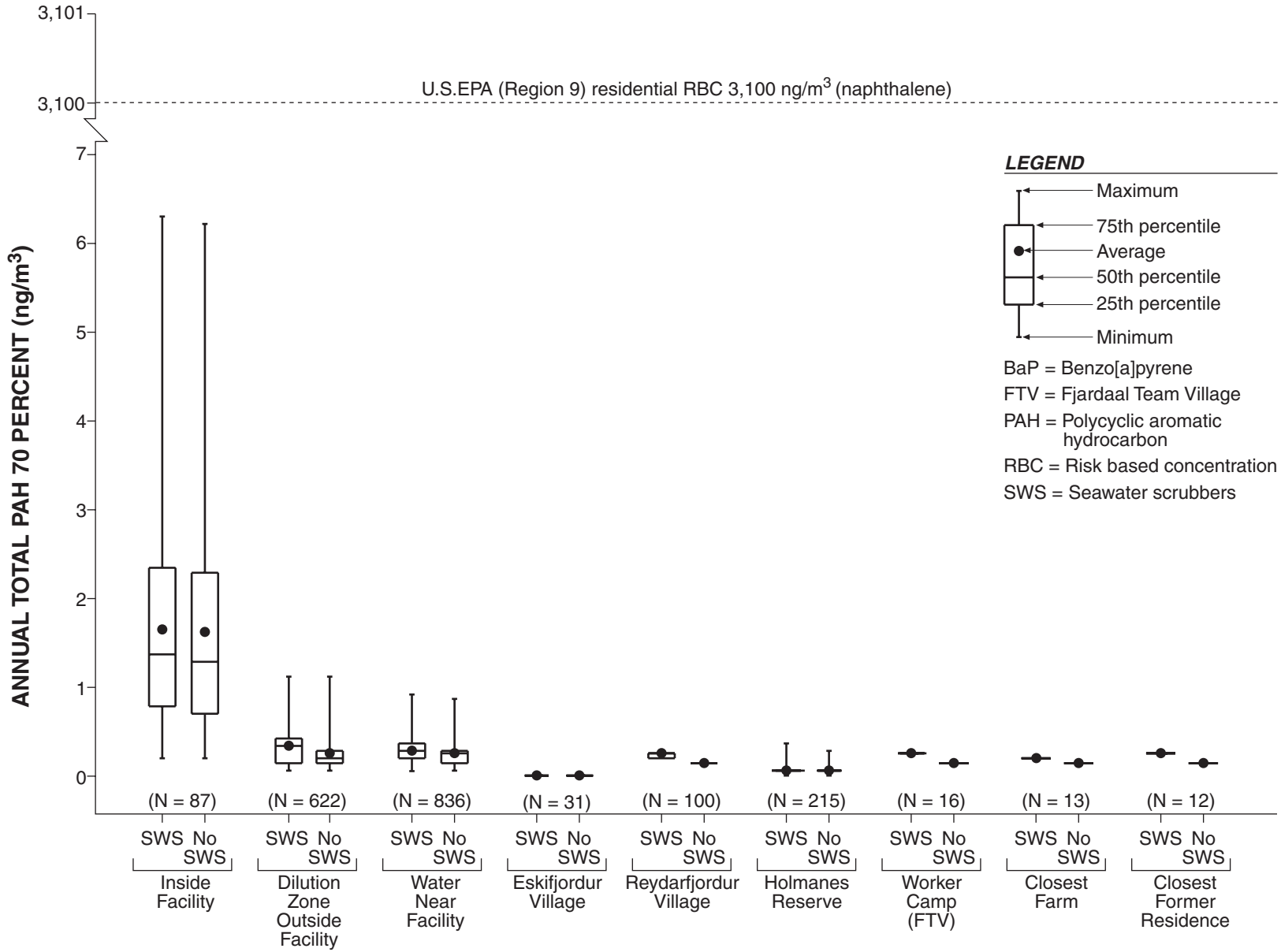

Figure 2-11. Boxplots showing the distribution of 70 percent total PAH annual average air concentration estimates (ng/m^3) at individual human health receptor locations (assuming naphthalene is 70 percent of total PAHs)

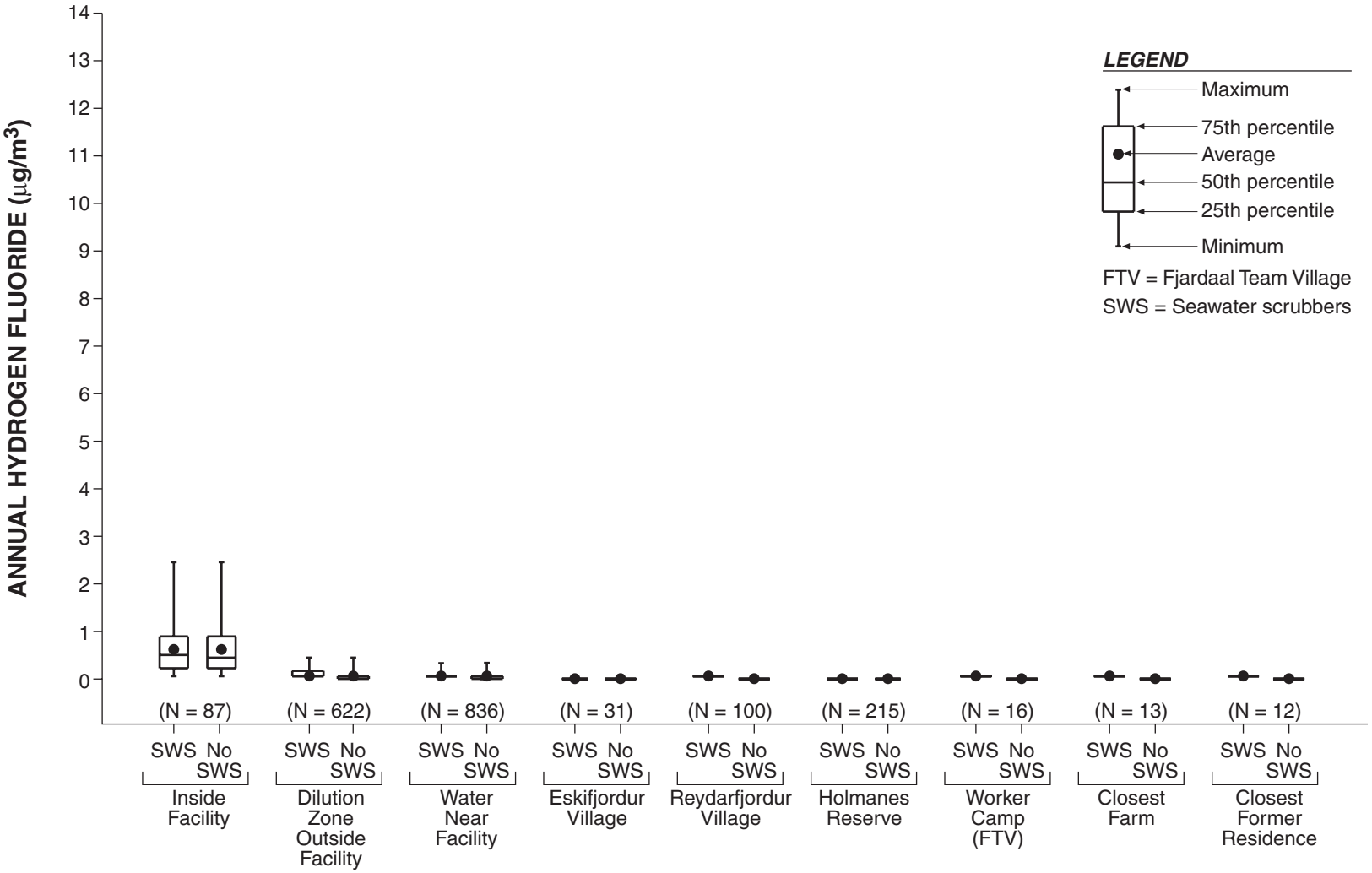

Figure 2-12. Boxplots showing the distribution of annual average ambient hydrogen fluoride air concentration estimates (µg/m^3) at individual human health receptor locations

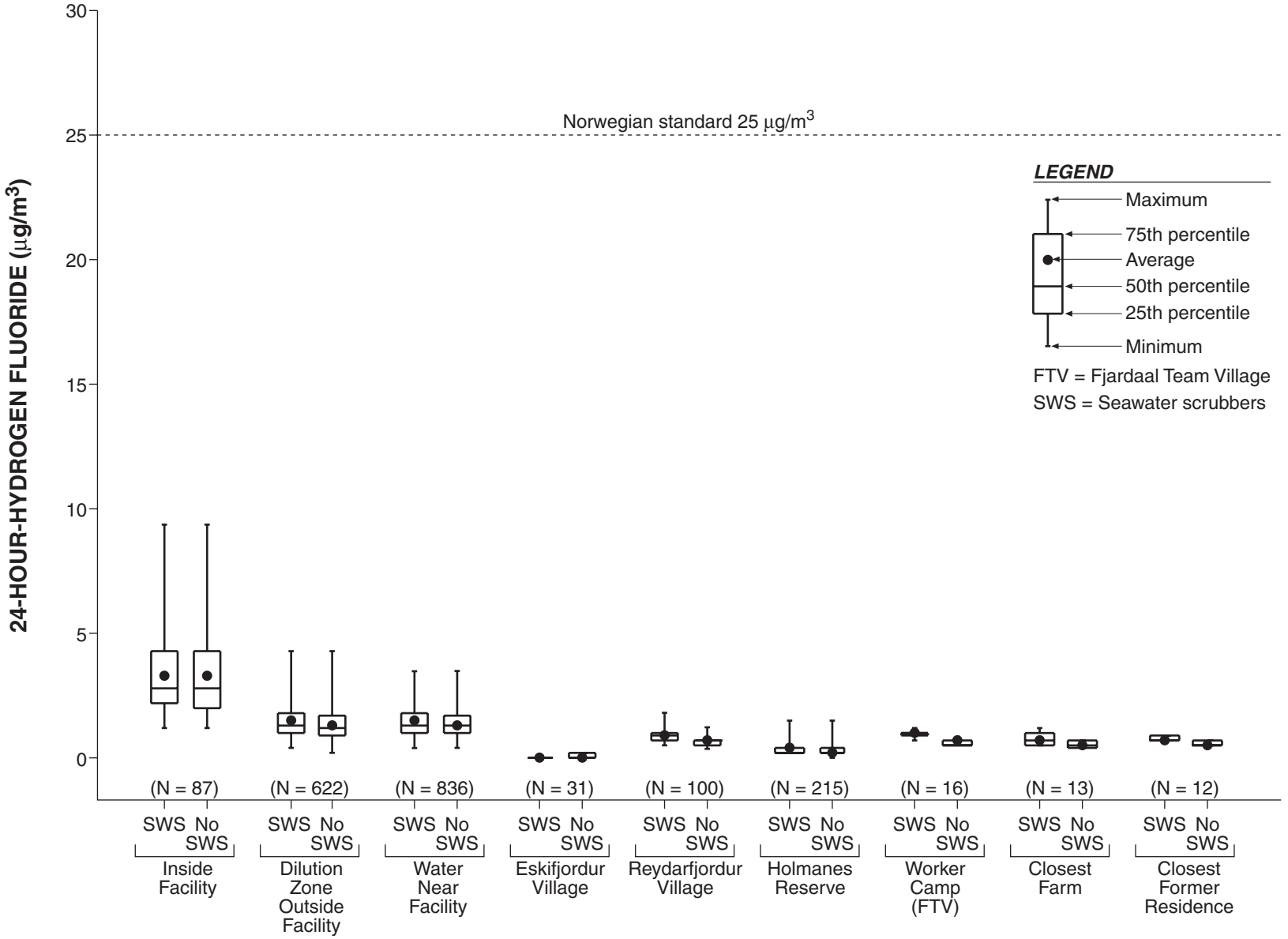

Figure 2-13. Boxplots showing the distribution of maximum 24-hour average ambient hydrogen fluoride air concentration estimates (µg/m3) at individual human health receptor locations

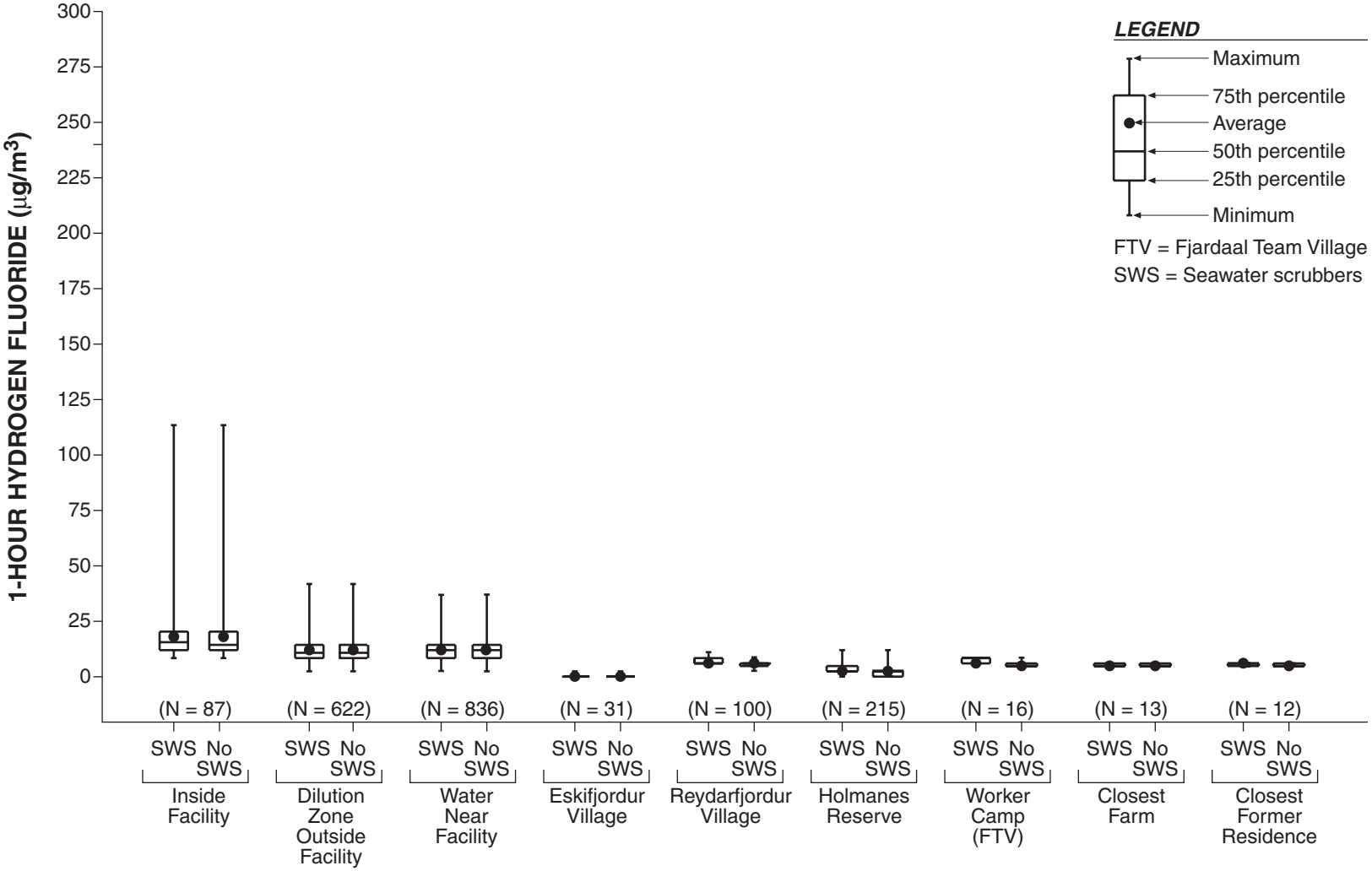

Figure 2-14. Boxplots showing the distribution of maximum 1-hour average ambient hydrogen fluoride air concentration estimates (µg/m3) at individual human health receptor locations

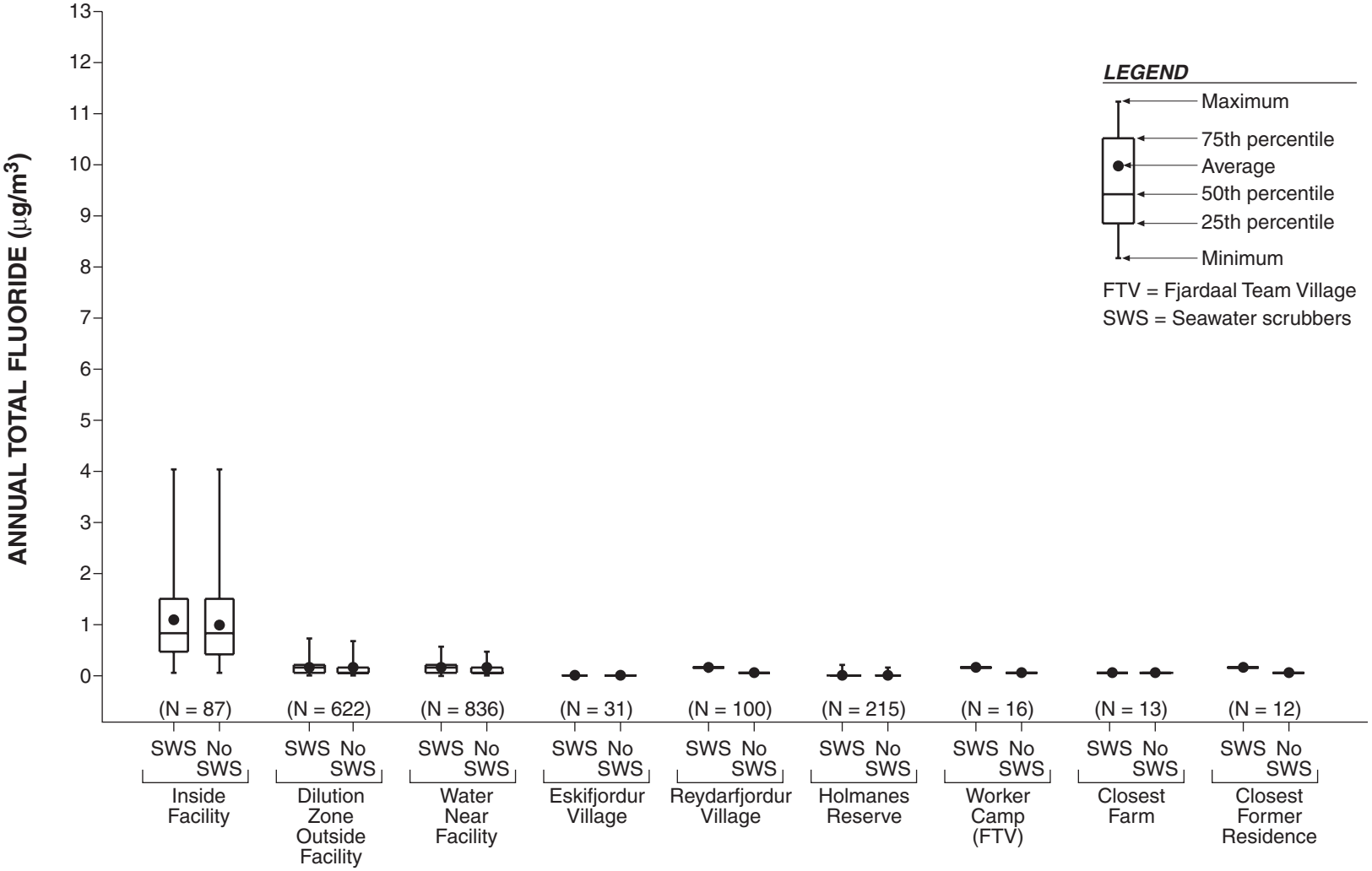

Figure 2-15. Boxplots showing the distribution of annual average ambient total fluoride air concentration estimates (µg/m^3) at individual human health receptor locations

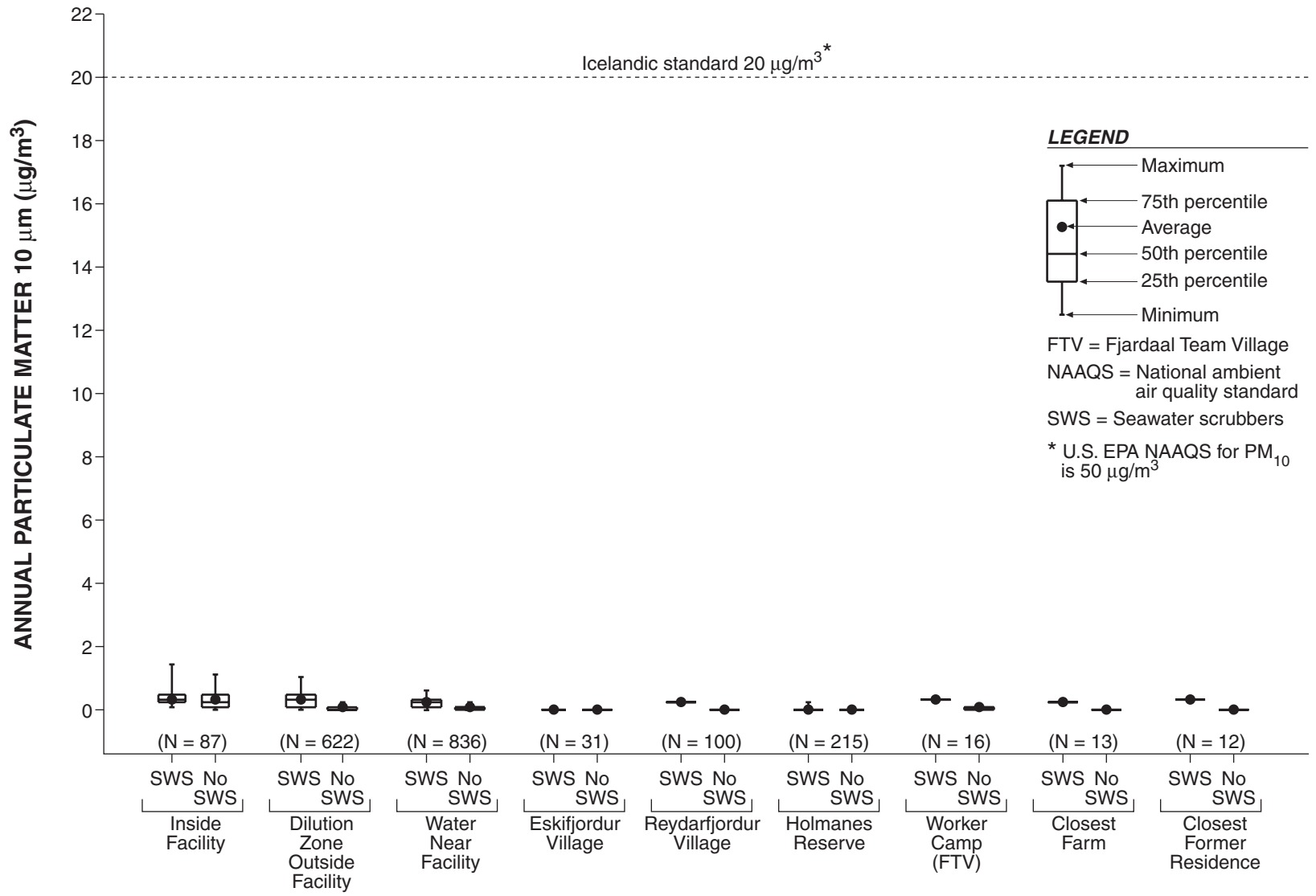

Figure 2-16. Boxplots showing the distribution of annual average ambient air concentration estimates for particulate matter (PM_{10}) (μg/m3) at individual human health receptor locations

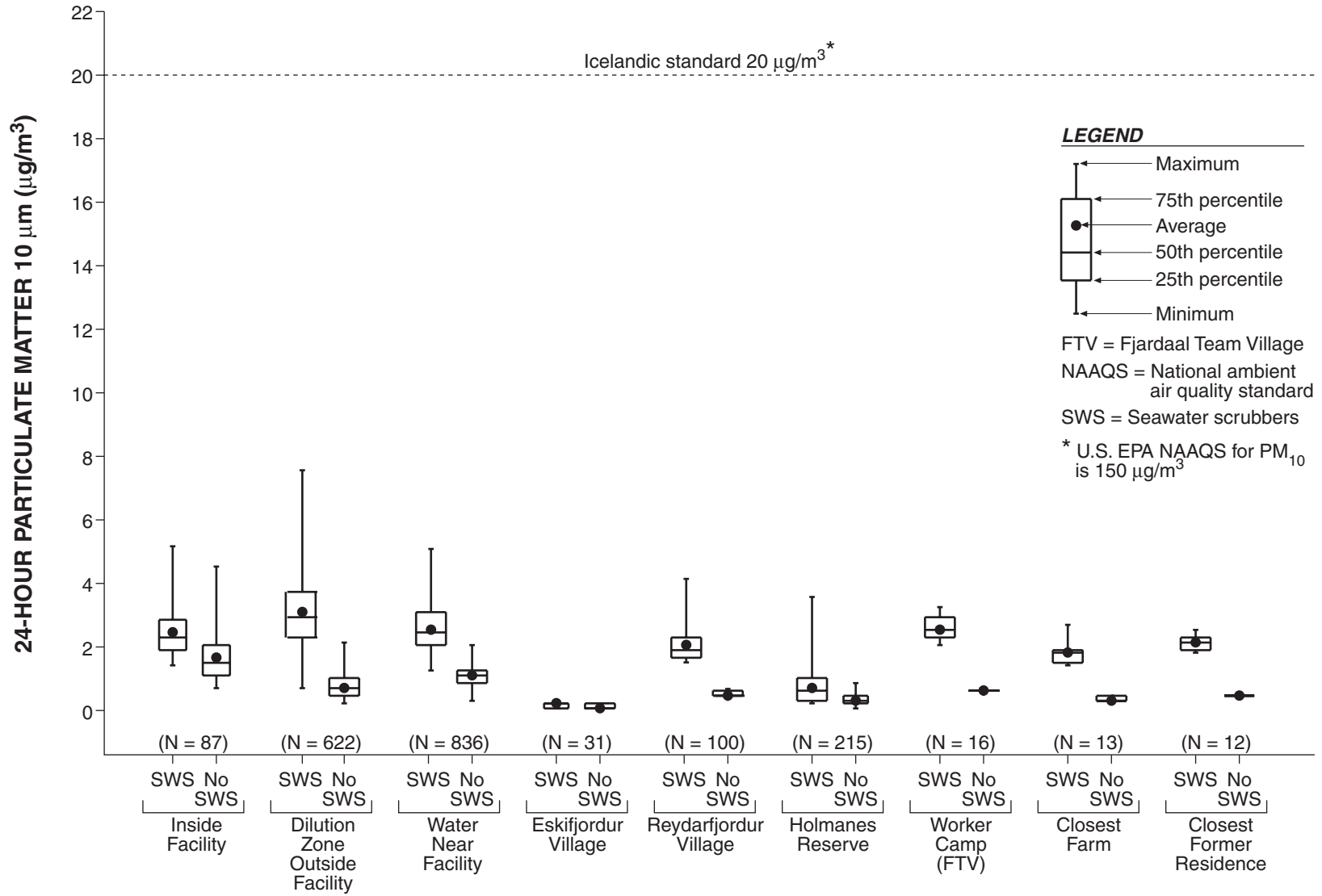

Figure 2-17. Boxplots showing the distribution of maximum 24-hour average ambient air concentration estimates for particulate matter (PM_{10}) (μg/m3) at individual human health receptor locations

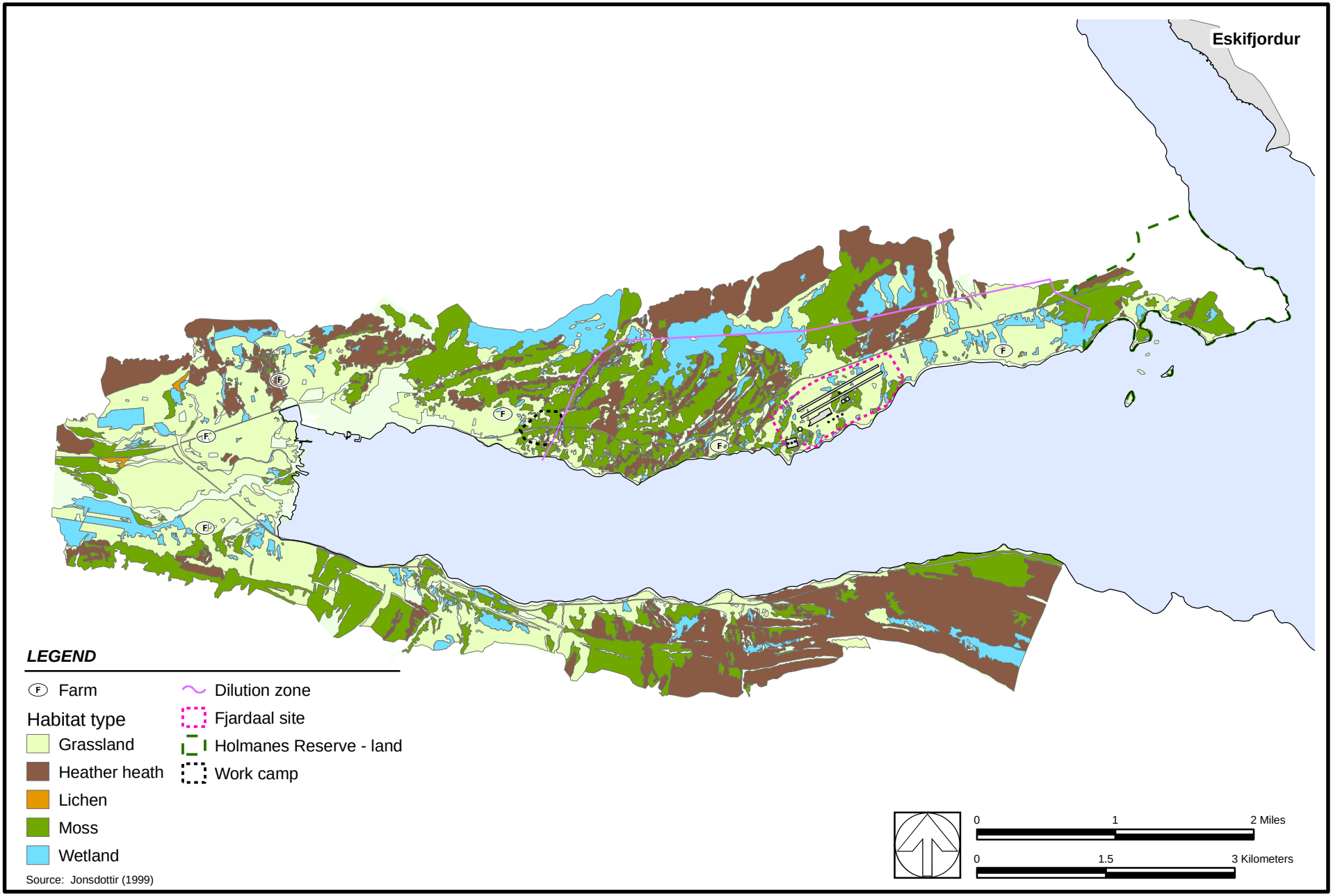

Figure 2-18. Smelter area vegetation

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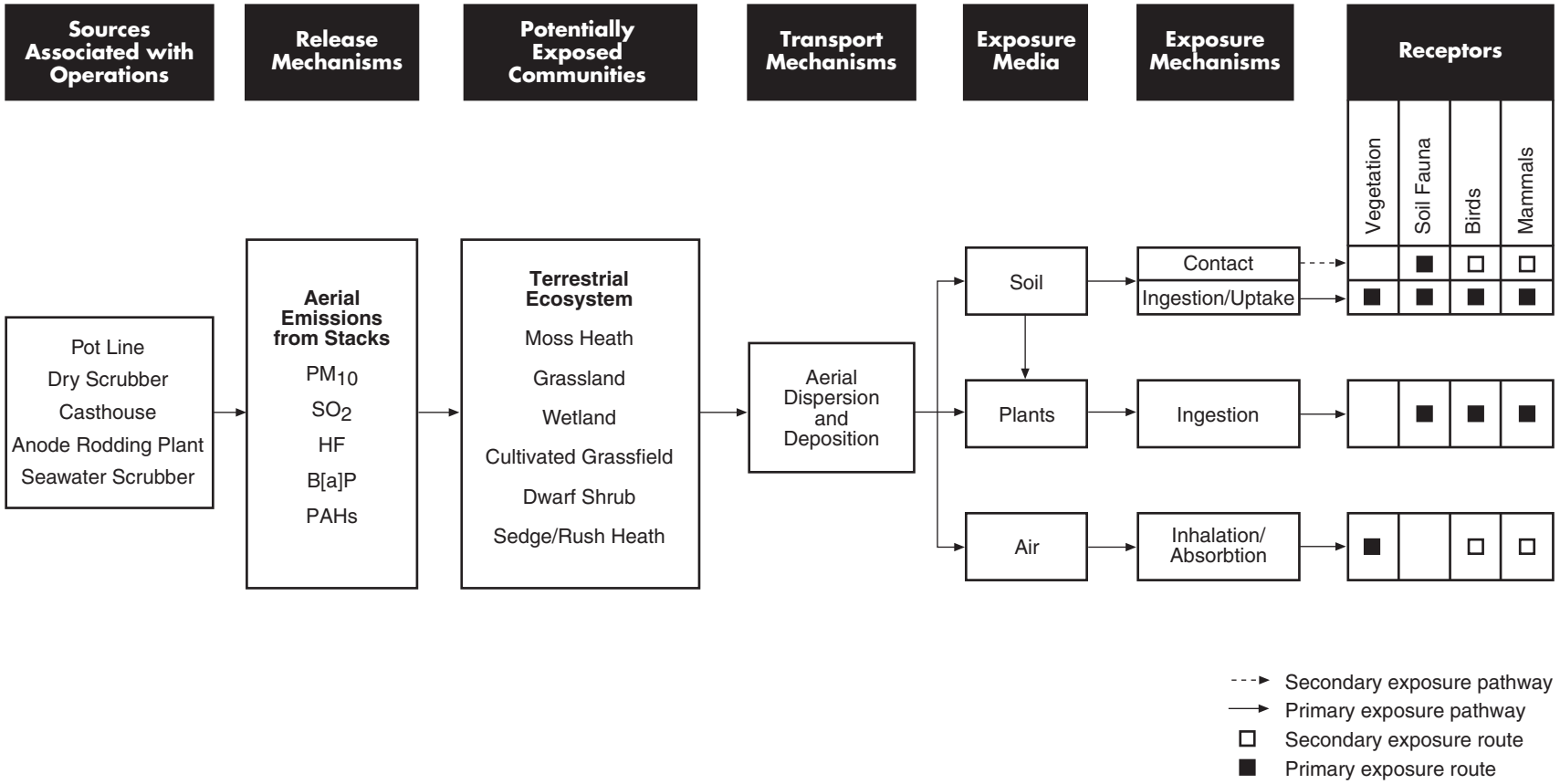

Figure 2-19. Conceptual site model for the Fjardaal screening ecological risk assessment

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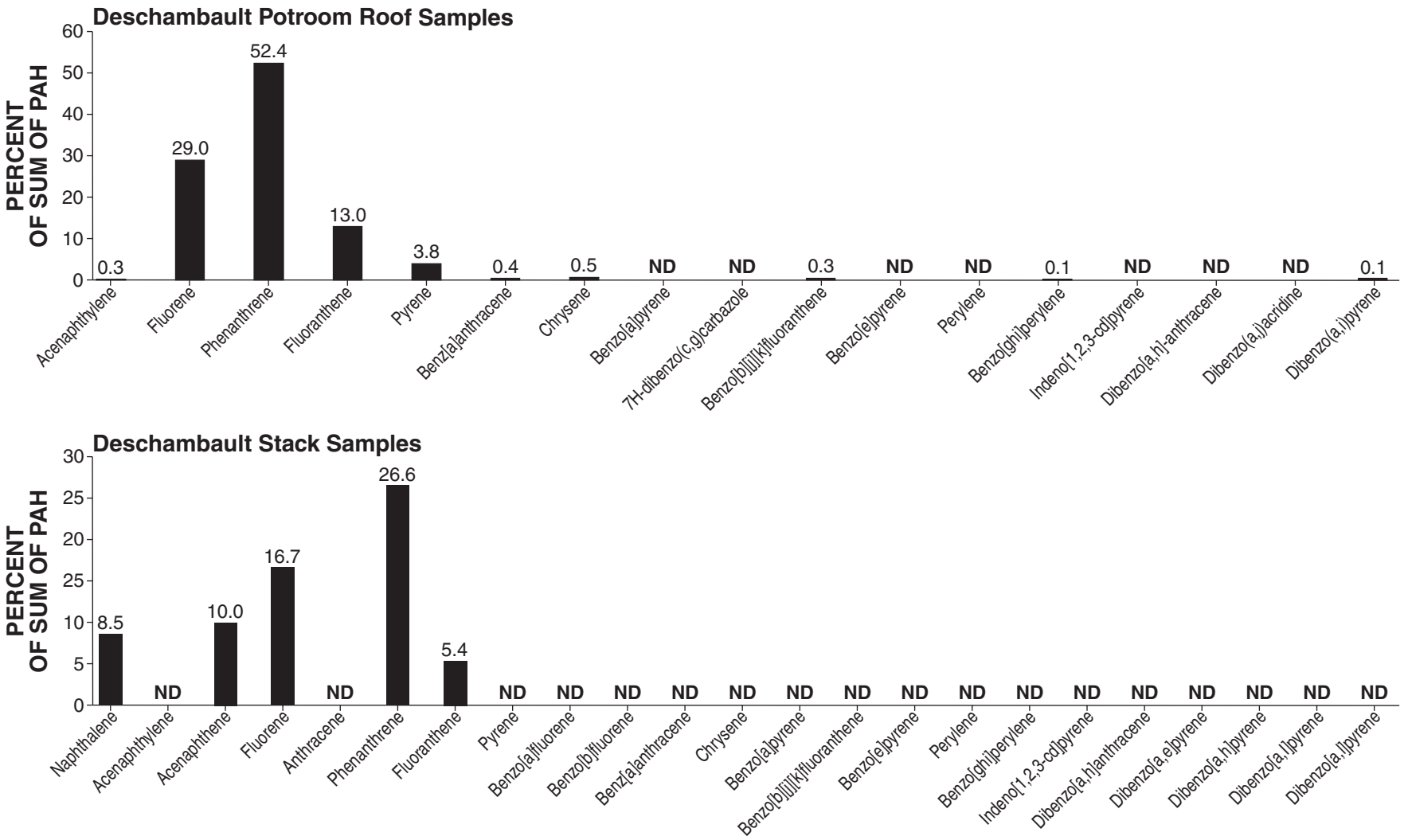
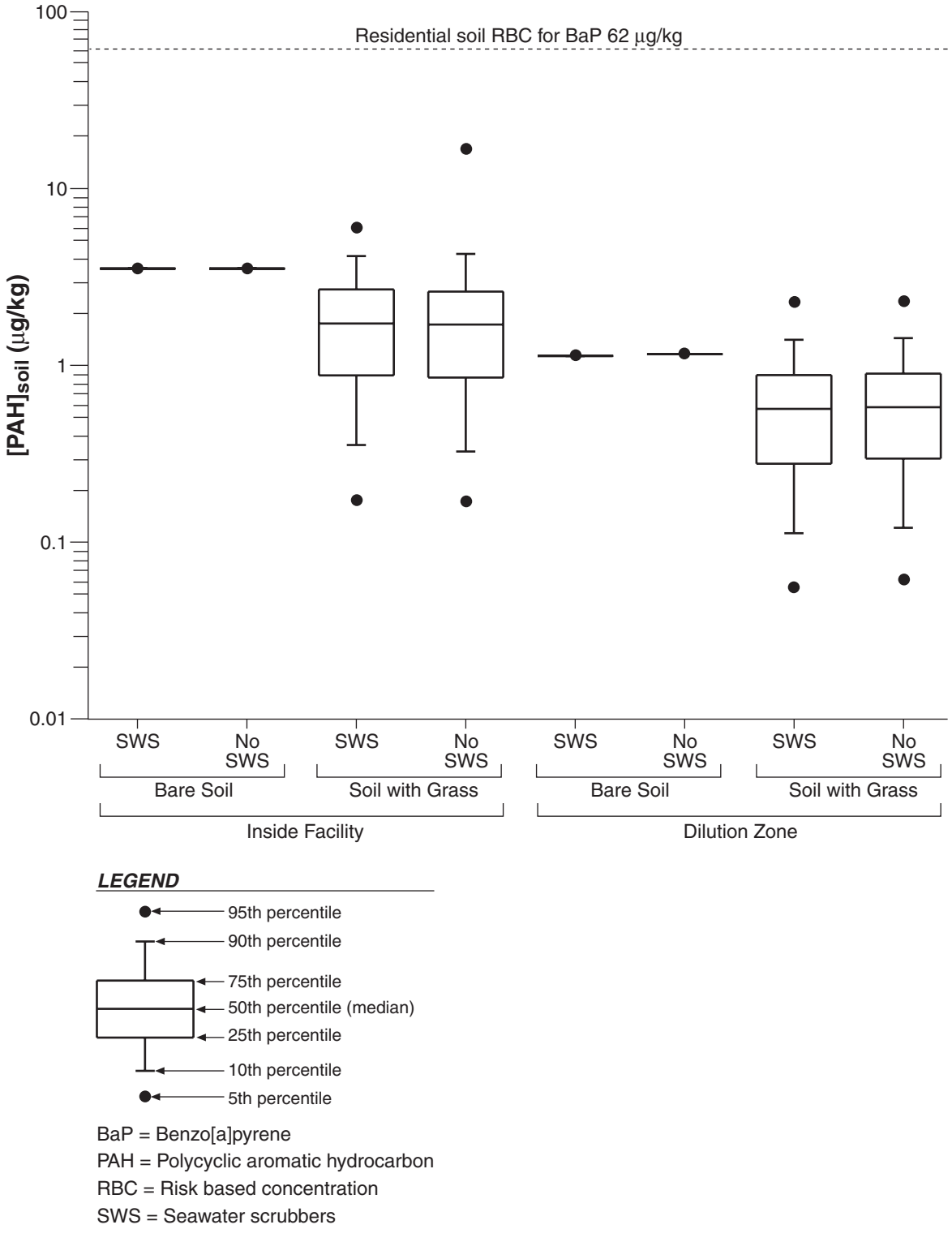


Note: Total PAHs are sums of the gaseous and particulate PAHs. Undetected results in samples 1 and 2 are reported at the detection limit but averaged at half the detection limit.

ND = Not detected

PAH = Polycyclic aromatic hydrocarbon

Figure 3-1. Percent composition for total PAHs (gaseous plus particulate) in Deschambault potroom roof samples and stack samples



BaP = Benzo[a]pyrene
PAH = Polycyclic aromatic hydrocarbon
RBC = Risk based concentration
SWS = Seawater scrubbers

Figure 3-2. Maximum annual soil PAH concentration estimates (µg/kg) inside the facility and in the dilution zone modeled as bare soil or grass with and without seawater scrubbers

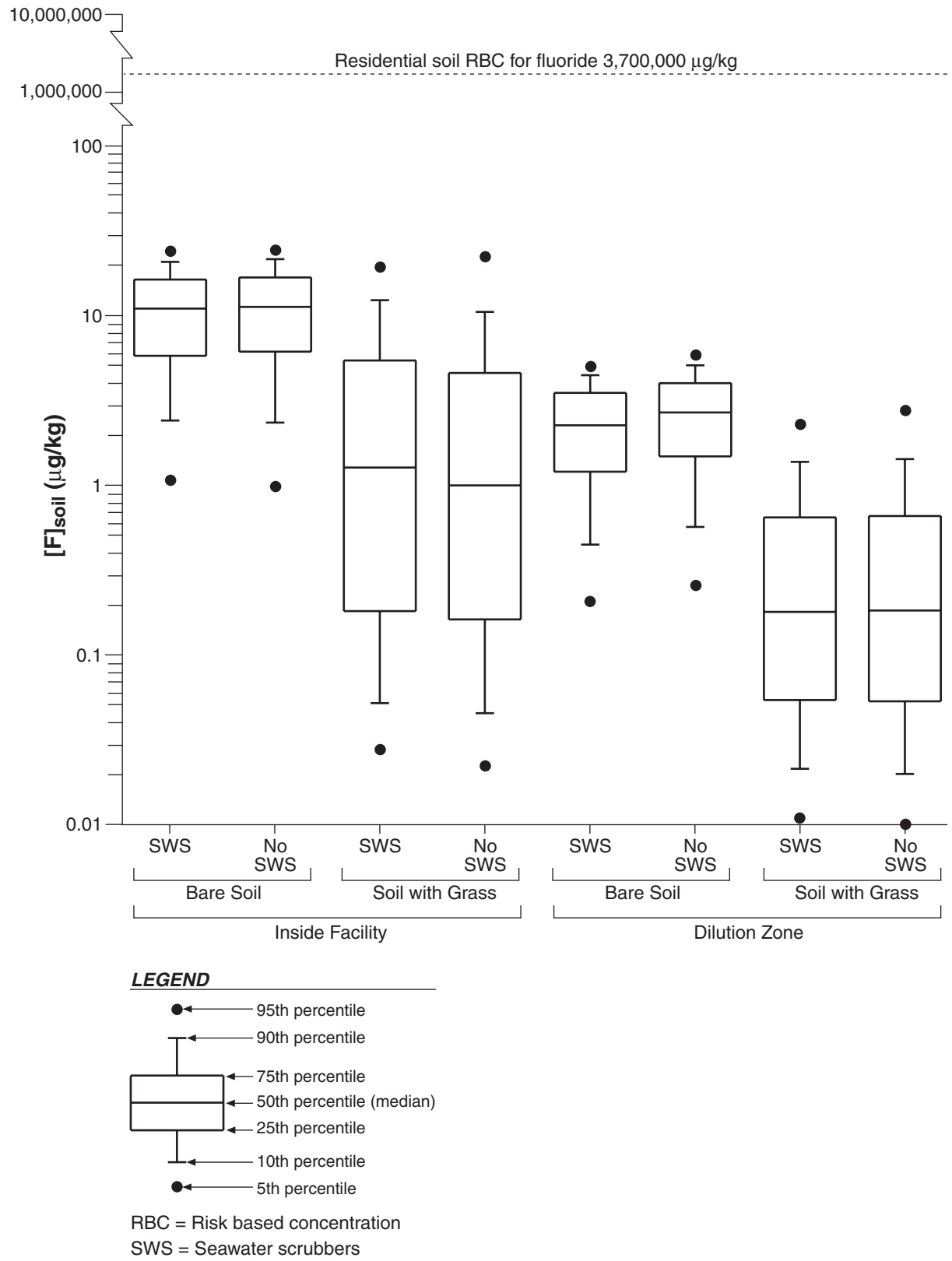


Figure 3-3. Maximum annual soil fluoride concentration estimates (µg/kg) inside the facility and in the dilution zone modeled as bare soil or grass with and without seawater scrubbers

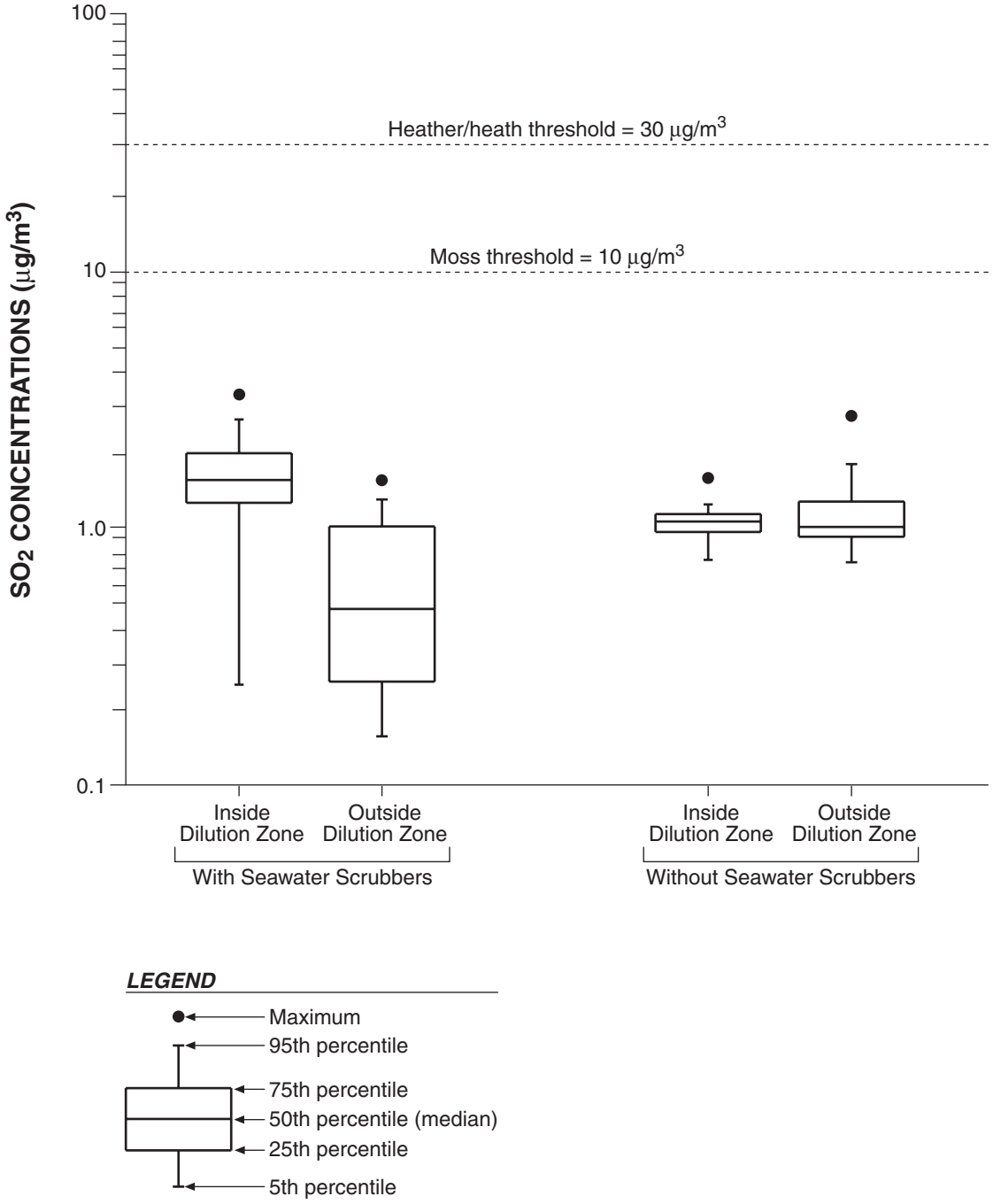


Figure 4-1. Modeled sulfur dioxide concentrations with and without seawater scrubbers, compared to toxicity thresholds for sensitive plant communities (fine grid data)

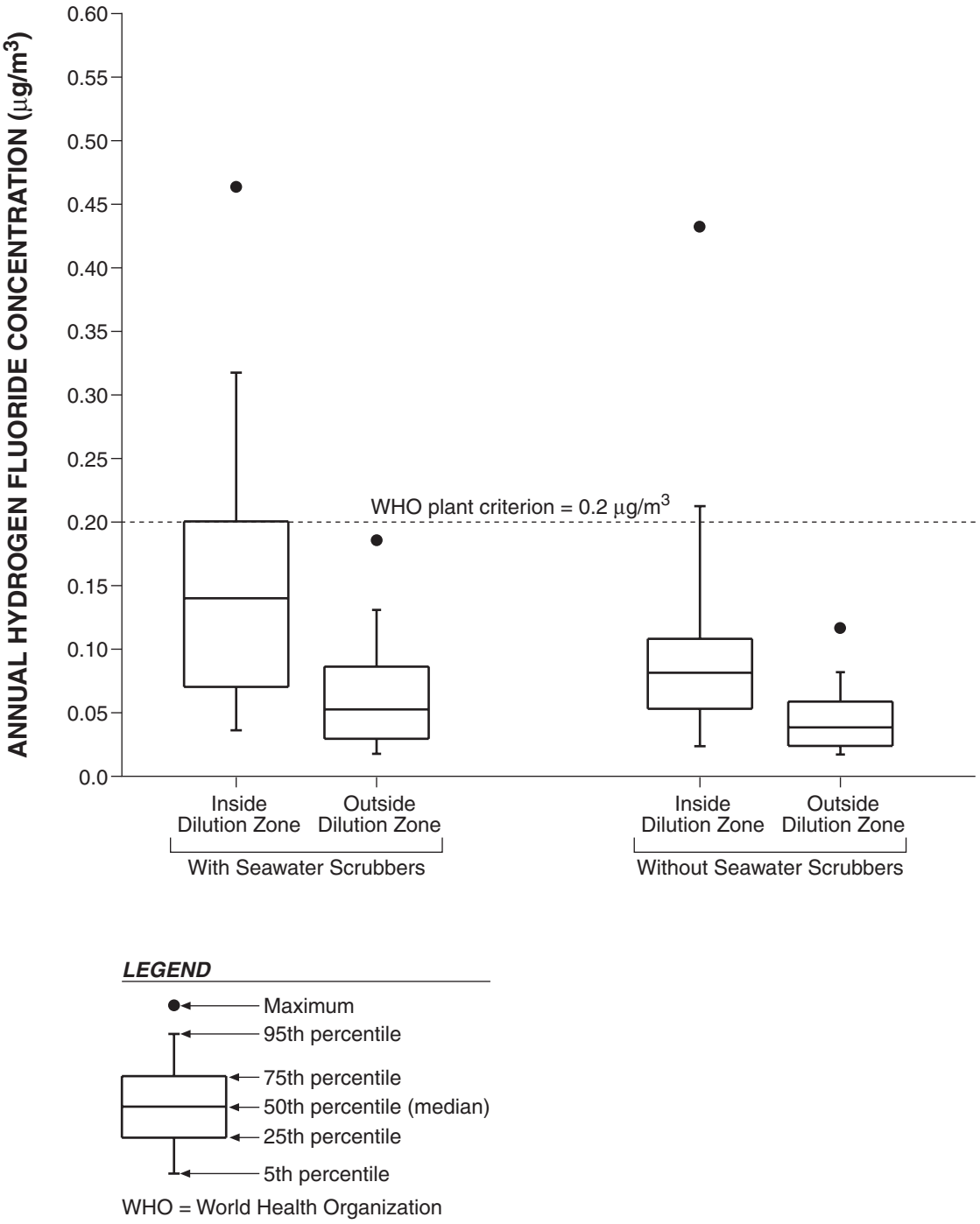


Figure 4-2. Distribution of modeled annual hydrogen fluoride air concentration data with and without seawater scrubbers, compared to the WHO criterion for sensitive plants (fine grid data)

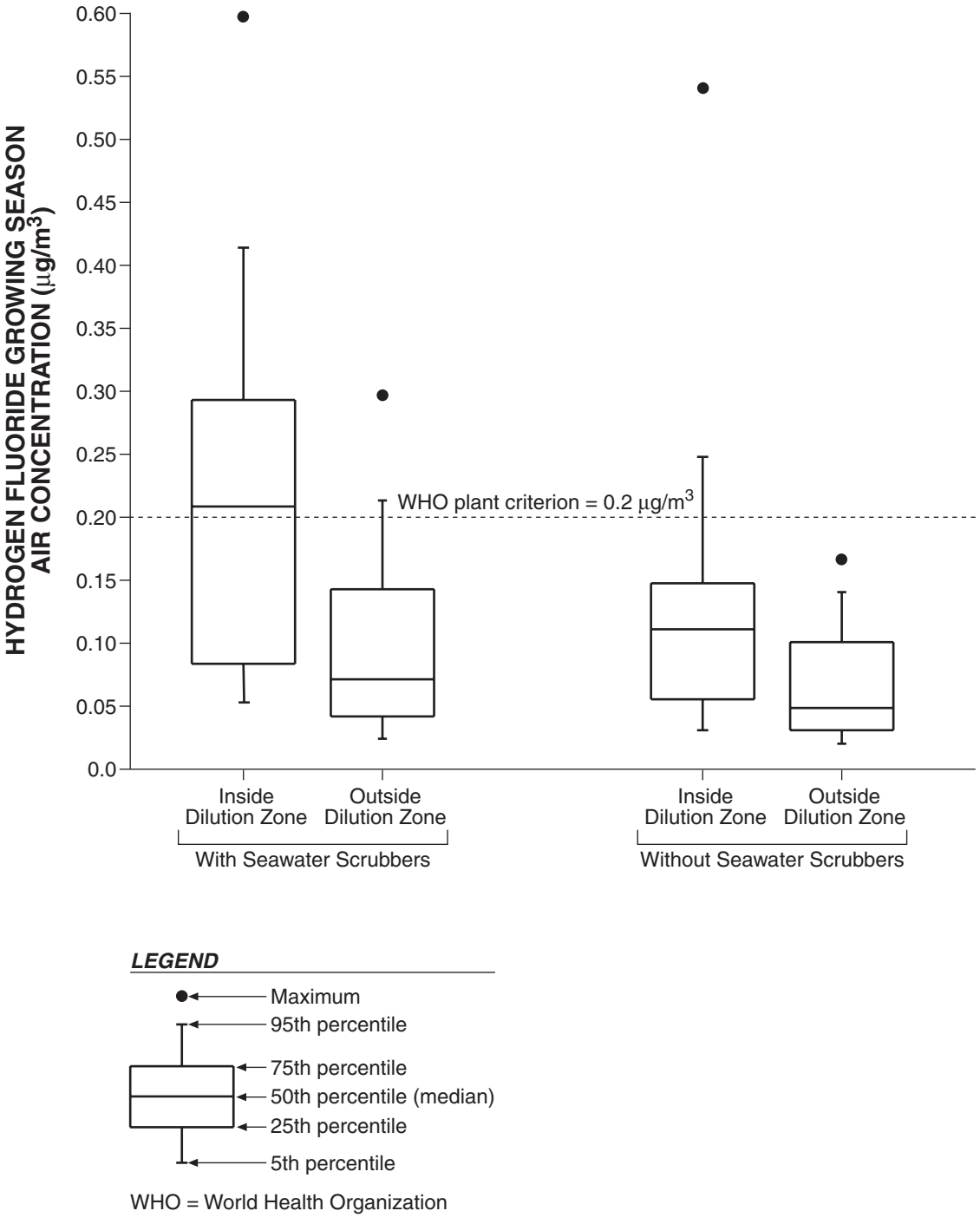


Figure 4-3. Distribution of modeled hydrogen fluoride growing season air concentration data (April 1 through September 30) with and without seawater scrubbers, compared to the WHO criterion for sensitive plants (fine grid data)

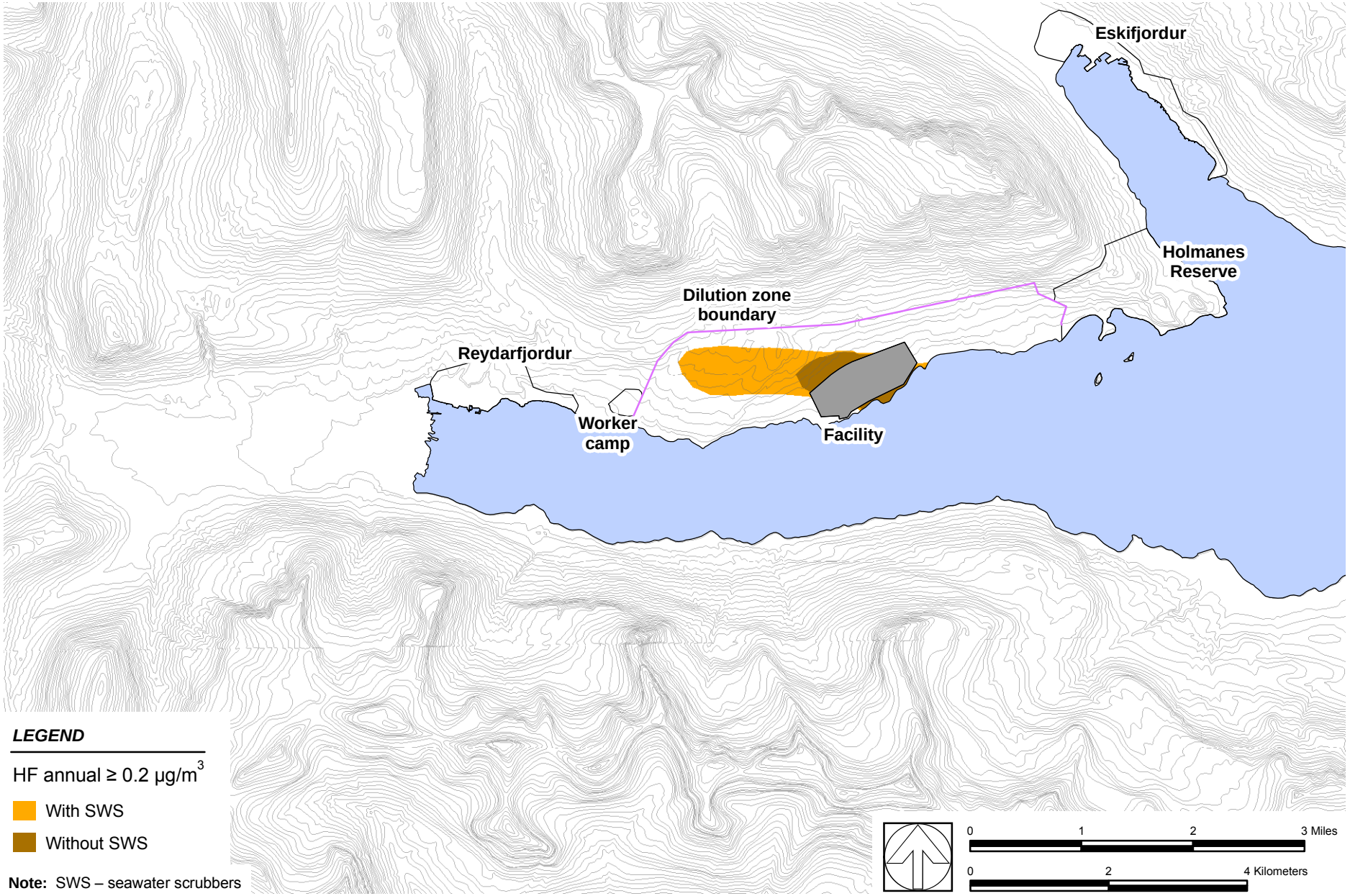


Figure 4-4. Annual emissions of hydrogen fluoride exceeding sensitive plant community criterion equivalent to 0.2 µg/m^3

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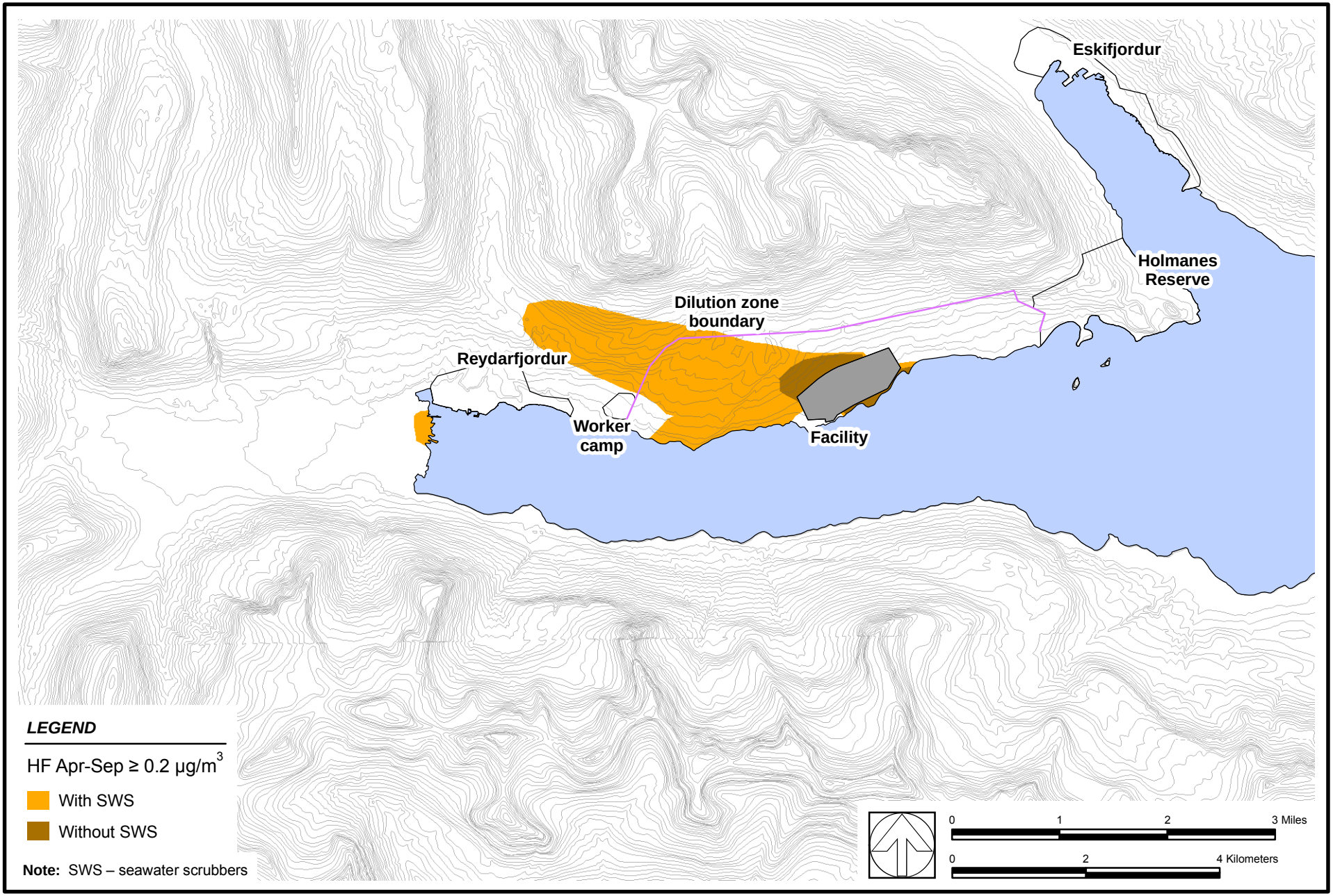

Figure 4-5. Growing season (April through September) emissions of hydrogen fluoride exceeding sensitive plant community criterion equivalent to 0.2 µg/m3

Exponent®

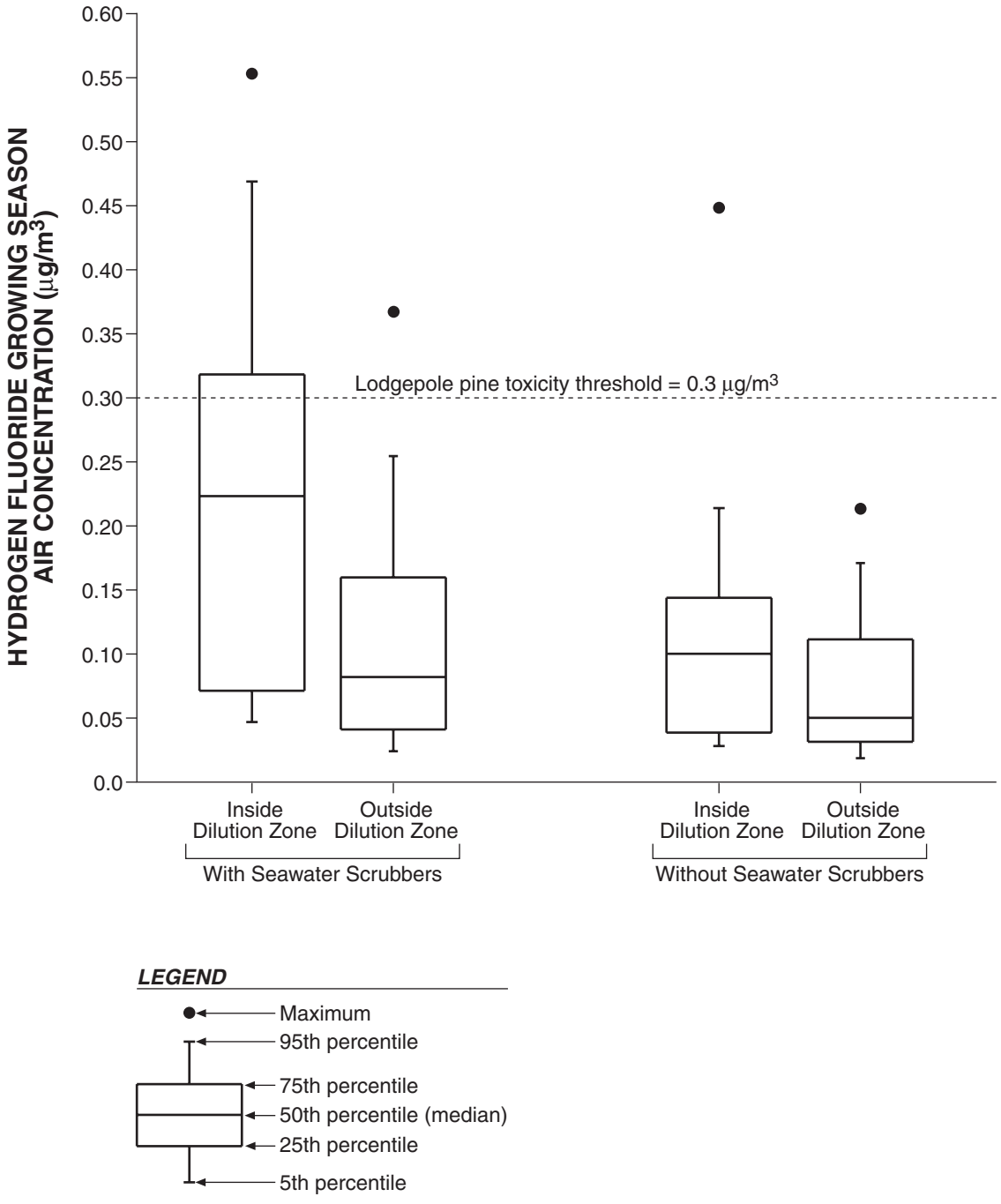

Figure 4-6. Distribution of modeled hydrogen fluoride air concentration data during lodgepole pine needle expansion (June 1 through August 30) with and without seawater scrubbers, compared to the toxicity threshold for lodgepole pine (fine grid data)

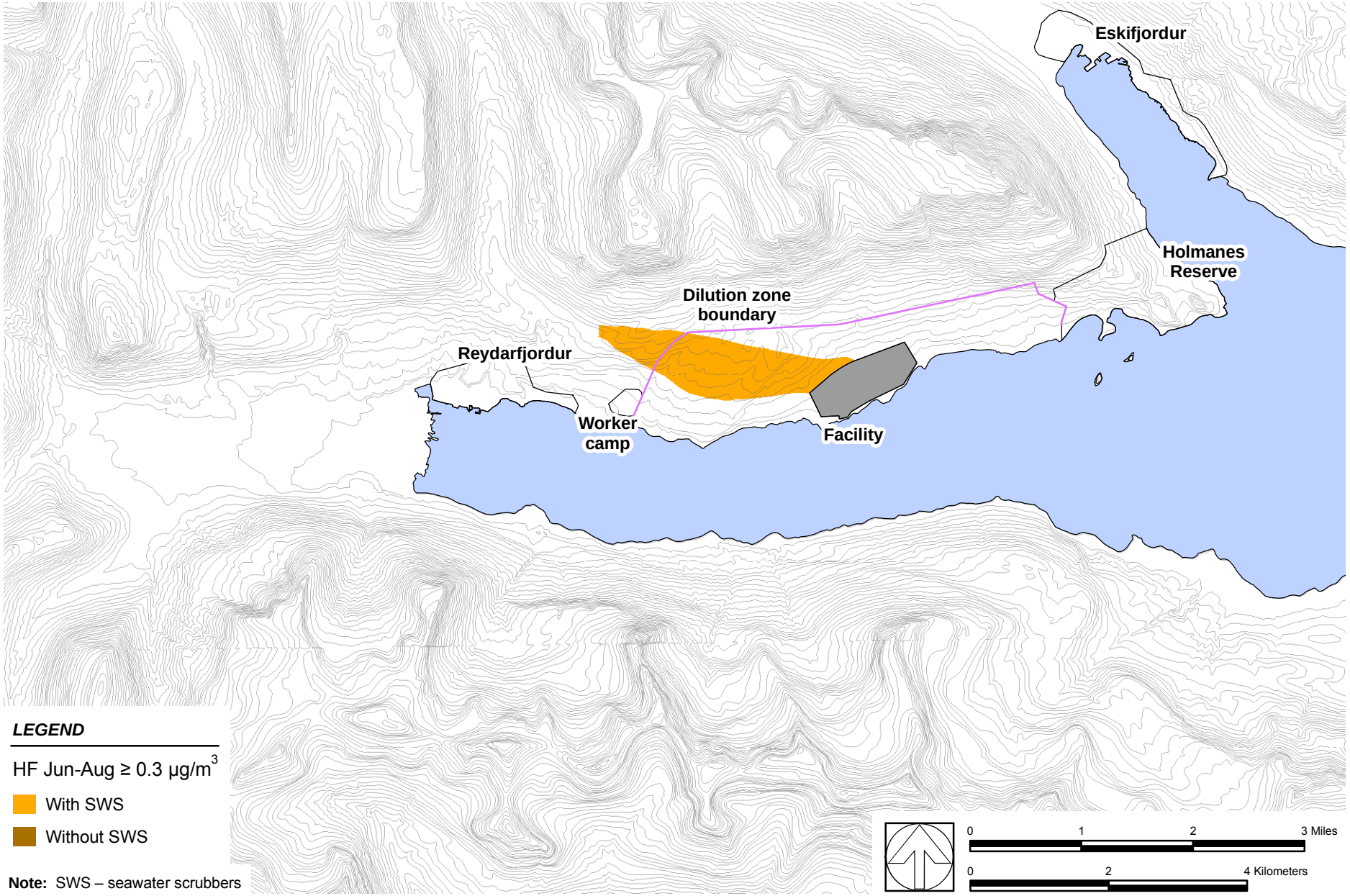

Figure 4-7. Emissions of hydrogen fluoride during lodgepole pine needle expansion (June through August) exceeding lodgepole pine criterion equivalent to 0.3 µg/m^3

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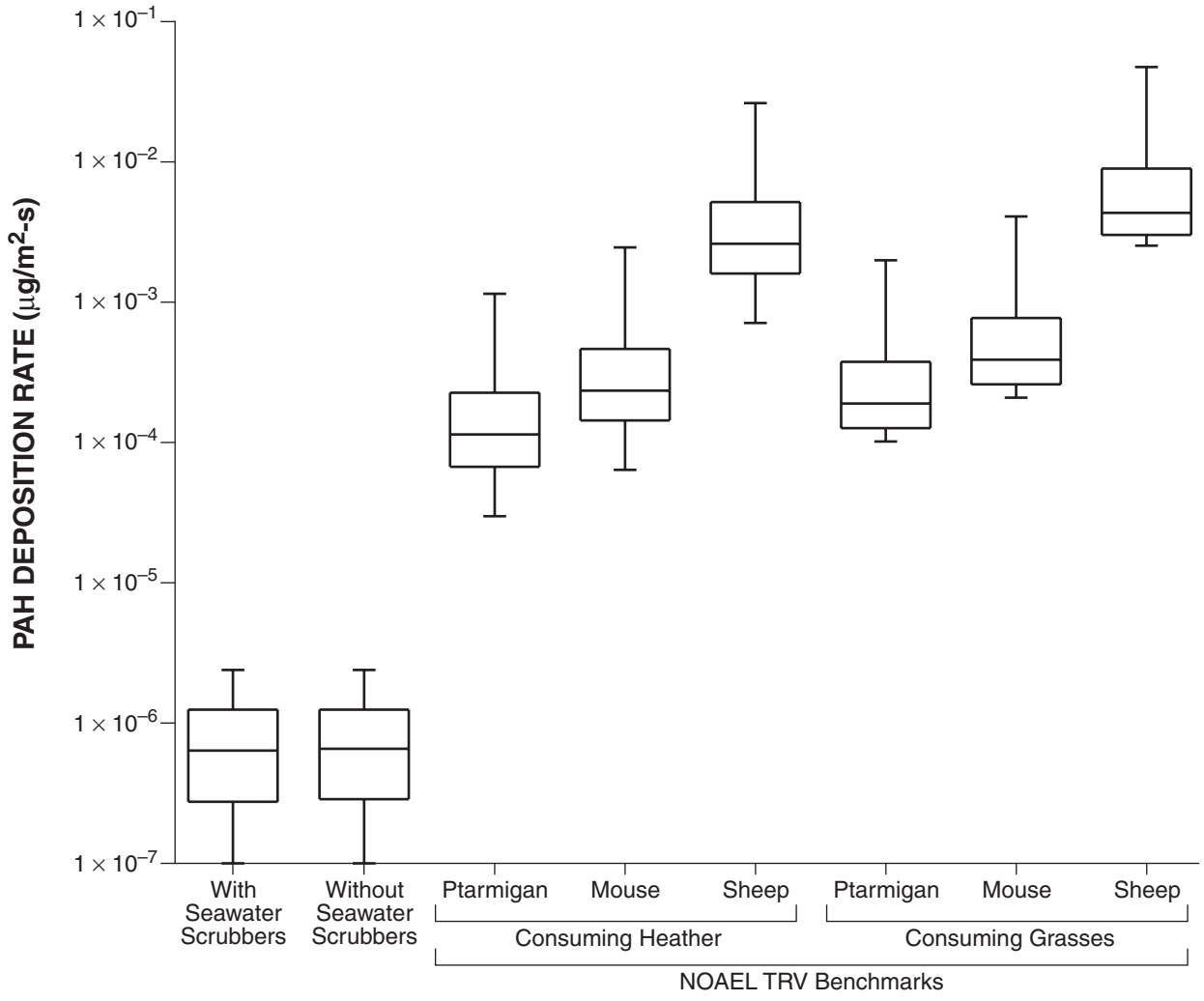
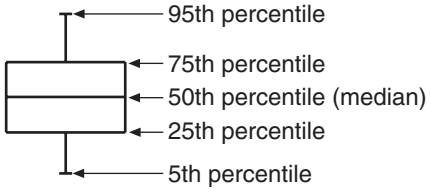


LEGEND



PAH = Polycyclic aromatic hydrocarbons

Figure 4-8. Modeled PAH air deposition rates with and without seawater scrubbers compared to NOAEL TRV benchmarks for ptarmigan, wood mouse, and sheep receptors, consuming either heather or grasses

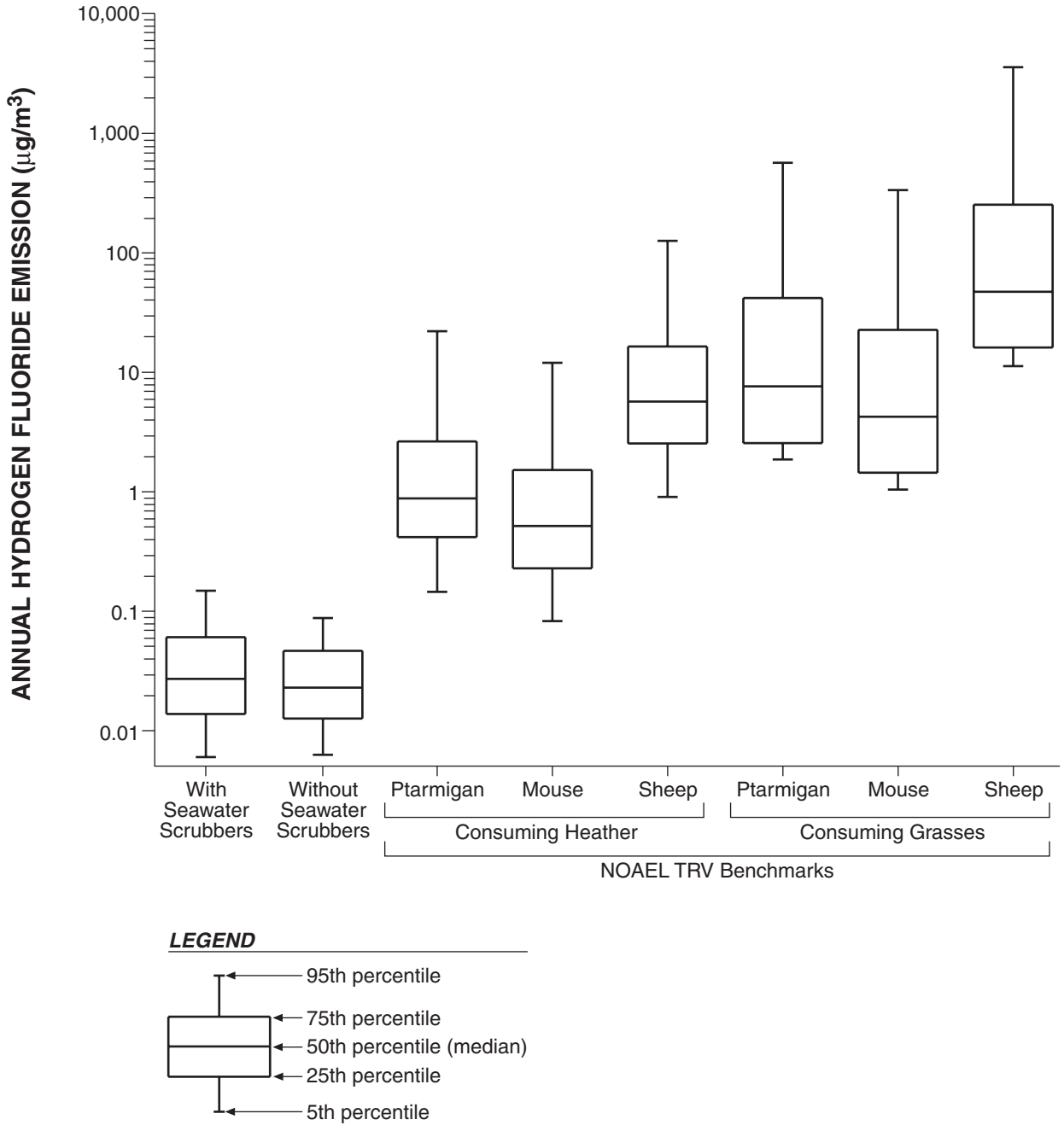


Figure 4-9. Modeled hydrogen fluoride concentrations with and without seawater scrubbers compared to NOAEL TRV benchmarks for ptarmigan, wood mouse, and sheep receptors, consuming either heather or grasses

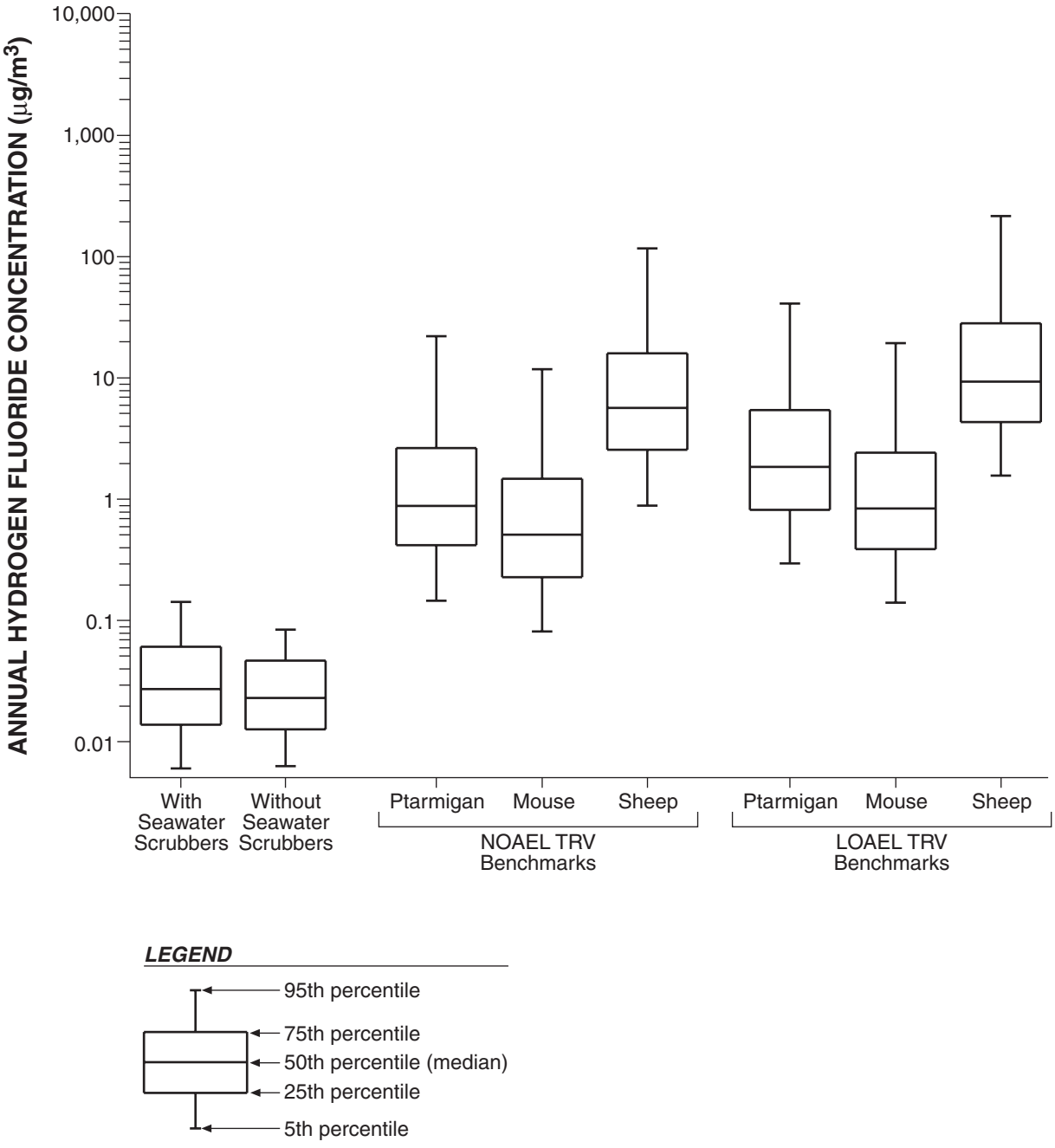
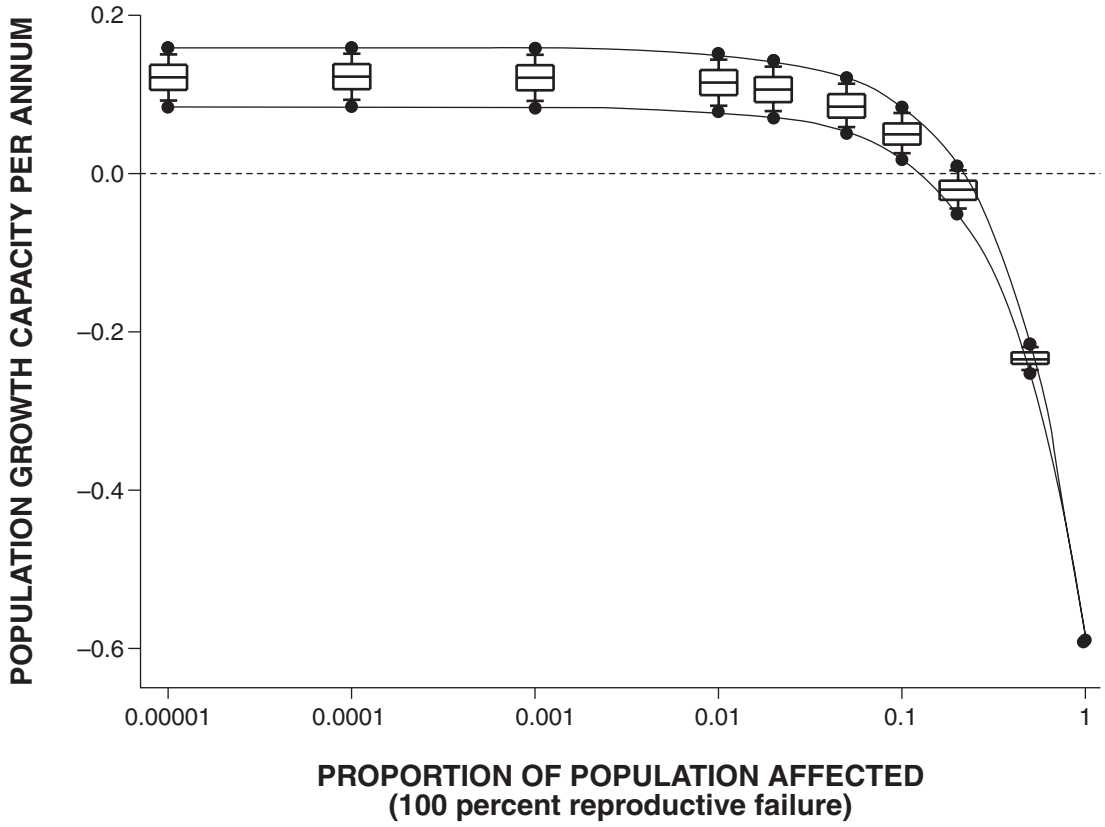


Figure 4-10. Modeled hydrogen fluoride concentrations with and without seawater scrubbers compared to NOAEL and LOAEL TRV benchmarks for ptarmigan, wood mouse, and sheep receptors, consuming heather



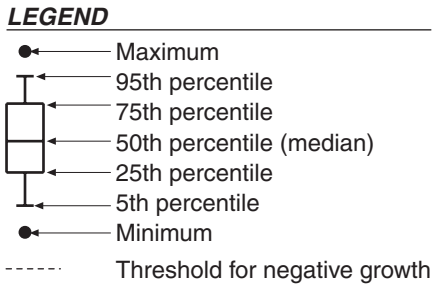
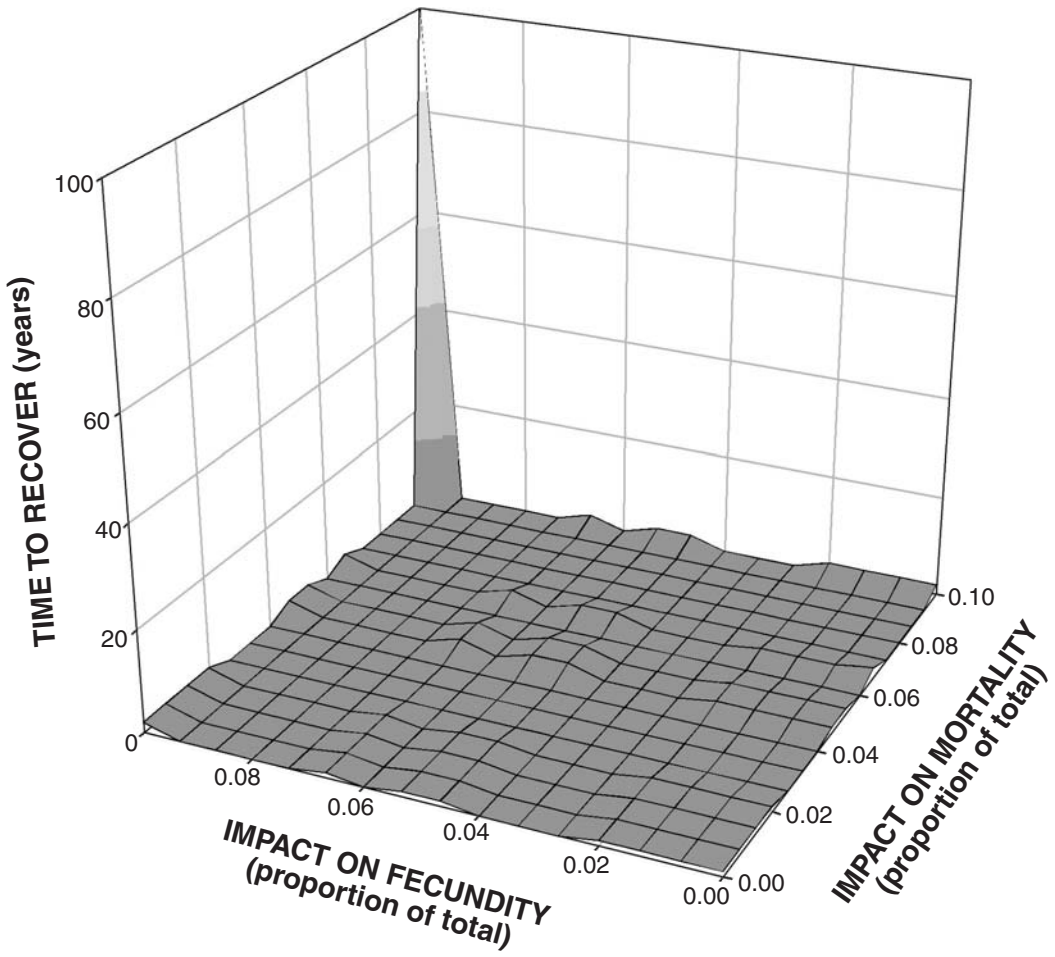
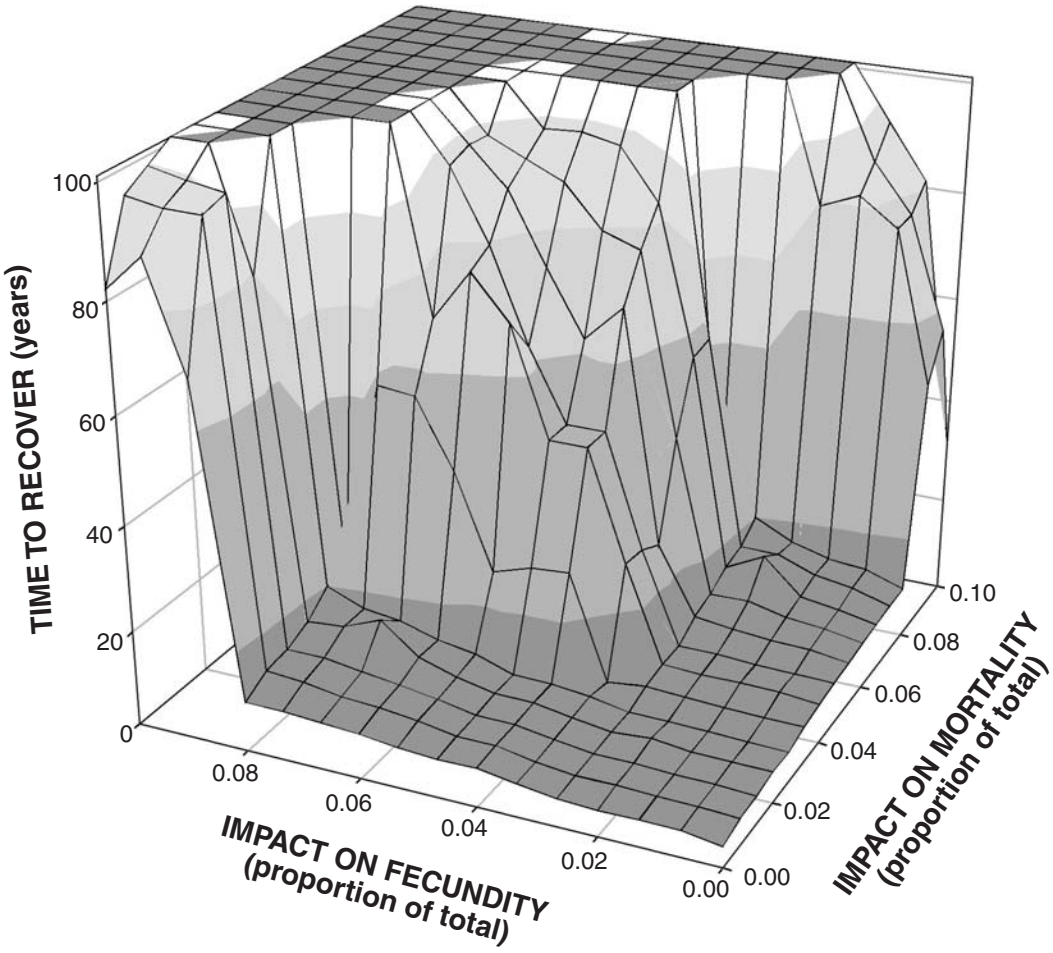


Figure 4-11. Predicted changes in annual population growth capacity for the rock ptarmigan



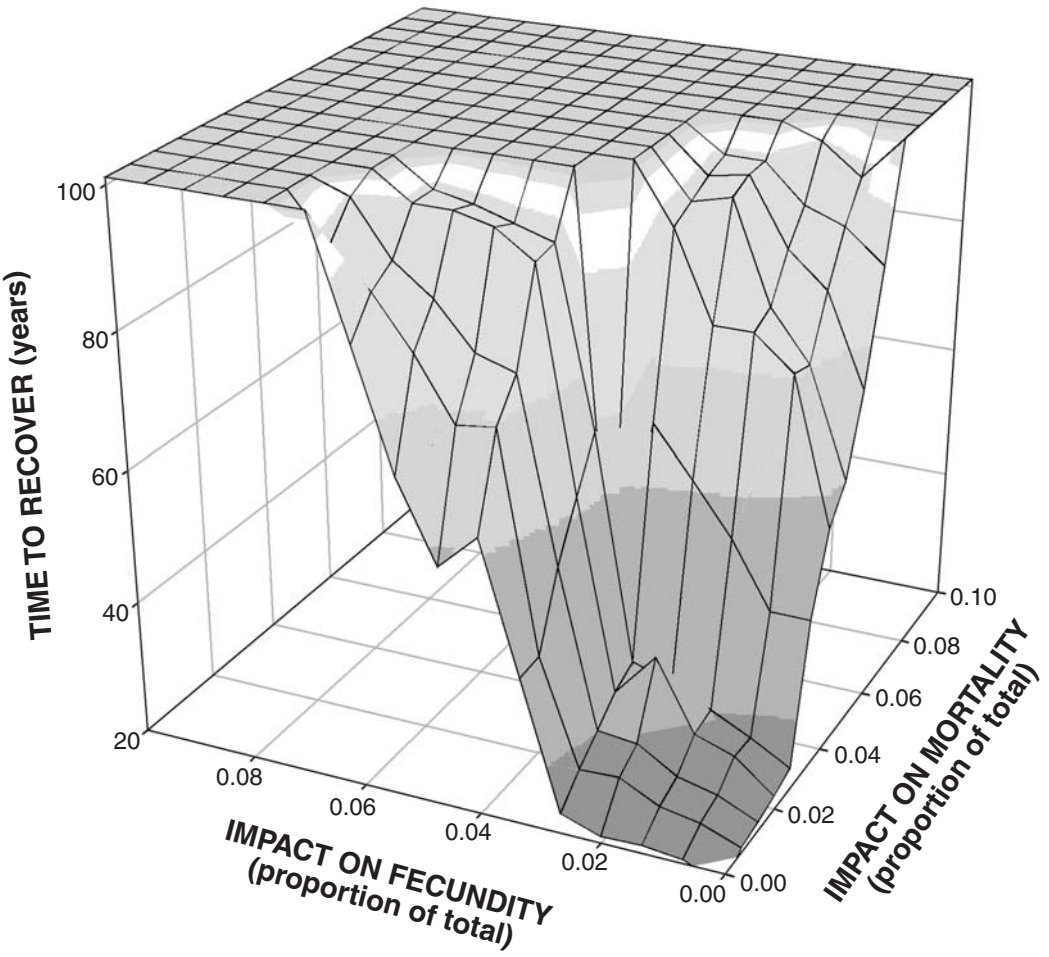
Note: Recovery times greater than 100 years indicate no recovery

Figure 4-12. Fifth percentile prediction of the time required to recover from one-half to full carrying capacity for a wood mouse subpopulation associated with the Fjardaal area



Note: Recovery times greater than 100 years indicate no recovery

Figure 4-13. Fiftieth percentile prediction of the time required to recover from one-half to full carrying capacity for a wood mouse subpopulation associated with the Fjardaal area



Note: Recovery times greater than 100 years indicate no recovery

Figure 4-14. Ninety-fifth percentile prediction of the time required to recover from one-half to full carrying capacity for a wood mouse subpopulation associated with the Fjardaal area

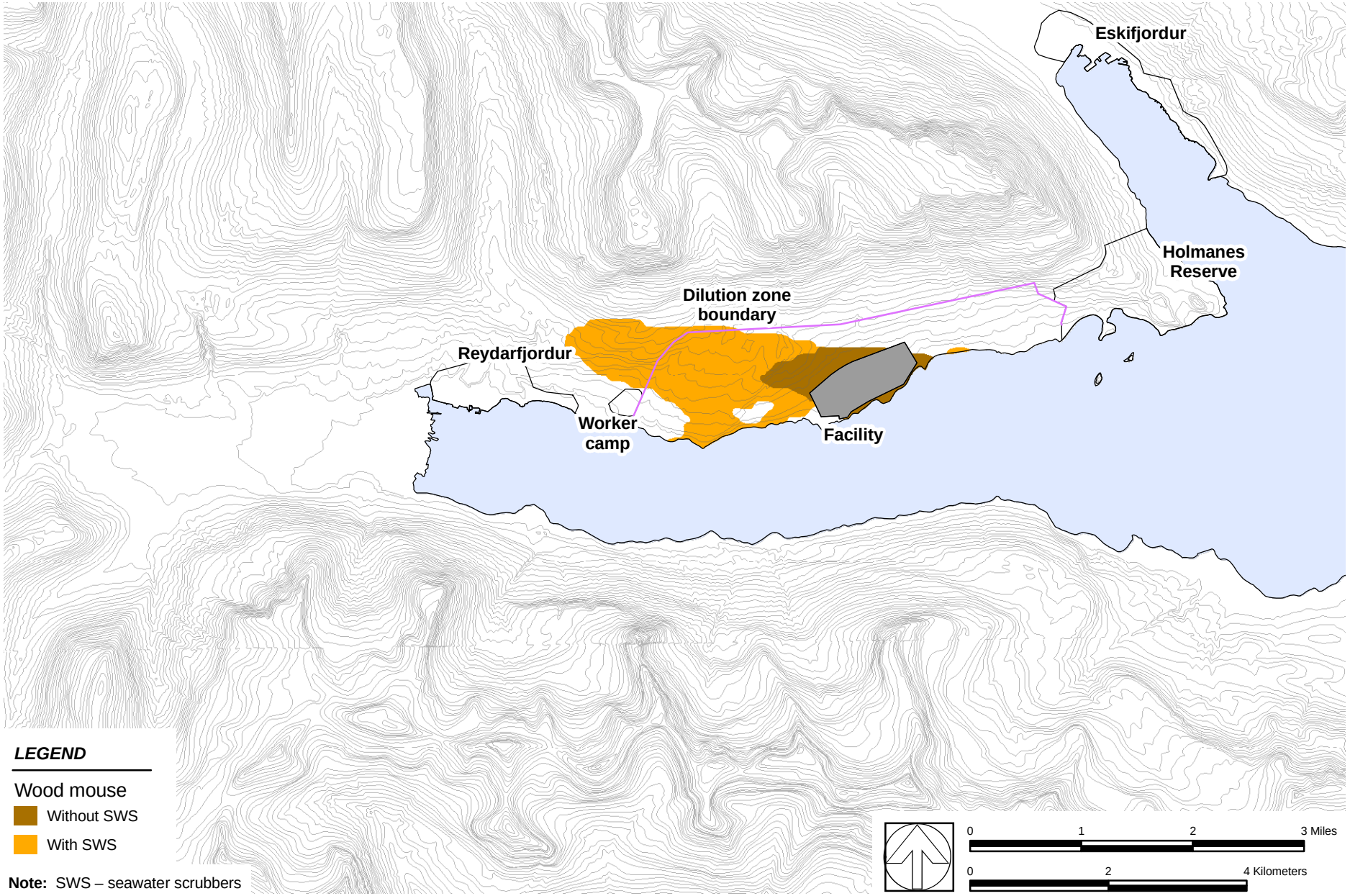


Figure 4-15. Areal extent of stable wood mouse population potentially below carrying capacity with and without seawater scrubbers

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Tables

Table 2-1. Point source parameters and emissions rates for base case and alternative case

Source	Exit Velocity (m/s)	Exit Temperature (K)	Emission Rate (g/s) Hydrogen Fluoride (g/s)	Particulate Fluoride (g/s)	Sulfur Dioxide (g/s)	PM_{10} (g/s)	BaP[a] (g/s)	PAHs (g/s)
Fume Stack[b]								
Base case (no seawater scrubbers)	19.0	362.15	0.94	0.086	142.8	2.16	2.81E-05	1.41E-03
Alternative case (with seawater scrubbers)	3.17	228.15	0.33	0.018	0	0.45	0	0
Casthouse Furnace Stacks (3)[b,c]	12.0	553.15	0	0	1.21	0.05	0	0
Potline Roofs (2)								
Base Case (no seawater scrubbers)								
Annual emissions	1.0	296.15	0.59	0.395	1.46[d]	0.29	2.14E-05	2.14E-03
Growing season emissions	1.0	296.15	0.76	0.505	1.46[d]	0.29	2.14E-05	2.14E-03
Alternative Case (with seawater scrubbers)								
Annual emissions	1.0	296.15	0.59	0.395	2.43[e]	0.29	2.14E-05	2.14E-03
Growing season emissions	1.0	296.15	0.76	0.505	2.43[e]	0.29	2.14E-05	2.14E-03
Seawater Scrubber Stacks (4)[b,f]	14.0	288.15	0.02	0.012	1.2	0.30	3.54E-06	1.77E-04

Note: BaP - benzo[a]pyrene
PAH - polycyclic aromatic hydrocarbon
PM_{10} - particulate matter <10 μm

[a] BaP is assumed to be 1 percent of PAHs.

[b] No difference between growing season and annual average.

[c] Same parameters for both base case and alternative case.

[d] Based on 1.8 percent sulfur coke.

[e] Based on 3 percent sulfur coke.

[f] Alternative case only.

Table 2-2. Summary of air modeling results used in the human health and ecological risk assessments

	Base Case (no seawater scrubbers)			Alternative Case (with seawater scrubbers)		
	With No Deposition	With Wet and Dry Depostion		With No Deposition	With Wet and Dry Depostion	
	Air Concentration	Air Concentration	Deposition Rate	Air Concentration	Air Concentration	Deposition Rate
Total Polycyclic Aromatic Hydrocarbons						
Annual mean	X	X	X	X	X	X
Growing season 1	X	--	X	--	--	X
Benzo[a]pyrene						
Annual mean	X	X	X	X	X	X
Growing season 1	--	--	X	--	--	X
Total Fluoride						
Annual mean	X	X	X	X	X	X
Growing season 1	--	--	X	--	--	X
Growing season 2	--	--	X	--	--	X
Particulate Fluoride						
Annual mean	X	--	X	X	--	X
Growing season 1	--	--	X	--	--	X
Growing season 2	--	--	X	--	--	X
Growing season 3	--	--	X	--	--	X
Growing season 4	--	--	X	--	--	X
Hydrogen Fluoride						
Annual mean	X	X	X	X	X	X
Growing season 1	--	X	X	--	X	X
Growing season 2	--	X	X	--	X	X
Growing season 3	--	X	--	--	X	--
Growing season 4	--	X	--		X	--
24 hour max	X	X	--	X	X	--
1 hour max	X	X	--	X	X	--
Sulfur Dioxide						
Annual mean	X	X	X	X	X	X
24 hour max	X	X	--	X	X	--
Days exeed 24 hour max (50)	--	X	--	--	X	
3 hour max	X	X	--	X	X	--
1 hour max	X	X	--	X	X	--
Days exeed 1 hour max (350)	--	X	--	--	X	
Days exeed 1 hour max (524)	--	X	--	--	X	
Particulate Matter <10 μm						
Annual mean	X	X	X	X	X	X
24 hour max	X	X	--	X	X	--

Note: X - results used in one or both risk assessments (10,784 values for each point in modeling grid)
-- - not available
Growing season 1: Apr 1 to Sep 30 calculated with annual emission rates for fluorides.
Growing season 2: Apr 1 to Sep 30 calculated with growing season emission rates for fluorides.
Growing season 3: Jun 1 to Aug 31 calculated with annual emission rates for fluorides.
Growing season 4: Jun 1 to Aug 31 calculated with growing season emission rates for fluorides.

Table 3-1. Inhalation toxicity values, risk-based concentrations, and standards

Chemical	Iceland Regulation No. 251/2002[a] 1-Hour ($\mu g/m^3$)	Iceland Regulation No. 251/2002[a] 24-Hour ($\mu g/m^3$)	Iceland Regulation No. 251/2002[a] Annual ($\mu g/m^3$)	Guideline for Protection of Human Health[a] ($\mu g/m^3$)	U.S. EPA NAAQS 3-Hour ($\mu g/m^3$)	U.S. EPA NAAQS 24-Hour ($\mu g/m^3$)	U.S. EPA NAAQS Annual (mean) ($\mu g/m^3$)	U.S. EPA IRIS RfC_i (mg/m^3)	U.S. EPA IRIS RfD_i (mg/kg-day)	U.S. EPA NCEA CSF_i $(mg/kg\text{-}day)^{-1}$	U.S. EPA Region 9 Inhalation RBC ($\mu g/m^3$)
Total Fluorides	--	--	--	--	--	--	--	--	--	--	--
Hydrogen fluoride	--	--	--	25 (24-hour)[b]	--	--	--	--	--	--	--
Particulate fluoride	--	--	--	--	--	--	--	--	--	--	--
Sulfur Dioxide	350	125–50	20	--	1,300 [c]	365 [c]	80	--	--	--	--
Particulate Matter <10 μm	--	50–20	20–10	--	--	150 [c]	50 [d]	--	--	--	--
PAHs (carcinogens)											
Benz[a]anthracene	--	--	--	--	--	--	--	--	--	0.73 r	0.0092
Benzo[b]fluoranthene	--	--	--	--	--	--	--	--	--	0.73 r	0.0092
Benzo[k]fluoranthene	--	--	--	--	--	--	--	--	--	0.073 r	0.092
Benzo[a]pyrene	--	--	--	0.0001–0.001 (annual)[e]	--	--	--	--	--	7.3 r	0.00092
Chrysene	--	--	--	--	--	--	--	--	--	0.0073 r	0.92
Dibenz[ah]anthracene	--	--	--	--	--	--	--	--	--	7.3 r	0.00092
Indeno[1,2,3-cd]pyrene	--	--	--	--	--	--	--	--	--	0.73 r	0.0092
PAHs (non-carcinogens)											
Acenaphthylene	--	--	--	--	--	--	--	--	--	--	--
Acenaphthene	--	--	--	--	--	--	--	--	0.060 r	--	219
Anthracene	--	--	--	--	--	--	--	--	0.30 r	--	1,095
Benzo[ghi]perylene	--	--	--	--	--	--	--	--	--	--	--
Fluoranthene	--	--	--	--	--	--	--	--	0.040 r	--	146
Fluorene	--	--	--	--	--	--	--	--	0.040 r	--	146
Naphthalene	--	--	--	--	--	--	--	0.0030	0.00086	--	3.1
Phenanthrene	--	--	--	--	--	--	--	--	--	--	--
Pyrene	--	--	--	--	--	--	--	--	0.030 r	--	110

Note: CSF_i - inhalation cancer slope factor
IRIS - Integrated Risk Information System (U.S. EPA 2005a)
NAAQS - national ambient air quality standard (U.S. EPA 2005c)
NCEA - National Center for Environmental Assessment (U.S. EPA 2005b)
PAH - polycyclic aromatic hydrocarbon
r - based on route-to-route extrapolation as provided in the risk-based screening tables from U.S. EPA Region 9; route-to-route extrapolation introduces some uncertainty into calculations as described in text
RBC - risk-based concentration
REL - reference exposure level
RfC_i - reference concentration for chronic inhalation exposure
RfD_i - reference dose for chronic inhalation exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] As cited in Earth Tech (2005a, 2006).

[b] Norwegian guideline for protection of human health

[c] Not to be exceeded more than once per year.

[d] To attain this standard, the 3-year average of the weighted annual mean PM_{10} concentration at each monitor within an area must not exceed 50 $\mu g/m^3$.

[e] BaP range of standards is identified as ambient air standard in Belgium, France, Italy, Netherlands, Sweden and United Kingdom (Earth Tech 2005a, 2006 pg. 8-4).

Table 3-2. Statistical summary of PAH concentration estimates at receptors, with and without seawater scrubbers (ng/m^3)

		With Seawater Scrubbers				Statistical Basis - (Scrubber)	
Receptor Location	N	Min.	Average	Max.	95%UCL	Distribution	UCL method
Annual Total							
Inside facility - onsite worker	87	0.30	2.4	9.0	2.7	Gamma	95% Approximate Gamma
Fenceline resident	622	0.088	0.45	1.6	0.50	Non-parametric	95% Chebyshev
Seagoing worker	836	0.097	0.43	1.3	0.47	Non-parametric	95% Chebyshev
70 percent of total annual PAH concentration (assumed all to be napththalene)							
Inside facility - onsite worker	87	0.21	1.7	6.3	1.9	Gamma	95% Approximate Gamma
Fenceline resident	622	0.061	0.32	1.1	0.35	Non-parametric	95% Chebyshev
Seagoing worker	836	0.068	0.30	0.90	0.33	Non-parametric	95% Chebyshev
Carcinogenic PAH concentrations (based on modeling of applying percentages of carcinogenic PAH from Tables 3-3 and 3-4)							
Inside facility - onsite worker	87	0.0024	0.0055	0.017	0.0060	Lognormal	95% H-statistic
Fenceline resident	622	0.0015	0.0082	0.025	0.0092	Non-parametric	95% Chebyshev
Seagoing worker	836	0.0016	0.0056	0.016	0.0060	Non-parametric	95% Chebyshev

		Without Seawater Scrubbers				Statistical Basis - (No Scrubber)	
Receptor Location	N	Min	Average	Max	95%UCL	Distribution	UCL method
Annual Total							
Inside facility - onsite worker	87	0.26	2.3	8.9	2.7	Gamma	95% Approximate Gamma
Fenceline resident	622	0.069	0.33	1.6	0.37	Non-parametric	95% Chebyshev
Seagoing worker	836	0.081	0.35	1.2	0.38	Non-parametric	95% Chebyshev
70 percent of total annual PAH concentration (assumed all to be napththalene)							
Inside facility - onsite worker	87	0.18	1.6	6.2	1.9	Gamma	95% Approximate Gamma
Fenceline resident	622	0.048	0.23	1.1	0.26	Non-parametric	95% Chebyshev
Seagoing worker	836	0.057	0.25	0.86	0.27	Non-parametric	95% Chebyshev
Carcinogenic PAH concentrations (based on modeling of applying percentages of carcinogenic PAH from Tables 3-3 and 3-4)							
Inside facility - onsite worker	87	0.00066	0.0023	0.0075	0.0026	Gamma	95% Approx. Gamma
Fenceline resident	622	0.00045	0.00075	0.0017	0.00077	Non-parametric	95% Modified-t
Seagoing worker	836	0.00055	0.00081	0.0015	0.00081	Non-parametric	95% Modified-t

Note: PAH - polycyclic aromatic hydrocarbon
UCL - upper confidence limit

Table 3-3. Deschambault data for total and carcinogenic PAHs used in modeling and risk estimates: Roof samples

Analyte (μg)	11/9/01 Total (μg)	11/23/01 Total (μg)	Average (μg)	With One-half Detection Limit	Percent of Total PAH	Relative Potency Estimate	Adjusted Percent Carcinogenic PAH
Roof Measurements (gaseous)							
Acenapthylene	0.09	0.11	0.10	0.10	0.30		
Chrysene	0	0.04	0.02	0.02	0.060	0.001	0.000060
Benzo[a]pyrene	0	0	0	0.0015	0.0045	1.0	0.0045
Benzo[ghi] perylene	0.03	0	0.015	0.015	0.045		
Fluorene	2.4	17.31	9.86	9.86	29.7		
Fluoranthene	0.66	8.00	4.33	4.33	13.1		
Benzo[b]+[j]+[k]fluorenthene	0.01	0	0.01	0.005	0.015	0.055	0.00083
Indeno[1,2,3-cd]pyrene	0	0	0	0.0015	0.0045	0.1	0.00045
Pyrene	0	2.62	1.31	1.31	4.0		
Phenanthrene	5.62	29.32	17.47	17.47	52.7		
Benz[a]anthracene	0	0	0	0.0015	0.0045	0.1	0.00045
Benzo[e]pyrene	0	0	0	0.0015	0.0045		
Dibenzo[a,h]anthracene	0	0	0	0.0015	0.0045	1.0	0.0045
Perylene	0	0	0	0.0015	0.0045		
Dibenzo[a,j]acridine	0	0	0	0.0015	0.0045		
7H-dibenzo[c,g] carbazole	0	0	0	0.0015	0.0045		
Dibenzo[a,i]pyrene	0.04	0	0.02	0.02	0.060		
Total	8.85	57.40	33.13	33.14	100		0.011
Roof Measurements (particulate)							
Acenapthylene	0	0	0	0.0015	0.15		
Chrysene	0.01	0.30	0.155	0.155	15.1	0.001	0.015
Benzo[a]pyrene	0	0	0	0.0015	0.15	1	0.15
Benzo[ghi] perylene	0.03	0.03	0.03	0.03	2.9		
Fluorene	0.03	0.08	0.055	0.055	5.4		
Fluoranthene	0.03	0.16	0.095	0.095	9.3		
Benzo[b]+[j]+[k]fluorenthene	0	0.20	0.10	0.10	9.8	0.055	0.54
Indeno[1,2,3-cd]pyrene	0	0	0	0.0015	0.15	0.1	0.015
Pyrene	0	0	0	0.0015	0.15		
Phenanthrene	0.37	0.49	0.43	0.43	42.0		
Benz[a]anthracene	0.01	0.28	0.145	0.145	14.1	0.1	1.4
Benzo[e]pyrene	0	0	0	0.0015	0.15		
Dibenzo[a,h]anthracene	0	0	0	0.0015	0.15	1	0.15
Perylene	0	0	0	0.0015	0.15		
Dibenzo[a,j]acridine	0	0	0	0.0015	0.15		
7H-dibenzo[c,g] carbazole	0	0	0	0.0015	0.15		
Dibenzo[a,i]pyrene	0	0	0	0.0015	0.15		
Total	0.48	1.54	1.01	1.03	100		2.3

Source: Palazzolo (2005b, pers. comm.)

Note: Deschambault roof sample—total based on set of six simultaneous cassettes in potroom roof.
Method detection limit is approximately 0.003 μg.
Carcinogenic PAHs are boxed.
PAH - polycyclic aromatic hydrocarbon

Table 3-4. Deschambault data for total and carcinogenic PAHs used in modeling and risk estimates: Stack samples

	8/28/2003 4:30:00 PM	8/28/2003 10:41:00 PM	8/29/2003 5:10:00 PM	Average	Percent of Total PAH	Relative Potency Estimate	Adjusted Average Carcinogenic PAH	Adjusted Percent Carcinogenic PAH
PAH–Particulate (mg)								
Naphtalene	0.0001	0.0002	0.0004	0.0002	6.0			
Acenaphtylene	0.0001	0.0001	0.0001	0.0001	2.5			
Acénaphtene	0.0001	0.0005	0.0009	0.0005	12.1			
Fluorene	0.0001	0.0004	0.0006	0.0004	9.1			
Phenanthrene	0.0001	0.0009	0.0017	0.0009	22.7			
Anthracene	0.0001	0.0001	0.0001	0.0001	2.5			
Fluoranthene	0.0001	0.0001	0.0004	0.0002	4.6			
Pyrene	0.0001	0.0001	0.0001	0.0001	2.5			
Benz[a]anthracene	0.0001	0.0001	0.0001	0.0001	2.5	0.1	0.00001	0.25
Chrysene	0.0001	0.0001	0.0001	0.0001	2.5	0.001	0.0000001	0.0025
Benzo[a]fluorene	0.0001	0.0001	0.0001	0.0001	2.5			
Benzo[b]+[j]+[k]fluoranthene	0.0002	0.0002	0.0002	0.0002	3.7	0.055	0.00000825	0.20
Benzo[b]fluorene	0.0001	0.0001	0.0001	0.0001	2.5			
Benzo[e] pyrene	0.0001	0.0001	0.0001	0.0001	2.5			
Benzo[a]pyrene	0.0001	0.0001	0.0001	0.0001	2.5	1	0.0001	2.5
Perylene	0.0001	0.0001	0.0001	0.0001	2.5			
Indeno[1,2,3-cd]pyrene	0.0001	0.0001	0.0001	0.0001	2.5	0.1	0.00001	0.25
Dibenz[a,h]anthracene	0.0001	0.0001	0.0001	0.0001	2.5	1	0.0001	2.5
Benzo[ghi]perylene	0.0001	0.0001	0.0001	0.0001	2.5			
Dibenzo[al]pyrene	0.0001	0.0001	0.0001	0.0001	2.5			
Dibenzo[a,e]pyrene	0.0001	0.0001	0.0001	0.0001	2.5			
Dibenzo[a,i] pyrene	0.0001	0.0001	0.0001	0.0001	2.5			
Dibenzo[a,h]pyrene	0.0001	0.0001	0.0001	0.0001	2.5			
Total Particulate	0.0024	0.0040	0.0059	0.0041	100	**Total Average Carcinogenic PAH**		5.6

Table 3-4. (cont.)

	8/28/2003 4:30:00 PM	8/28/2003 10:41:00 PM	8/29/2003 5:10:00 PM	Average	Percent of Total PAH	Relative Potency Estimate	Adjusted Average Carcinogenic PAH	Adjusted Percent Carcinogenic PAH
PAH–Gaseous (mg)								
Naphtalene	0.0005	0.0001	0.0001	0.0002	6.6			
Acenaphtylene	0.0001	0.0001	0.0001	0.0001	2.8			
Acénaphtene	0.0001	0.0001	0.0001	0.0001	2.8			
Fluorene	0.0004	0.0006	0.0007	0.0006	16.0			
Phenanthrene	0.0001	0.0005	0.0012	0.0006	17.1			
Anthracene	0.0001	0.0001	0.0001	0.0001	2.8			
Fluoranthene	0.0001	0.0001	0.0003	0.0002	5.2			
Pyrene	0.0001	0.0001	0.0001	0.0001	2.8			
Benz[a]anthracene	0.0001	0.0001	0.0001	0.0001	2.8	0.1	0.00001	0.28
Chrysene	0.0001	0.0001	0.0001	0.0001	2.8	0.001	0.0000001	0.0028
Benzo[a]fluorene	0.0001	0.0001	0.0001	0.0001	2.8			
Benzo[b]+[j]+[k]fluoranthene	0.0002	0.0002	0.0002	0.0002	4.2	0.055	0.00000825	0.23
Benzo[b]fluorene	0.0001	0.0001	0.0001	0.0001	2.8			
Benzo[e] pyrene	0.0001	0.0001	0.0001	0.0001	2.8			
Benzo[a]pyrene	0.0001	0.0001	0.0001	0.0001	2.8	1.0	0.0001	2.8
Perylene	0.0001	0.0001	0.0001	0.0001	2.8			
Indeno[1,2,3-cd]pyrene	0.0001	0.0001	0.0001	0.0001	2.8	0.1	0.00001	0.28
Dibenz[a,h]anthracene	0.0001	0.0001	0.0001	0.0001	2.8	1.0	0.0001	2.8
Benzo[ghi]perylene	0.0001	0.0001	0.0001	0.0001	2.8			
Dibenzo[al]pyrene	0.0001	0.0001	0.0001	0.0001	2.8			
Dibenzo[a,e]pyrene	0.0001	0.0001	0.0001	0.0001	2.8			
Dibenzo[a,i] pyrene	0.0001	0.0001	0.0001	0.0001	2.8			
Dibenzo[a,h]pyrene	0.0001	0.0001	0.0001	0.0001	2.8			
Total Gaseous	0.0031	0.0032	0.0044	0.0035	100	**Total Average Carcinogenic PAH**		6.5

Source: Palazzolo (2005b, pers. comm.)

Note: Deschambault GTC stack–total based on set of three test runs.

Method detection limit is approximately 0.0002 mg (0.0003 mg for some compounds).

Carcinogenic PAHs are boxed.

PAH - polycyclic aromatic hydrocarbon

Table 3-5. Statistical summary of estimated sulfur dioxide concentrations at receptors, with and without seawater scrubbers

		With Seawater Scrubbers				Without Seawater Scrubbers			
Receptor Location	N	Min.	Average	Max.	95%UCL	Min	Average	Max	95%UCL
1 Hour									
Inside facility	87	38.9	87.4	466	97.6	141	253	526	268
Dilution zone	622	35.9	81.9	314	84.1	81.7	237	668	243
Site hydro	836	22.6	85.6	286	87.6	96.4	294	740	301
Esk. Village	31	3.8	5.1	8.9	5.5	81.0	137	226	151
Rey. Village	100	31.1	53.6	97.1	56.4	81.2	200	490	246
Holm. Reserve	215	8.4	24.5	125	26.3	85.4	218	420	227
Work camp	16	46.4	53.4	61.7	55.6	204	246	280	257
Close farm	13	22.1	31.7	61.6	37.4	205	301	388	334
Close residence	12	36.0	42.8	47.6	44.6	135	164	199	176
3 Hour									
Inside facility	87	19.2	43.7	173	47.4	63.4	115	177	120
Dilution zone	622	14.6	41.5	109	42.5	31.8	101	237	103
Site hydro	836	14.0	39.2	134	40.0	48.5	145	391	149
Esk. Village	31	1.4	2.6	5.4	2.8	37.3	59.0	80.5	62.6
Rey. Village	100	20.9	32.9	61.8	34.6	33.9	82.8	172	89.0
Holm. Reserve	215	5.0	11.7	45.5	13.8	43.9	111	242	117
Work camp	16	21.4	25.3	29.6	26.1	70.0	83.5	94.8	87.0
Close farm	13	12.7	21.5	42.5	26.2	84.4	115	144	126
Close residence	12	18.0	20.0	21.6	20.6	51.1	64.0	79.6	68.0
24 Hour									
Inside facility	87	5.5	14.7	40.3	15.8	16.0	22.1	29.6	22.6
Dilution zone	622	2.2	11.0	23.0	11.3	7.1	17.6	44.8	18.0
Site hydro	836	2.9	8.9	22.0	9.1	10.8	25.9	67.8	26.6
Esk. Village	31	0.47	0.53	0.81	0.56	8.0	12.1	15.4	12.8
Rey. Village	100	5.2	7.4	16.2	7.8	9.5	14.3	30.3	15.2
Holm. Reserve	215	0.78	2.3	8.7	2.7	10.9	24.1	53.9	25.3
Work camp	16	5.8	7.6	9.4	8.1	17.7	20.5	23.4	21.3
Close farm	13	4.9	5.8	8.9	6.3	12.2	19.1	27.0	21.2
Close residence	12	5.2	6.5	7.7	6.9	13.5	15.5	17.7	16.1

Table 3-5. (cont.)

Receptor Location	N	With Seawater Scrubbers				Without Seawater Scrubbers			
		Min.	Average	Max.	95%UCL	Min	Average	Max	95%UCL
Annual									
Inside facility	87	0.56	3.0	10.9	3.4	0.90	2.3	6.8	2.5
Dilution zone	622	0.22	1.2	3.3	1.4	0.68	1.0	1.8	1.0
Site hydro	836	0.22	0.94	2.5	1.0	0.78	1.1	1.6	1.1
Esk. Village	31	0.05	0.058	0.064	0.059	0.56	0.62	0.71	0.64
Rey. Village	100	0.79	0.94	1.2	0.96	0.89	1.2	2.3	1.2
Holm. Reserve	215	0.10	0.19	1.1	0.20	0.51	0.81	1.2	0.83
Work camp	16	1.1	1.2	1.4	1.3	0.87	0.90	0.93	0.91
Close farm	13	0.67	0.72	0.80	0.74	1.4	1.6	1.9	1.7
Close residence	12	1.1	1.1	1.2	1.1	0.87	0.90	0.93	0.91

Note: UCL - upper confidence limit

Table 3-6. Values used for daily intake calculations for inhalation of PAHs

Inhalation Exposure Estimates CDI = (CA*IRa*EF*ED)/(BW*AT)			Onsite Adult Worker	Seagoing Worker	Fenceline Resident–Adult	Fenceline Resident–Child
Exposure Assumptions[a]						
Estimated concentration of PAH in air	CA	mg/m^3	*	*	*	*
Inhalation rate for air	IRa	m^3/day or workday	13 [b]	13 [b]	20 [c]	10 [c]
Exposure frequency	EF	days/year	250 [d]	100 [e]	350 [d]	350 [d]
Exposure duration[d]	ED	years	25 [d]	25 [d]	30 [d]	6 [d]
Body weight	BW	kg	70 [a]	70 [a]	70 [a]	15 [a]
Averaging time - carcinogenic	AT.c	days	25,550 [a]	25,550 [a]	25,550 [a]	25,550 [a]
Averaging time - noncarcinogenic	AT.n	days	9,125 [a]	9,125 [a]	10,950 [a]	2,190 [a]

Note: * - see Table 3-2
PAH - polycyclic aromatic hydrocarbon
U.S. EPA - U.S. Environmental Protection Agency

[a] General methodology based on guidance in U.S. EPA (1989, 1997b).

[b] Represents two hours at heavy activity level (2.5 m^3/hour) and 6 hours at average activity level (1.3 m^3/hour) (U.S. EPA 1997b, Table 5-23).

[c] Exposure assumptions from EPA Region 9 (U.S. EPA 2005b).

[d] Exposure frequency and duration assumptions from U.S. EPA (1997b).

[e] Exposure frequency for seagoing worker based on best professional judgment.

Table 3-7. Summary of total excess lifetime cancer risks for reasonable maximum exposure scenarios

Cancer Risk Estimates Assuming Scrubber			Cancer Risk Estimates Assuming No Scrubber		
Receptor/Exposure Pathway	Cancer Risk	Cancer Risk	Receptor/Exposure Pathway	Cancer Risk	Cancer Risk
Planned Fjardaal Smelter Site	Adult	Child	**Planned Fjardaal Smelter Site**	Adult	Child
On-site Outside Worker			**On-site Outside Worker**		
Inhalation	2E-9	--	Inhalation	9E-10	--
Total Cancer Risk:	**2E-9**	--	**Total Cancer Risk:**	**9E-10**	--
Fence-line Resident			**Fence-line Resident**		
Inhalation	8E-9	4E-9	Inhalation	7E-10	3E-10
Total Cancer Risk:	**8E-9**	**4E-9**	**Total Cancer Risk:**	**7E-10**	**3E-10**
Seagoing Worker			**Seagoing Worker**		
Inhalation	8E-10	--	Inhalation	1E-10	--
Total Cancer Risk:	**8E-10**	--	**Total Cancer Risk:**	**1E-10**	--

Note: PAH - polycyclic aromatic hydrocarbons

Table 3-8. Summary of total noncancer hazard indices for reasonable maximum exposure scenarios

Hazard Indicies Assuming Scrubber			Hazard Indicies Assuming No Scrubber		
Receptor/Exposure Pathway	Hazard Index	Hazard Index	Receptor/Exposure Pathway	Hazard Index	Hazard Index
Planned Fjardaal Smelter Site	Adult	Child	**Planned Fjardaal Smelter Site**	Adult	Child
On-site Outside Worker			**On-site Outside Worker**		
Inhalation	0.00028	--	Inhalation	0.00028	--
Total Noncancer Risk:	**0.00028**	--	**Total Noncancer Risk:**	**0.00028**	--
Fence-line Resident			**Fence-line Resident**		
Inhalation	0.00011	0.00026	Inhalation	0.000083	0.00019
Total Noncancer Risk:	**0.00011**	**0.00026**	**Total Noncancer Risk:**	**0.000083**	**0.00019**
Seagoing Worker			**Seagoing Worker**		
Inhalation	0.000019	--	Inhalation	0.000016	--
Total Noncancer Risk:	**0.000019**	--	**Total Noncancer Risk:**	**0.000016**	--

Note: PAH - polycyclic aromatic hydrocarbons

Table 4-1. Wildlife exposure parameters

Parameters	Units	Wood Mouse *Apodemus sylvaticus*		Rock Ptarmigan *Lagopus muta*		Icelandic (Domestic) Sheep *Ovis aries*	
Mean adult body weight	g	30.7 [a]	Silva and Downing (1995)	422 [a]	Dunning (1993)	70,000 [a]	OSU (1996)
Food ingestion rate (dw basis)	g/day	4.70 [b]	Nagy (2001)	19.2 [c]	Nagy (2001)	948 [d]	Nagy (2001)
Mean adult body weight	kg	0.030700	Silva and Downing (1995)	0.422000	Dunning (1993)	70.000000	OSU (1996)
Food ingestion rate (dw basis)	kg/day	0.004701	Nagy (2001)	0.019215	Nagy (2001)	0.947796	Nagy (2001)
Proportion of soil in diet	fraction	0.0240 [e]	Beyer and Fries (2003)	0.0930 [f]	Beyer and Fries (2003)	0.0450 [g]	Beyer and Fries (2003)
Soil ingestion rate	kg/day	0.00011	Beyer and Fries (2003)	0.00179	Beyer and Fries (2003)	0.04265	Beyer and Fries (2003)
Toxicity reference value (NOAEL) - PAH	mg/kg-day	1.00	Mackenzie and Angevine (1981)	0.143	Hough et al. (1993)	1.00	Mackenzie and Angevine (1981)
Toxicity reference value (LOAEL) - PAH	mg/kg-day	10.0	Mackenzie and Angevine (1981)	1.43	Hough et al. (1993)	10.0	Mackenzie and Angevine (1981)
Toxicity reference value (NOAEL) - Fluoride	mg/kg-day	24.5	NTP (1990)	13.2	Nahorniak et al. (1983)	24.5	NTP (1990)
Toxicity reference value (LOAEL) - Fluoride	mg/kg-day	42	NTP (1990)	26.5	Nahorniak et al. (1983)	42	NTP (1990)
Proportion of diet that is plant matter	fraction	1	Assumption	1	Assumption	1	Assumption

Note: LOAEL - lowest-observed-adverse-effect level
NOAEL - no-observed-adverse-effect level
PAH - polycyclic aromatic hydrocarbon

[a] Mean or median adult body weight, females.

[b] Food ingestion rate (Rodentia) = $0.332\ BW^{0.774}$.

[c] Food ingestion rate (Galliformes) = $0.088\ BW^{0.891}$.

[d] Food ingestion rate (Herbivores) = $0.859\ BW^{0.628}$.

[e] Value is for meadow vole.

[f] Value is for wild turkey.

[g] Yearly average for sheep, pasture as only feed source.

Table 4-2. Area associated with the probability of exposure to hydrogen fluoride by single individual at a concentration exceeding the TRV

Scenario	Probability	Number of Hectares Associated with Risk Levels	Percent of Total Study Area
Rock Ptarmigan			
With Seawater Scrubber - NOAEL TRV Benchmark			
	< 1/10,000	17,938	92.47
	< 1/1,000	32	0.16
	< 1/100	248	1.28
	< 1/10	1,033	5.33
	< 1/5	128	0.66
	< 1/2	21	0.11
	Total	19,400	100.00
Without Seawater Scrubber - NOAEL TRV Benchmark			
	< 1/10,000	18,785	96.83
	< 1/1,000	31	0.16
	< 1/100	279	1.44
	< 1/10	267	1.38
	< 1/5	24	0.12
	< 1/2	14	0.07
	Total	19,400	100.00
Wood Mouse			
With Seawater Scrubber - NOAEL TRV Benchmark			
	< 1/10,000	15,307	78.90
	< 1/1,000	106	0.55
	< 1/100	821	4.23
	< 1/10	2,468	12.72
	< 1/5	527	2.72
	< 1/2	164	0.85
	> 1/2	7	0.03
	Total	19,400	100.00
Without Seawater Scrubber - NOAEL TRV Benchmark			
	< 1/10,000	16,330	84.18
	< 1/1,000	109	0.56
	< 1/100	876	4.51
	< 1/10	1,973	10.17
	< 1/5	71	0.36
	< 1/2	37	0.19
	> 1/2	5	0.02
	Total	19,400	100.00
With Seawater Scrubber - LOAEL TRV Benchmark			
	< 1/10,000	15,914	82.03
	< 1/1,000	80	0.41
	< 1/100	719	3.70
	< 1/10	2,050	10.57
	< 1/5	476	2.45
	< 1/2	161	0.83
	Total	19,400	100.00
Without Seawater Scrubber - LOAEL TRV Benchmark			
	< 1/10,000	16,995	87.60
	< 1/1,000	88	0.45
	< 1/100	574	2.96
	< 1/10	1,642	8.46
	< 1/5	62	0.32
	< 1/2	40	0.21
	Total	19,400	100.00

Note: The diet is assumed to consist of 100 percent heather for all scenarios.

LOAEL - lowest-observed-adverse-effect level
NOAEL - no-observed-adverse-effect level
TRV - toxicity reference value

Table 4-3. Input parameters for matrix modeling of wood mouse populations

Percentiles	Carrying Capacity (K) (females/ha)	Fecundity (births/litter)	Monthly Survival Adult (Jun–Aug)	Monthly Survival Juvenile (Jun–Aug)	Monthly Survival Adult (Aug–Nov)	Monthly Survival Sub-adult (Aug–Nov)	Monthly Survival Juvenile (Aug–Nov)	Monthly Survival Adult (Nov–Jun)	Monthly Survival Sub-adult (Nov–Jun)
0	10.0	4.00	0.403	0.000	0.363	0.624	0.385	0.555	0.706
5	14.4	4.00	0.562	0.264	0.501	0.673	0.550	0.689	0.745
10	15.8	5.00	0.589	0.319	0.524	0.680	0.571	0.713	0.751
15	16.7	5.00	0.607	0.354	0.540	0.685	0.586	0.729	0.755
20	17.5	5.00	0.622	0.385	0.553	0.689	0.597	0.741	0.758
25	18.2	5.00	0.634	0.413	0.565	0.692	0.607	0.751	0.761
30	18.7	6.00	0.645	0.437	0.574	0.695	0.616	0.761	0.764
35	19.3	6.00	0.656	0.457	0.583	0.698	0.625	0.770	0.766
40	19.8	6.00	0.666	0.477	0.592	0.701	0.633	0.778	0.768
45	20.3	6.00	0.676	0.497	0.600	0.703	0.640	0.787	0.770
50	20.8	7.00	0.686	0.515	0.609	0.706	0.647	0.795	0.772
55	21.3	7.00	0.695	0.535	0.618	0.708	0.655	0.802	0.774
60	21.9	7.00	0.705	0.553	0.626	0.711	0.663	0.810	0.776
65	22.4	7.00	0.715	0.574	0.635	0.714	0.670	0.818	0.779
70	22.9	7.00	0.726	0.596	0.645	0.716	0.678	0.827	0.781
75	23.5	7.00	0.736	0.621	0.655	0.720	0.687	0.836	0.784
80	24.2	8.00	0.749	0.646	0.665	0.723	0.697	0.846	0.786
85	25.0	8.00	0.764	0.676	0.678	0.727	0.709	0.858	0.790
90	25.9	8.00	0.782	0.715	0.695	0.732	0.723	0.874	0.794
95	27.4	9.00	0.812	0.773	0.717	0.739	0.746	0.896	0.800
100	32.7	9.00	0.979	1.10	0.835	0.792	0.861	1.05	0.835

Appendix A

HHRA Risk Tables

Table A-1-RME- Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Onsite Outside Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	6.0E-09	mg/m^3	6.0E-09	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	1.9E-06	mg/m^3	1.9E-06	mg/m^3	M	2.4E-7	mg/kg-day	8.6E-4	mg/kg-day	0.00028
										Hazard Index:	0.00028

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-2-RME- Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Fenceline Resident - Adult

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	9.2E-09	mg/m^3	9.2E-09	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	3.5E-07	mg/m^3	3.5E-07	mg/m^3	M	9.6E-8	mg/kg-day	8.6E-4	mg/kg-day	0.00011
										Hazard Index:	0.00011

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-3-RME-Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Fenceline Resident - Child

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	9.2E-09	mg/m^3	9.2E-09	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	3.5E-07	mg/m^3	3.5E-07	mg/m^3	M	2.2E-7	mg/kg-day	8.6E-4	mg/kg-day	0.00026
										Hazard Index:	0.00026

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-4-RME- Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Seagoing Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	6.0E-09	mg/m^3	6.0E-09	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	3.3E-7	mg/m^3	3.3E-7	mg/m^3	M	1.7E-8	mg/kg-day	8.6E-4	mg/kg-day	0.000019
										Hazard Index:	0.000019

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-5-RME- No Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Onsite Outside Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	2.6E-09	mg/m^3	2.6E-09	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	1.9E-06	mg/m^3	1.9E-06	mg/m^3	M	2.4E-7	mg/kg-day	8.6E-4	mg/kg-day	0.00028
										Hazard Index:	0.00028

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-6-RME- No Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Fenceline Resident - Adult

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	7.7E-10	mg/m^3	7.7E-10	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	2.6E-07	mg/m^3	2.6E-07	mg/m^3	M	7.1E-8	mg/kg-day	8.6E-4	mg/kg-day	0.000083
										Hazard Index:	0.000083

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-7-RME-No Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Fenceline Resident - Child

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	7.7E-10	mg/m^3	7.7E-10	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	2.6E-07	mg/m^3	2.6E-07	mg/m^3	M	1.7E-7	mg/kg-day	8.6E-4	mg/kg-day	0.00019
										Hazard Index:	0.00019

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-8-RME- No Scrubber
Calculation of Noncancer Hazards
Planned Fjardaal Smelter Site
Seagoing Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Non-cancer)	Intake (Non-cancer) Units	Reference Dose[a]	Reference Dose Units	Hazard Quotient
Inhalation	**PAHs (carcinogens)**										
	Benzo[a]pyrene	8.1E-10	mg/m^3	8.1E-10	mg/m^3	M	--	mg/kg-day	ND	mg/kg-day	--
	PAHs (non-carcinogens)										
	Naphthalene	2.7E-7	mg/m^3	2.7E-7	mg/m^3	M	1.4E-8	mg/kg-day	8.6E-4	mg/kg-day	0.000016
										Hazard Index:	0.000016

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either U.S. EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from U.S. EPA Region 9 (U.S. EPA 2005a).

Table A-9-RME - Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Onsite Outside Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	6.0E-09	mg/m^3	6.0E-09	mg/m^3	M	2.7E-10	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	2E-9
	PAHs (non-carcinogens)										
	Naphthalene	1.9E-06	mg/m^3	1.9E-06	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
										Total Risk:	2E-9

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-10-RME- Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Fenceline Resident - Adult

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	9.2E-09	mg/m^3	9.2E-09	mg/m^3	M	1.1E-9	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	8E-9
	PAHs (non-carcinogens)										
	Naphthalene	3.5E-07	mg/m^3	3.5E-07	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
										Total Risk:	8E-9

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-11-RME- Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Fenceline Resident - Child

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	9.2E-09	mg/m^3	9.2E-09	mg/m^3	M	5.1E-10	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	4E-9
	PAHs (non-carcinogens)										
	Naphthalene	3.5E-07	mg/m^3	3.5E-07	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
											4E-9

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-12-RME- Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Seagoing Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	6.0E-9	mg/m^3	6.0E-9	mg/m^3	M	1.1E-10	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	8E-10
	PAHs (non-carcinogens)										
	Naphthalene	3.3E-7	mg/m^3	3.3E-7	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
											8E-10

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-13-RME - No Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Onsite Outside Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	2.6E-09	mg/m^3	2.6E-09	mg/m^3	M	1.2E-10	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	9E-10
	PAHs (non-carcinogens)										
	Naphthalene	1.9E-06	mg/m^3	1.9E-06	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
										Total Risk:	9E-10

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-14-RME- No Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Fenceline Resident - Adult

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	7.7E-10	mg/m^3	7.7E-10	mg/m^3	M	9.0E-11	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	7E-10
	PAHs (non-carcinogens)										
	Naphthalene	2.6E-07	mg/m^3	2.6E-07	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
											7E-10

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-15-RME- No Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Fenceline Resident - Child

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	7.7E-10	mg/m^3	7.7E-10	mg/m^3	M	4.2E-11	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	3E-10
	PAHs (non-carcinogens)										
	Naphthalene	2.6E-07	mg/m^3	2.6E-07	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
											3E-10

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Table A-16-RME- No Scrubber
Calculation of Cancer Risks
Planned Fjardaal Smelter Site
Seagoing Worker

Exposure Route	Chemical of Concern	Medium EPC Value[a]	Medium Units	Route EPC	Route EPC Units	EPC Applied	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor[a]	Cancer Slope Factor Units	Cancer Risk
	PAHs (carcinogens)										
	Benzo[a]pyrene	8.1E-10	mg/m^3	8.1E-10	mg/m^3	M	1.5E-11	mg/kg-day	7.3	$(mg/kg\text{-}day)^{-1}$	1E-10
	PAHs (non-carcinogens)										
	Naphthalene	2.7E-7	mg/m^3	2.7E-7	mg/m^3	M	--	mg/kg-day	ND	$(mg/kg\text{-}day)^{-1}$	--
											1E-10

Note:

-- - not applicable
EPC - exposure point concentration
M - medium-specific
ND - not determined (U.S. EPA)/not considered a carcinogen
PAHs - polycyclic aromatic hydrocarbons
RME - reasonable maximum exposure
U.S. EPA - U.S. Environmental Protection Agency

[a] Toxicity values obtained from either EPA Integrated Risk Information System (IRIS) September (U.S. EPA 2005b) or from EPA Region 9 (2005a).

Appendix B

Soil Concentration Estimate Derivation

Table B-1. Parameter estimates used to project PAH concentrations in plants

	Heather		Grass	
Percentiles	Standing Biomass (BMa) (kg/m^{-2})	Senescence Rate (L) (year)	Standing Biomass (BMa) (kg/m^{-2})	Senescence Rate (L) (year)
0%	0.561	0.000331	1.32	0.000127
5%	0.731	0.206	1.33	0.0759
10%	0.901	0.407	1.34	0.155
15%	1.07	0.594	1.36	0.230
20%	1.25	0.797	1.37	0.306
25%	1.44	0.987	1.38	0.376
30%	1.63	1.19	1.40	0.450
35%	1.80	1.39	1.41	0.524
40%	1.97	1.59	1.42	0.598
45%	2.14	1.79	1.43	0.672
50%	2.31	1.99	1.45	0.749
55%	2.49	2.20	1.46	0.822
60%	2.68	2.38	1.47	0.904
65%	2.85	2.59	1.49	0.977
70%	3.02	2.79	1.50	1.05
75%	3.19	2.98	1.51	1.13
80%	3.37	3.17	1.52	1.20
85%	3.54	3.38	1.54	1.27
90%	3.72	3.59	1.55	1.35
95%	3.91	3.80	1.56	1.43
100%	4.08	4.00	1.57	1.50

Note: Standing biomass predicted based on a cycling rate of 4 years for heather and 1.5 years for grasses.
The adhesion factor was conservitively asumes to be 1.0.
All data developed based on the results of Thorsteinsson et al. (1971).

Table B-2. Parameter estimates used to project fluoride concentrations in heather

Percentiles	Translation Constant (b)[a] (mg/kg^{-1})	Uptake Coefficient (m)[a] (mg/kg^{-1}-day^{-1})	Senescence Period (L)[b] (days)	Rowan to Heather Conversion Factor (f(Rowan->Heather))[a] (Unitless)
0%	-53.2	1.29	0.197	0.00000160
5%	-23.2	1.53	71.7	0.0311
10%	-18.6	1.57	147	0.0610
15%	-15.3	1.59	221	0.0940
20%	-12.6	1.61	286	0.130
25%	-10.3	1.63	366	0.177
30%	-8.35	1.65	434	0.216
35%	-6.24	1.66	504	0.241
40%	-4.46	1.67	582	0.265
45%	-2.71	1.69	653	0.288
50%	-1.08	1.70	724	0.315
55%	0.67	1.71	803	0.337
60%	2.39	1.73	877	0.364
65%	4.19	1.74	947	0.425
70%	6.02	1.75	1,021.00	0.542
75%	8.09	1.77	1,090.00	0.646
80%	10.4	1.78	1,160.00	0.780
85%	13.0	1.80	1,240.00	0.962
90%	16.2	1.83	1,310.00	1.14
95%	21.3	1.86	1,390.00	1.32
100%	47.3	2.08	1,460.00	1.50

[a] Values derived from Horntvedt (1997).

[b] Senescence period derived from NZDC (1995).

Table B-3. Parameter estimates used to project fluroide concentration in grass

Percentiles	Probability of Rain P(rain)[a] (Unitless)	Uptake Coefficient at 10 Days ($(k_{air})_{10}$) ($mg/kg^{-1}\ \mu g^{-1}/m^3\text{-}day^{-1}$)	Uptake Coefficient at 20 Days ($(k_{air})_{20}$) ($mg/kg^{-1}\ \mu g^{-1}/m^3\text{-}day^{-1}$)	Growth Period (G)[b] (days)	Correction Coefficient (M) (Unitless)	Corrected Uptake Coefficient (S) ($mg/kg^{-1}\ \mu g^{-1}/m^3$)
0%	0.316	8.57	5.50	10.0	0.0000129	0.00222
5%	0.316	10.1	6.48	10.0	0.00325	0.517
10%	0.323	10.1	6.72	19.0	0.0086	1.29
15%	0.323	10.1	6.97	28.0	0.0182	2.58
20%	0.357	10.9	6.97	37.0	0.0312	4.35
25%	0.377	10.9	6.97	46.0	0.0530	6.88
30%	0.377	10.9	7.21	55.0	0.0831	10.1
35%	0.407	10.9	7.21	64.0	0.126	14.5
40%	0.407	11.6	7.21	73.0	0.190	20.5
45%	0.413	11.6	7.46	83.0	0.281	28.8
50%	0.417	11.6	7.46	92.0	0.413	38.9
55%	0.417	11.6	7.46	102	0.615	51.3
60%	0.429	11.6	7.70	110	0.886	66.6
65%	0.429	12.4	7.70	119	1.28	84.0
70%	0.448	12.4	7.70	127	1.83	101
75%	0.463	12.4	7.94	136	2.70	116
80%	0.463	12.4	7.94	145	3.86	126
85%	0.465	13.1	8.19	154	5.51	137
90%	0.465	13.1	8.19	163	7.99	149
95%	0.468	13.9	8.43	172	10.9	166
100%	0.468	16.2	9.66	183	15.4	1,190.00

Note: k_{air} without precipitation at 10 days = 8.57 ($mg/kg^{-1}\ \mu g^{-1}/m^3\text{-}day^{-1}$) (Less et al. 1975)

k_{air} with precipitation at 10 days = 16.2 ($mg/kg^{-1}\ \mu g^{-1}/m^3\text{-}day^{-1}$) (Less et al. 1975)

k_{air} without precipitation at 20 days = ($mg/kg^{-1}\ \mu g^{-1}/m^3\text{-}day^{-1}$) (Less et al. 1975)

k_{air} with precipitation at 20 days = 10.4 ($mg/kg^{-1}\ \mu g^{-1}/m^3\text{-}day^{-1}$) (Less et al. 1975)

[a] Values derived from data provided by WMO (2005).

[b] Based on an assumed growth season of April 1 to September 30.

Table B-4. Parameter estimates and projections of fluoride soil concentrations at locations with maximum estimated deposition rates

Percentiles	Probability of Rain P(rain)[a] (Unitless)	Leafdrop (Bm_a/L)[b] (kg/m^{-2}-day^{-1})	Concentration of Fluoride in Grass ($f(k_{dep})$) (mg/kg^{-1})	Rate of Bulk Elimination from Soil ($k_{out\ 1}$) (kg/m^{-2}-day^{-1})	Area Grass Biomass (BM_a)[b] (kg/m^{-2})	Fluoride Concetration in Bare Soil [F] (mg/kg^{-1})	Fluoride Concetration in Vegtitated Soil [F] (mg/kg^{-1})
0%	0.316	0.00161	5.02	0.000401	0.880	0.0115	0.000641
5%	0.316	0.00178	6.41	0.122	0.889	3.74	0.198
10%	0.323	0.00186	8.41	0.217	0.897	6.98	0.349
15%	0.323	0.00192	11.3	0.288	0.906	9.59	0.509
20%	0.357	0.00198	15.4	0.351	0.914	12.1	0.696
25%	0.377	0.00204	21.5	0.402	0.923	14.2	0.947
30%	0.377	0.00211	29.7	0.444	0.931	16.1	1.32
35%	0.407	0.00219	41.1	0.483	0.940	17.9	1.85
40%	0.407	0.00227	56.9	0.525	0.948	19.8	2.50
45%	0.413	0.00237	75.2	0.559	0.957	21.5	3.32
50%	0.417	0.00249	100	0.593	0.965	23.2	4.43
55%	0.417	0.00262	132	0.623	0.974	24.8	5.79
60%	0.429	0.00279	173	0.651	0.982	26.3	7.30
65%	0.429	0.00298	221	0.682	0.990	28.1	9.17
70%	0.448	0.00322	263	0.714	1.00	30.0	11.4
75%	0.463	0.00350	290	0.745	1.01	31.8	14.1
80%	0.463	0.00390	318	0.781	1.02	34.0	17.0
85%	0.465	0.00447	345	0.814	1.02	36.1	21.1
90%	0.465	0.00535	371	0.848	1.03	38.4	26.6
95%	0.468	0.00769	417	0.894	1.04	41.5	38.3
100%	0.468	0.205	1,840	0.972	1.05	47.2	755

Note: Illustrative annual mean deposition rate = $0.0132\ \mu g/m^{-2}$ -s^{-1}
Annual mean concentration = $2.47\ \mu g/m^{-3}$

[a] Values derived from data provided by WMO (2005).

[b] Leafdrop was determined based on area biomass and scenescence rate for grasses in Iceland (Thorsteinsson et al. 1971).

Table B-5. Parameter estimates and projections of total PAH soil concentrations at locations with maximum estimated deposition rates

Percentiles	Probability of Rain P(rain)[a] (Unitless)	Leafdrop (Bm_a/L)[b] (kg/m^{-2}-day^{-1})	Concentration of PAH in Grass ($f(k_{dep})$) (mg/kg^{-1})	Rate of Bulk Elimination from Soil[c] ($k_{out\ 1}$) (kg/m^{-2}-day^{-1})	Area Grass Biomass (BM_a)[b] (kg/m^{-2})	PAH Concetration in Bare Soil [PAH] (mg/kg^{-1})	PAH Concetration in Vegtitated Soil [PAH] (mg/kg^{-1})
0%	0.316	0.00161	0.00414	4.02E-07	0.880	3.47	0.0000339
5%	0.316	0.00178	31.4	4.02E-07	0.888	3.47	0.174
10%	0.323	0.00185	63.3	4.10E-07	0.897	3.47	0.347
15%	0.323	0.00192	92.6	4.10E-07	0.906	3.47	0.515
20%	0.357	0.00197	124	4.53E-07	0.914	3.47	0.674
25%	0.377	0.00204	154	4.53E-07	0.923	3.47	0.848
30%	0.377	0.00212	182	4.80E-07	0.931	3.47	1.01
35%	0.407	0.00219	214	5.17E-07	0.940	3.47	1.19
40%	0.407	0.00228	245	5.17E-07	0.948	3.47	1.37
45%	0.413	0.00237	277	5.25E-07	0.957	3.47	1.53
50%	0.417	0.00248	306	5.25E-07	0.966	3.47	1.69
55%	0.417	0.00261	335	5.29E-07	0.974	3.47	1.86
60%	0.429	0.00277	366	5.45E-07	0.983	3.48	2.05
65%	0.429	0.00296	397	5.45E-07	0.991	3.48	2.23
70%	0.448	0.00321	425	5.70E-07	1.00	3.48	2.44
75%	0.463	0.00349	456	5.70E-07	1.01	3.48	2.67
80%	0.463	0.00390	489	5.88E-07	1.02	3.48	2.97
85%	0.465	0.00458	522	5.90E-07	1.02	3.49	3.43
90%	0.465	0.00560	553	5.90E-07	1.03	3.49	4.24
95%	0.468	0.00805	590	5.94E-07	1.04	3.49	6.02
100%	0.468	0.280	678	5.94E-07	1.05	3.49	85.7

Note: Illustrative annual mean deposition rate = 0.0190 ng/m^{-2}-s^{-1}

[a] Values derived from data provided by WMO (2005).

[b] Leafdrop was determined based on area biomass and scenescence rate for grasses in Iceland (Thorsteinsson et al. 1971).

[c] Bulk elimination based on a K_{oc} for benzo[a]pyrene of $7.87x10^5$ (U.S. EPA 2000). Biodegredation for PAHs based on a biological halflife in soil of 60 days ($K_{out\ 2}$ = 0.0116 day^{-1}).

Appendix C

Toxicity Data Test Results from the Scientific Literature

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Plants					
Horsman and Wellburn (1976)		*Lycopersicon, Phaseolus*	Increased free sugars	1.3 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Phaseolus*	Decreased keto-acid levels	1.7–7.6 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Chenopodium Polygonum*	Stimulated pentose phosphate pathway	5 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Glycine*	Inhibited UDP-glucose-fructose transglycosylase; stimulated phosphoenolpyruvate; inhibited phosphoglucomutase	25 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Phaseolus*	Stimulated enolase	1.7–2.6 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Glycine*	Stimulated enolase	8.2 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Sorghum*	Stimulated enolase	5 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Sorghum*	Initially stimulated, then inhibited catalase	5 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Phaseolus*	Stimulated catalase	(1.7–2.6 μg/m^3)	Controlled air
Horsman and Wellburn (1976)		*Glycine*	Stimulated catalase	51-96 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Sorghum*	Stimulated pyruvate kinase	5 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Pisum*	Stimulated glucose-6-phosphate dehydrogenase	(0.5–5 mM)	Controlled air
Horsman and Wellburn (1976)		*Glycine*	Stimulated glucose-6-phosphate dehydrogenase, cytochorme oxidase and peroxidase; inhibted polyphenol oxidase; initially stimulated and then inhibited ascorbate oxidase	51–96 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Solanum pseudocapsicum*	Stimulated peroxidase, glucose-6-phosphate dehydrogenase, acid phosphatase, phosphoglucomutase	2 μg/m^3	Controlled air
Horsman and Wellburn (1976)		*Pinus strobus*	Plasma-membrane ATPase	1.6 μg/m^3	Controlled air
As reviewed by Weinstein and Davison (2004)		*Cornus florida, Liquidambar styraciflua, Plantanus occidentalis, Liriodendron tulipifera, Acer rubrum, Oxydendrum arboreum, Pinus strobus, Pinus taeda, Pinus echinata*	Decrease in net photosynthesis	(1 mM NaF)	

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
As reviewed by Weinstein and Davison (2004)		*Glycine max*	Decrease in photophosphorylation	(10 mM NaF)	
As reviewed by Weinstein and Davison (2004)		*Azalea cvs*	Decrease in net photosynthesis	(20 mM KF)	
As reviewed by Weinstein and Davison (2004)		*Gladiolus*	Decrease in net photosynthesis; increase in injury	0.8–8 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Hordeum*	Decrease in net photosynthesis	32 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Medicago*	Total inhibition of net photosynthesis with recovery in hours or days	200 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Lycopersicon*	No effect	0.9–11.2 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)	Fruit trees	NA	14% decrease in net photosynthesis; 10% injury	2.1 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Gladiolus*	Reduction in net photosynthesis	3.1–5.2 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Citrus*	No effect	0.32–0.77 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Gossypium*	No effect	13.6 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Fragaria*	No effect	2.3 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Fragaria*	Decrease in net photosynthesis	38 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Prunus*	No effect	1.6 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Zea*	No effect	2.7 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Hordeum, Medicago*	Decrease in net photosynthesis during expsure, recovery after exposure	32 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Sorghum*	No effect	0.7 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Sorghum*	Decrease in net photosynthesis	3.5 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Vicia faba*	Decrease in net photosynthesis in 24 hours, followed by partial recovery	41 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Pinus sylvestris*	Decrease in net photosynthesis	20 $\mu g/m^3$ HF	Controlled air

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
As reviewed by Weinstein and Davison (2004)		*Pinus ellottii*	No effect	1.2–4 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Pinus strobus*	No effect on net photosynthesis	0.4–1.6 $\mu g/m^3$ HF	Controlled air
As reviewed by Weinstein and Davison (2004)		*Mangifera indica*	Decrease in net photosynthesis	12–24 $\mu g/m^3$ HF	Controlled air
Weinstein and Davison (2003)	White clover	*Trifolium repens*	Injury	0.24–0.29 $\mu g/m^3$ (0.25–0.3 $\mu g/m^3$ HF)	Controlled air
Palmason and Magnusson (1998)	Red clover		Chlorosis	0.81 $\mu g/m^3$ (0.85 $\mu g/m^3$ HF)	Controlled air
Weinstein and Davison (2003)	White clover	*Trifolium repens*	Severe narcosis	1.05 $\mu g/m^3$ (1.1 $\mu g/m^3$ HF)	Controlled air
Palmason and Magnusson (1998)	White clover		Chlorosis	2.47 $\mu g/m^3$ (2.6 $\mu g/m^3$ HF)	Contolled air
Palmason and Magnusson (1998)	Red clover		No effect on yield	2.7 $\mu g/m^3$ (2.8 $\mu g/m^3$ HF)	Controlled air
Palmason and Magnusson (1998)	Grasses	NA	No effect on grass yield	2.8 $\mu g/m^3$	Controlled air
Vike (1999)	Various vegetation		Leaf injury	30 mg/kg	Ambient air
Palmason and Magnusson (1998)	Oats	NA	Decrease in yield by 25%	4.8 $\mu g/m^3$ (5.1 $\mu g/m^3$ HF)	Controlled air
Belandria et al. (1989)	Lichen	*X. parietina; P. distorta; P. canina*	80% loss of germination	19 mg/L (1,000 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*P. distorta*	37% loss of germination	0.95 mg/L (50 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*Xanthoria parientina; P. distorta; P. canina*	Spore germination inhibition	19 mg/L (1,000 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*L. conizaeoides*	Spore germination inhibition	38 mg/L (2,000 μM)	Controlled solution
Vike and Habjorg (1995)	Various vegetation	*Betula pubescens, Salix caprea, and Sorbus aucuparia*	Leaf injury	100–170 mg/kg	Tissue
Palmason and Magnusson (1998)	Grasses	NA	No effect	137–186 mg/kg	Tissue
Palmason and Magnusson (1998)	Timothy grass	*Phleum pratense*	No effect	145 mg/kg	Tissue
Palmason and Magnusson (1998)	White clover		Chlorosis	222 mg/kg	Tissue
Weinstein and Davison (2003)	Lichen		Threshold level at which injury is produced	25–80 mg/kg	Tissue

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Palmason and Magnusson (1998)	Grasses	NA	Leaf tip damage	282–582 mg/kg	Tissue
Stevens et al. (2000)	Plants	NA	Toxic threhold	35 mg/kg	Shoot tissue
Stevens et al. (2000)	Cattle herbage	*subterranean clover, thistle, barley grass, cocksfoot and sorrel*	Exceedance of safe levels for cattle herbage	40 mg/kg in foliage	Tissue
Vike and Habjorg (1995)	Scots pine	*Pinus sylvestris*	Leaf injury	50 mg/kg	Tissue
Palmason and Magnusson (1998)	Timothy grass	*Phleum pratense*	Unspecified effects	530 mg/kg	Tissue
Domingos et al. (2003)	Rainforest vegetation	*Tibouchina pulchra*	Metabolic changes, visible injury	700 mg/kg	Foliage tissue
Weinstein and Davidson (2003)	Vegetation		Background concentration in plants	950–9,500 $\mu g/m^3$ (1,000–10,000 $\mu g/m^3$ HF)	Tissue
Stevens et al. (2000)	Soil		Fluoride concentration in soil solution from a variety of soils	28.5–34.96 mg/L (1.5–1.84 mM)	Soil
Perkins et al. (1980)	Lichen		Critical concentration in thalli; chlorotic and necrotic with disruption in algal component	30–80 mg/kg	NA
Perkins et al. (1980)	Lichen		Decrease in abundance	>50 mg/kg	NA
Perkins et al. (1980)	Lichen		Lichen killed	>100 mg/kg	NA
Horsman and Wellburn (1976)	Soybean	*Glycine*	Inhibited chlorophyll synthesis	(13 mM NaF)	NA
Horsman and Wellburn (1976)		*Phaseolus*	Decreased Hill-reaction activity	(35 mM KF)	NA
Horsman and Wellburn (1976)		*Avena*	Inhibited cellulose synthesis	(5 mM)	NA
Horsman and Wellburn (1976)		*Avena*	Inhibited phosphoglucomutase	(10 mM NaF)	NA
Horsman and Wellburn (1976)		*Pisum*	Inhibited enoclase	(0.1–10 mM NaF)	NA
Horsman and Wellburn (1976)		*Phaseolus, Zea*	Reduced number of ribsomal activity	(0.5–5 mN NaF)	NA
Invertebrates					
Stratus Consulting (2000)	Fruitflies		Chromosonal damage	1.23–2.47 mg/kg (1.3–2.6 mg/kg HF)	Controlled air
Stratus Consulting (2000)	Worker bees		Reduced lifespan	4,000–5,000 mg/kg	Controlled air
Port et al. (1998)	Cabbage looper	*Trichoplusia ni*	Reduction in larval development, feeding, growth and rate of development	47.5–190 mg/kg (50–200 mg/kg HF)	Controlled air
Stratus Consulting (2000)	Fruitflies		Chromosonal damage	4,940 mg/kg (5,200 mg/kg HF)	Ambient air
Port et al. (1998)	Mexican bean beetles	*Epilachna varivestis*	Fewer egg laying	950 mg/kg (1,000 mg/kg HF)	Controlled air

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Camargo (1996)	Caddisflies	*Hydropsyche bronta*	LC0.01	2.6 mg/L	Controlled water
Camargo (1996)	Caddisflies	*Hydrosyche occidentalis*	LC0.01	6.4 mg/L	Controlled water
Camargo (1996)	Caddisflies	*Cheumatopsyche pettiti*	LC0.01	7.7 mg/L	Controlled water
Port et al. (1998)	flour beetles	*Tribolium confususum*	No effect	10–100 mg/kg	In food
Van Wensem and Adema (1991)	Isopod	*Porcellio scaber*	NOEC for decreased ability to decompose leaf litter	100.7 mg/kg (5.3 μmol/g)	Added to leaf litter
Mitterbock and Fuhrer (1988)	Nun-moth larvae	*Lymantria monarcha*	75% increase in mortality and delay in development	365 mg/kg	In food/ vegetation
Port et al. (1998)	Cabbage white butterfly	*Pieris brassicae*	No adverse effects on pupal weight	114 mg/kg (120 mg/kg HF)	In food/ vegetation
Davies et al. (1992)	Pine sawfly		No effect on pupae weight change	143 mg/kg	Food
Port et al. (1998)	Cabbage looper	*Trichoplusia ni*	Reduction in larval development, feeding, growth and rate of development	13.1–81.9 mg/kg (40–250 mg/kg KF)	In food
Port et al. (1998)	Cabbage looper	*Trichoplusia ni*	Reduction in larval development, feeding, growth and rate of development	22.6–90.5 mg/kg (50–200 mg/kg NaF)	In food
As reviewed by Weinstein and Davison (2004)	Silkworms		Toxicity threshold	30 mg/kg	In food (mulberry leaves)
As reviewed by Weinstein and Davison (2004)	Silkworms		Lethal dose	120–200 mg/kg	In food (mulberry leaves)
Port et al. (1998)	Flour beetles	*Tribolium confususum*	Decrease in egg production	1,000 mg/kg	In food
Port et al. (1998)	Flour beetles	*Tribolium confususum*	Mortality and decrease in egg production	10,000 mg/kg	In food
As reviewed by Weinstein and Davison (2004)	Mexican bean beetle larvae	*Epilachna varivestis*	Decreased body weight	1,000 mg/kg	In food/ vegetation
Mitterbock and Fuhrer (1988)	Nun-moth larvae	*Lymantria monarcha*	Mortality	1,400–1,500 mg/kg	In food/ vegetation
Van Wensem and Adema (1991)	Isopod	*Porcellio scaber*	Mortality	3,230 mg/kg (170 μmol/g)	Added to leaf litter
Port et al. (1998)	Caterpillars	*Scotia segentum*	Mortality	271.4–1,085.7 mg/kg (600–2,400 mg/kg NaF)	In food
Stratus Consulting (2000)	Bee		LD50	10 μg/bee	Tissue
Stratus Consulting (2000)	Honeybee		Toxicity (unspecified)	100–200 mg/kg	Tissue
Stratus Consulting (2000)	Bee		Lethality	130–170 mg/kg	Tissue
Stratus Consulting (2000)	Honeybees/ bumblebees		NA	29–406 mg/kg	Tissue
Port et al. (1998)	Lepidopteran larvae	*Pieris brassicae*	No effect	500 mg/kg	Tissue

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Van Wensem and Adema (1991)	Silkworms		Mortality	30.4–49.4 mg/kg (1.6–2.6 μmol/g)	NA
Mammals					
Newman and Markey (1976)	Deer mouse		Weight loss, dental disfigurement, structural bone changes, mineralization of tendons	38 mg/kg	Food
NAS (1974)	Young beef/ dairy heifer		Dietary tolerance	40 mg/kg	Food
Stratus Consulting (2000)	Cattle		Chronic fluorosis; osteosclerosis; osteonecrosis; failure of modeling of bone, teeth and gum problems	40 mg/kg in foliage	Food
NAS (1974)	Mature beef/ dairy heifer		Dietary tolerance	50 mg/kg	Food
NAS (1974)	Breeding ewe		Dietary tolerance	60 mg/kg	Food
NAS (1974)	Horse		Dietary tolerance	60 mg/kg	Food
Boulton et al. (1994b)	Voles		Mild dental lesions	80 mg/kg	Food
NAS (1974)	Finishing cattle		Dietary tolerance	100 mg/kg	Food
NAS (1974)	Growing dog		Dietary tolerance	100 mg/kg	Food
Boulton et al. (1994b)	Voles	*Microtus*	Reduced live-weight gain; 40–100% mortality; marked dental lesions	100–300 mg/kg	Food
Mehdi et al. (1978)	Mouse		Depression, emaciated, dehydrated, arched back, mottled incisors	125 mg/kg	Food
NAS (1974)	Feeder lamb		Dietary tolerance	150 mg/kg	Food
NAS (1974)	Finishing pig		Dietary tolerance	150 mg/kg	Food
NAS (1974)	Breeding sow		Dietary tolerance	150 mg/kg	Food
Mehdi et al. (1978)	Sheep		Decreased blook levels of copper and cadmium, decreased packed cell volume	232 mg/kg	Food
Mehdi et al. (1978)	Mouse		Arched backs, death after eighth week	500 mg/kg	Food
Newman and Markey (1976)	Deer mouse		Mortality after 9 weeks exposure	1,065 mg/kg	Food
Newman and Markey (1976)	Deer mouse		Dental disfigurement, structural bone changes, mineralization in tendons	1,355 mg/kg	Food
Newman and Markey (1976)	Deer mouse		Mortality after 2 weeks exposure	1,936 mg/kg	Food
Shupe et al. (1987)	Mink		No effects	4.75 mg/kg	Tissue

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Pillai et al. (1989)	Mouse		Decreased hemoglobin	5.2 mg/kg	Tissue
Pillai et al. (1989)	Mouse		Decreased red blood cells	17.3 mg/kg	Tissue
Pillai et al. (1987)	Mouse		Mortality of 20% of test animals	46 mg/kg	Tissue
Pillai et al. (1987)	Mouse		LD50	51.6–54.4 mg/kg	Tissue
Stratus Consulting (2000)	White-tailed deer	*Odocoileus virginianus*	Fluorosis	55 μg/deer/day	Tissue
Boulton et al. (1994a)	White mouse		No effect	117 mg/kg	Tissue
Shupe et al. (1987)	Mink		Recommented maximum ingestion for breeding mink	135 mg/kg	Tissue
Boulton et al. (1994a)	Bank vole		Weight loss	390 mg/kg	Tissue
Boulton et al. (1994a)	Field vole		Mortality	580 mg/kg	Tissue
Stratus Consulting (2000)	Meadow vole			1,310 mg/kg	Tissue
Cooke et al. (1996)	Vole		Slight dental lesions	1,866 mg/kg	Tissue (femur)
Cooke et al. (1996)	Small mammal		Dental fluorosis	2,500 mg/kg	Tissue (femur or whole skeleton)
Cooke et al. (1996)	Small mammal		Dental fluorosis	2,000 mg/kg	Tissue (teeth)
Boulton (1992)	Field vole		Flurosis in teeth	189 mg/kg	Tissue (femur)
Stratus Consulting (2000)	Meadow voles	*Microtus pennsylvanicus*	Fluorosis; increased tooth wear and lesions	1,310–5,599 mg/kg	Tissue (femur)
Newman and Yu (1976)	Black-tailed deer	*Odocoileus hemionus columbianus*	Dental lesions	2,800–6,800 mg/kg	Tissure (ribs)
Karstad (1967)	Mule deer		Chronic fluorosis; mottling, pitting and discoloration of teeth	4,300–7,125 mg/kg	Tissue (mandibular bone)
Stratus Consulting (2000)	Short-tailed shrews	*Blarina brevicada*	Fluorosis; increased tooth wear and lesions	5,284–8,678 mg/kg	Tissue (femur)
Stratus Consulting (2000)	Cattle		Fluorosis (bone disease)	NA	Tissue
As reviewed by Weinstein and Davison (2004)	Herbivorous vole and bank vole	*M. agrestis* and *Clethrionymus glareolus*	Premature mortality	40 mg/L	Water
As reviewed by Weinstein and Davison (2004)	Mouse		Toxic effects on female reproduction and fetal and newborn mice development	110 mg/L (200 mg/L NaF)	Water
Mehdi et al. (1978)	Rat		Decreased hemoglobin and incorporation of iron into red blood cells and spleen, increased iron incorporation into liver and bone marrow	150 mg/L	Water
Stratus Consulting (2000)	Short-tailed shrew			8,678 mg/kg	

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Birds					
Bird and Massari (1983)	American kestrels		Comparable sized clutches with higher fertility than controls	22.6 mg/kg	Food
Bird and Massari (1983)	American kestrels		Greater eggshell thickness	27.5 mg/kg (50 mg/kg NaF)	Food
Pattee et al. (1988)	Screech owls		No effect level for reproduction	56.5 mg/kg	Food
Hoffman et al. (1985)	Screech owls		No effect level for reproduction	56.5 mg/kg	Food
Pattee et al. (1988)	Screech owls		Lower reproductive success	56.5 mg/kg	Food
Stratus Consulting (2000)	Eastern screech owls	*Otus asio*	Reduced number of eggs per hatch; reduced hatching success	200 mg/kg	Food
Nahorniak et al. (1983)	Turkeys		No effect on survival and growth	203 mg/kg	Food
Pattee et al. (1988)	Screech owls		Decrease in fertile eggs and young per clutch	232 mg/kg	Food
Hoffman et al. (1985)	Owls		Smaller eggs and hatchlings	232 mg/kg	Food
Bird and Massari (1983)	American kestrels		Mortality from fluorosis	226 mg/kg	Food
NAS (1974)	Browing or broiler chicken		Dietary tolerance	300 mg/kg	Food
Fleming (1996)	European starlings		No effect level on reproduction	360 mg/kg	Food
NAS (1974)	Laying or breeding hen		Dietary tolerance	400 mg/kg	Food
NAS (1974)	Turkey		Dietary tolerance	400 mg/kg	Food
Nahorniak et al. (1983)	Turkeys		Decrease in body weight	400 mg/kg	Food
Guenter and Hahn (1986)	Chicken		No effect on survival, growth, reproduction	700 mg/kg	Food
Chan et al. (1973)	Japanese quail		Lowest effect level on survival and bone strength	750 mg/kg	Food
Chan et al. (1973)	Japanese quail		Increased mortality, lower weights	750 mg/kg	Food
As reviewed by Weinstein and Davison (2004)	Male chicks		Lower weight gains	800 mg/kg	Food
As reviewed by Weinstein and Davison (2004)	Chicken		No effect on growth	800 mg/kg	Food
Rogler and Parker (1972)	Chicken		Lowest effect level on growth	850 mg/kg	Food
Rogler and Parker (1972)	Chicks		Lower weight gains	850 mg/kg	Food
Van Toledo and Combs (1984)	Chicken		No effect on growth and reproduction	972 mg/kg	Food
Guenter and Hahn (1986)	Leghorn hens		Decrease in body weight; lower egg weight; thinner eggshells	1,000 mg/kg	Food
Fleming (1996)	European starlings		Fewer hatchlings	1,080 mg/kg	Food

Table C-1. Fluoride toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Van Toledo and Combs (1984)	Leghorn hens		No effects on body weight, egg fertility, chick weight	1,200 mg/kg	Food
Van Toledo and Combs (1984)	Leghorn hens (HES genetic strain)		Decrease in hatching success	1,200 mg/kg	Food
Fleming (1996)	Snow geese		Toxicity	6.8–25 mg/kg	Tissue (brain)
Fleming et al. (1987)	Japanese quail	*Coturnix coturnix japonica*	No detrimental effect on growth	13–2,223 mg/kg (ash weight)	Tissue (tibia)
Fleming (1996)	Snow geese		Toxicity	32–129 mg/kg	Tissue (liver)
Vikoren and Stuve (1996)	Herring and common gulls		Increased concentraion in eggshell	NA	Tissue (femur)
Fleming et al. (1987)	European starlings (16-days old)	*Sturnus vulgaris*	Reduced growth rate	13 mg/kg	Dose
Fleming et al. (1987)	European starlings (16-days old)	*Sturnus vulgaris*	Death	17 mg/kg (LD50)	Lethal dose
Fleming et al. (1987)	European starlings (1-day old)	*Sturnus vulgaris*	Death	50 mg/kg (LD50)	Lethal dose

Note: Original unit concentrations in parentheses.

Concentrations for fluoride compounds in the original reports were converted to just the fluoride fraction (original fluoride compound concentrations in parentheses).

Table C-2. PAH toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Chemical	Concentration	Media
Plants						
Kipopoulou et al. (1999)	Vegetation	NA	Vegetation - soil concentration ratios	PAH	0.002–0.64 (ratio)	Ambient soil
Leyval and Binet (1998)	Mycorrhizal plants	NA	Survival	PAH	5 g/kg	Controlled soil
Wittig et al. (2003)	Poplar	*Populus nigra*	Transpiration rate	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Nutrient uptake	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Water content in leaves	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Dry weight of roots	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Dry weight of shoots	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Dry weight of leaves	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Shoot growth	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Leaf growth	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Chlorophyll content decrease	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Carotenoid content	PAH	10 mg/kg	Controlled soil and water
Wittig et al. (2003)	Poplar	*Populus nigra*	Net photosynthesis	PAH	10 mg/kg	Controlled soil and water
Joner and Leyval 2001	Clover and ryegrass		Decrease in root length density	PAH	500 mg/kg of anthracene and chrysene, 50 mg/kg dibenzo[a,h]anthracene in soil	Controlled soil
Leyval and Binet (1998)	Ryegrass	NA	50% decrease in dry weight	PAH	5,000 mg/kg	Controlled soil
Invertebrates						
Jager et al. (2000)	Earthworm	*Eisenia andrei*	Maximum biota-soil accumulation factor	BAP	2.4 kg/kg lip.	Controlled soil
Jager et al. (2000)	Earthworm	*Eisenia andrei*	Maximum biota-soil accumulation factor	Pyrene	3.9 kg/kg lip	Controlled soil
Jager et al. (2000)	Earthworm	*Eisenia andrei*	Maximum biota-soil accumulation factor	Phenanthrene	7.3 kg/kg lip	Controlled soil
Jager et al. (2000)	Earthworm	*Eisenia andrei*	Maximum biota-soil accumulation factor	Fluoranthene	8.2 kg/kg lip	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Acridine	25 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Phenanthrene	25 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Acridine	26 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Fluorene	28 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Dibenzothiphene	29 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Pyrene	29 mg/kg dry weight	Controlled soil

Table C-2. PAH toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Chemical	Concentration	Media
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Dibenzofuran	30 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Phenanthrene	31 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Carbazole	31 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Fluorene	31 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Dibenzothiphene	33 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Carbazole	35 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Dibenzofuran	36 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Pyrene	38 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Dibenzothiphene	44 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Fluorene	50 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Carbazole	54 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Dibenzofuran	61 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Fluorene	68 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Pyrene	71 mg/kg dry weight	Controlled soil
Kapustka (2004)	Springtail	*Folsomia candida*	NOAEC	Phenanthrene	75 mg/kg	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Dibenzofuran	78 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Phenanthrene	94 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	NOEC growth	Fluroanthene	98 mg/dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Carbazole	106 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC10 growth	Fluroanthene	113 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Acridine	125 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Dibenzothiphene	133 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Phenanthrene	134 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Pyrene	155 kg /kg lip	Controlled soil
Brown et al. (2004)	Earthworm	*Lumbricus rubellus*	Cocoon production reduction	PAH	160 mg/kg	Soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	EC50 lethality	Fluroanthene	166 mg/kg dry weight	Controlled soil
Kapustka (2004)	Earthworm		LC50	PAH	173 mg/kg	Soil
Kapustka (2004)	Springtail	*Folsomia candida*	MATC and EC50	Phenanthrene	175 mg/kg dry weight	Controlled soil
Kapustka (2004)	Springtail	*Folsomia candida*	Reproduction	Phenanthrene	220 mg/kg dry weight	Controlled soil
Brown et al. (2004)	Earthworm	*Lumbricus rubellus*	LC50 survival	PAH	283 mg/kg	Controlled soil
Kapustka (2004)	Springtail	*Folsomia candida*	Mortality	Phenanthrene	380 mg/kg	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Acridine	863 mg/kg dry weight	Controlled soil
Sverdrup et al. (2002)	Earthworm	*Eisenia veneta*	LC50 lethality	Fluroanthene	416 mg/kg dry weight	Controlled soil
Brown et al. (2004)	Earthworm	*Lumbricus rubellus*	LC50 survival	PAH	0.0068 mg/mL	Contact
Kapustka (2004)	Earthworm		LC50	PAH	171 μg/cm^2	Contact
Bonnet et al. (2005)	Eukaryotic microorganisms	*Saccharomyces cerevisia*	IC50 72 hour growth inhibition	Anthracene	20.44 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Rhodotorula glutinis*	IC50 72 hour growth inhibition	Anthracene	21.5 mg/kg	Controlled water/substrate

Table C-2. PAH toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Chemical	Concentration	Media
Bonnet et al. (2005)	Eukaryotic microorganisms	*Chaetomium elatum*	IC50 72 hour growth inhibition	Anthracene	21.75 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Aspergillus terreus*	IC50 72 hour growth inhibition	Anthracene	22.17 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Absidia fusca*	IC50 72 hour growth inhibition	Anthracene	24 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Cunninghamella elegans*	IC50 72 hour growth inhibition	Anthracene	25.27 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Phanerochaete chrysosporium*	IC50 72 hour growth inhibition	Anthracene	28.66 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Fusarium solani*	IC50 72 hour growth inhibition	Anthracene	27.35 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Paenicillium chrysogenum*	IC50 72 hour growth inhibition	Anthracene	30.34 mg/kg	Controlled water/substrate
Bonnet et al. (2005)	Eukaryotic microorganisms	*Cladosporium herbarum*	IC50 72 hour growth inhibition	Anthracene	66.8 mg/kg	Controlled water/substrate
Kapustka (2004)	Earthworm	*Eisenia fetida*	LOAEC	Benzo[a]pyrene	26 mg/kg dry weight	Food
Kapustka (2004)	Potworm	*Enchytraeus crypticus*	LOAEC	Benzo[a]pyrene	26 mg/kg dry weight	Food
Port et al. (1998)	Silkworms		Growth retardation	Alx(SiF6)3	30 μg/g	Food
Kapustka (2004)	Springtail	*Folsomia candida*	LOAEC	Phenanthrene	100 mg/kg	Food
Kapustka (2004)	Isopod	*P. scaber*	No effects on growth	Benzo[a]anthracene	285 mg/kg	Food
Kapustka (2004)	Isopod		No adverse effects	Benzo[a]pyrene	315 mg/kg dry soil	Food
Kapustka (2004)	Potworm	*Enchytraeus crypticus*	NOAEC	Fluorene	1212 mg/kg	Food
Bonnet et al. (2005)	Eukaryotic microorganisms	*Tetrahymena pyriformis*	Inhibition of cell proliferation rate IC50	Anthracene	33.4 mg/L	Controlled water
Kapustka (2004)	Isopod	*O. asellus*	LOAEC weight	Benzo[a]anthracene	28.5 mg/kg	NA
Mammals						
Kapustka (2004)	Unspecified mammals		TRV	PAH	1.25 mg/kg day	
Birds						
Hough et al. (1993)	Pigeons		TRV	Benzo[a]pyrene	10 mg/kg	Intramuscular

Table C-3. Sulfur dioxide toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Plants					
Ozolincius et al. (2005)	Trees	*Fraxinus excelsior; Picea abies; Betula* sp.	Crown defoliation	0.3–3.8 $\mu g/m^3$	Ambient air
McCune (1988)	Lichen		Cellular and ultrastructural SO_2	5–10 $\mu g/m^3$	Controlled air
Salemaa et al. (2004)	Lichen	Lichen and bryophyte	Plant species sequence from smelter	5–10 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	Pine tree		Necrosis of needles	28 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	Spruce	*Picea abies*	Chronic and acute injury	25–40 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	White pine	*Pinus strobus*	Reduction in growth	45 $\mu g/m^3$	Ambient air
Steiner (1995)	Tardigrade	*Tylocephalus auriculatus,*	Presence and persistence under fumigation for 28 months	47 $\mu g/m^3$	Controlled air
Liblik and Pensa (2001)	Lichen	*Sphagnum*	Complete degradation	60–250 $\mu g/m^3$	Ambient air
Hur and Wellburn (1993)	Water fern	*Azolla*	Growth retardation	68 mg/m^3 (25 nl/L)	Controlled air
Hur and Wellburn (1993)	Water fern	*Azolla*	Decrease in intracellular NH3	68 mg/m^3 (25 nl/L)	Controlled air
Hur and Wellburn (1993)	Water fern	*Azolla*	Destroys chlorophyll	68 mg/m^3 (25 nl/L)	Controlled air
Nieboer and Richardson (1981)	Lichen	*Usnea hirta*	Decrease in respiration rates	86 $\mu g/m^3$	Ambient air
As reviewed by Palmason and Magnusson (1998)	Various trees		Reduction in growth	56 $\mu g/m^3$	Ambient air
Dueck and Elderson (1992)	Heathland plants	*A. capillaris*	Reduce root growth	90 $\mu g/m^3$	Ambient air
Dueck et al. (1990/1991)	Scots pine	*P. sylvestris*	Electrolyte leakage in shoots	92 $\mu g/m^3$	Controlled air
Batty et al. (2003)	Lichen	*Parmelia saxatilis*	Absence	>100 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	Pine tree		Mortality	196–224 $\mu g/m^3$	Ambient air
Lavola (1998)	Birch trees	*Betula pendula and B. resinifera*	Reduced amount of anitoxidative phytochemicals; reduced resistance	200 $\mu g/m^3$	Controlled air
Nieboer and Richardson (1981)	Lichen	*Parmelia chlorochroa*	Decrease in respiration rates	246 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	Grasses	*Dactylis glomerata, Festuca rubrea, Holcus*	Tolerance	250–500 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	White pine	*Pinus strobus*	Necrotic lesions; short term	280 $\mu g/m^3$	Ambient air
Palmason and Magnusson (1998)	Douglas fir	*Pseudotsuga menziesii*	Necrosis; short term	812 $\mu g/m^3$	Ambient air
Richardson and Nieboer (1983)	Lichen	*Parmelia and Lecanora* spp.	Respiration	<1,310 $\mu g/m^3$	Controlled air
Richardson and Nieboer (1983)	Lichen	*Lobaria and Parmelia* spp.	Respiration and photosynthesis	<1,310 $\mu g/m^3$	Controlled air
Richardson and Nieboer (1983)	Lichen	*Physcia* spp.	Photosythnesis	2,500 $\mu g/m^3$	Controlled air
Marques et al. (2005)	Lichen	*Parmelia sulcata*	Cell membrane damage; leakage of k; lichen vitality	Various	Ambient air

Table C-3. Sulfur dioxide toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Richardson and Nieboer (1983)	Lichen	*Cladonia rangiferina*	K efflux	14,600 $\mu g/m^3$	Controlled air
Shaw et al. (1993)	Scots pine	*Pinus sylvestris*	Foliage necrosis	<0.01 mg/kg	Ambient air
Freer-Smith and Taylor (1992)	Silver birch	*Betula pendula*	Photosynthesis inhibition	0.070 mg/kg	Controlled air
Freer-Smith and Taylor (1992)	Scots pine	*Pinus sylvestris*	Photosynthesis inhibition	0.075 mg/kg	Controlled air
Sanz et al. (1992)	Lichen	*E. prunastri*	Reduced photosynthesis	0.1 mg/kg	Controlled air
Steiner (1995)	Moss		Bleaching of leaves; pH decreased from aboout 7.5 to 5	0.225 mg/kg	Controlled air
Sanz et al. (1992)	Lichen	*R. fraxinea*	Reduced photosynthesis	0.5 mg/kg	Controlled air
Kong et al. (1999)	Lichen	*Xanthoparmelia mexicana*	Chlorophyll content decrease	0.5 mg/kg	Controlled air
Palmason and Magnusson (1998)	Grasses	*Agrostis capillaris, Nardus stricta, Lolium*	No effects on growth	100 $\mu g/m^3$ (37 nL/L)	Controlled air
Newsham et al. (1992)	Fungi		Fungal colony decreases	0.06 $\mu L/L$	Ambient water on leaf litter
Belandria et al. (1989)	Lichen	*Xanthoria parientina; P. distorta*	Inhibited spore generation (slight)	3.2 mg/L (50 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*Xanthoria parientina*	Spore germination inhibition	3.2 mg/L (50 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*P canina*	50% less germination	3.2 mg/L (50 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*P. canina*	Spore germination inhibition	3.2 mg/L (50 μM)	Controlled solution
Marti (1983)	Lichen	*Menegazzia terebrata, Usnea florida, Parmelia sinuosa*	Photosynthesis inhibition	16 mg/L (0.25 mM)	Contolled water
Belandria et al. (1989)	Lichen	*Xanthoria parientina; P. distorta*	Complete spore generation inhibition	32 mg/L (500 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*P canina*	Complete loss of germination	32 mg/L (500 μM)	Controlled solution
Belandria et al. (1989)	Lichen	*L. conizaeoides*	50% loss germination	64 mg/L (1,000 μM)	Controlled solution
Nash and Gries (2002)	Lichen		Structural changes	0.05–1.0 mg/kg	NA
Nash and Gries (2002)	Lichen	*Evernia mesomorpha*	Decrease in protein and lipid biosynthesis and CO_2 fixation	0.31–0.1 mg/kg	NA
Richardson and Nieboer (1983)	Lichen	*Parmelia, Usnea and Lobaria*	Distribution	20–50 $\mu g/m^3$	NA
Palmason and Magnusson (1998)	Grasses	*Cladina rangifera*	Threshold level at which injury is produced	20–30 $\mu g/m^3$	Predicted from simulation model
Richardson and Nieboer (1983)	Lichen	*Parmelia squarrosa*	Chlorophyll a	21 $\mu g/m^3$	NA
Richardson and Nieboer (1983)	Lichen	*Usnea hirta*	Respiration	<47 $\mu g/m^3$	NA
Richardson and Nieboer (1983)	Lichen	*Cladonia spp and Cetraria cucullata*	Photosynthesis	<170 $\mu g/m^3$	NA
Richardson and Nieboer (1983)	Lichen	*Usnea hirta*	Respiration	<246 $\mu g/m^3$	NA

Table C-3. Sulfur dioxide toxicity effects

Citation	Receptor	Scientific Name	Effect/Endpoint	Concentration	Media
Shaw et al. (1993)	White pine	*Pinus strobus*	Foliage necrosis	0.03 mg/kg (30 ppb)	NA
Newberry (1974)	Corticolous lichens		Bleached chlorophyl, permanent plasmolysis and chloroplasts brown spots	5 mg/kg	NA
Invertebrates					
Steiner (1995)	Nematodes	*C. cf. andrassyi*	Decrease in abundance	0.025 mg/kg	Controlled air
Steiner (1995)	Tardigrade		Survived SO_2 experiement	0.025–0.075 mg/kg	Controlled air
Steiner (1995)	Nematodes	*Plectus acuminatus*	Survived SO_2 experiement	0.225	Controlled air
Mammals					
Oehme et al. (1996)	Dogs		No pulmonary function effects	0.5 mg/kg	NA

Note: Original unit concentrations in parentheses.