RAMAS

Risk Assessment and Risk Management
Procedure for Arsenic in the Tampere Region

Risk Assessment of Natural and Anthropogenic Arsenic in the Pirkanmaa Region, Finland

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ABSTRACT

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Arsenic (As) is a known human toxicant and in the environment its adverse effects on biota are diverse. To assess the risks of environmental arsenic to human beings and biota, we carried out case-specific, quantitative human health risk assessments (HRA) and ecological risk assessments (ERA). These risk assessments were focused on the specific site types previously identified in RAMAS –project. In the study area, Pirkanmaa region, such site types included former wood treatment plants which had used the Copper-Chromium-Arsenate -chemical (CCA), mine sites and areas with high level of natural arsenic in soil or groundwater.

The ecological risk assessment followed a tiered approach. In tier 0, the environmental concentrations of arsenic were compared with various ecological benchmark values. The results were used to identify the key organisms and specific site types on which we should focus. In tier 1, uptake and exposure models were used to assess As intake by the key organisms (earthworms, plants, shrews and birds). The concentration and dose estimates were divided by suitable benchmarks such as, No Observed Adverse Effect Levels (NOAEL), to derive deterministic risk estimates (hazard quotients, HQs). In tier 2 we ran probabilistic calculations with the CrystalBall© –software. In addition, we carried out ecotoxicity tests with soil samples and soil leachates. The results from the assessment based on chemical studies and those from ecotoxicity tests were combined to give two separate total risk scores. We derived these risk scores by using the calculation rules of the TRIAD methodology. The assessment of the risks to aquatic biota was mainly based on the comparison of As concentrations with the ecological benchmark values for surface waters and sediments. In addition, we carried out a survey on the diatom species in the sediment affected by the effluents from the Ylöjärvi mine site.

The health risk assessment was based on the determination of the average daily dose of inorganic As in different exposure scenarios. In the phase 1 HRA the data available before RAMAS –project were used as input values. In phase 2, we included the data produced in RAMAS –project. Exposure scenarios studied included exposure to water from drilled wells and dug wells, exposure in a residential area built on a former CCA-plant site and occupational exposure to air dust in the Ylöjärvi mine site. The estimated daily doses were compared with different acceptable daily intake values to produce deterministic risk estimates. Excess life-time cancer risks were determined on the basis of unit risk values. We also carried out a probabilistic assessment based on Monte Carlo simulation using the CrystalBall© –software. To verify the exposure calculations, we ran a biomonitoring study, in which the urinary As concentrations of 40 volunteers within Pirkanmaa were analysed. In addition, the incidences of cancer types associated with As exposure were determined.

The results from ERA show that all site types studied pose a significant risk to some terrestrial organisms. The total risk scores derived on the basis of chemical studies varied between 0.56 (sites with naturally occurring As) and 0.98 (Ylöjärvi mine site). These scores refer to the mean values of the samples representing the specific site type studied. The corresponding risk scores based on ecotoxicity studies were 0.37 and 0.46 with the highest risk score (0.82) associated with the former CCA-plant of Kauttu. The risk scores derived from the ecotoxicity tests are more reliable than those based on chemical studies since the high values of the latter mainly arise from the high total concentrations of arsenic. The HQ –values related to risks to wetland biota in the surroundings of the Ylöjärvi mine site varied between 16 and 410 showing at least moderate risks. Further away in lake Näsijärvi, the risks are expected to remain insignificant (max. HQ = 20).

The results from the HRA show considerable health risks if water from drilled wells is used as drinking water. This risk is highest in the southern part of the study area. Inclusion of background exposure (food) and exposure to As in soil resulted in an average total daily dose of 0.16-55 µg/kg/d mean value being 0.68 µg/kg/d (median 0.27). The acceptable daily intake values vary between 0.3 and 1.0 µg/kg/d, and hence, the highest estimate exceeded the lowest acceptable level 180-fold. The maxmum life time excess cancer risk was estimated to be $8.3*10^{-2}$. According to the statistical analysis, some 5.9 – 46 % of the population would be experiencing unacceptably high exposure from well water. Risks associated with the dug well waters would mainly be insignificant. At CCA-plant sites the maximum excess cancer risk was estimated to be $2.3*10^{-3}$. Hence, exposure to As in CCA plant sites may significantly add to the total risk particularly in the case of small children (age 1-6). The biomonitoring study confirmed the exposure to arsenic in well water. Moreover, both the total As concentration and the concentration of inorganic As in urine correlated well with the As concentration in the well water ($R^2 = 0.95$ ja $R^2 = 0.83$). The regional-scale epidemiological study showed elevated incidences of liver cancer compared with the reference area. In addition, it seemed that there is a higher incidence of bladder, skin and kidney cancers, however only part of the risk ratios determined were statistically significant. Overall, the results from the epidemiological study need to be interpreted with caution since there are several uncertainties involved.

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TIIVISTELMÄ

Sorvari, J., Schultz, E., Rossi, E., Lehtinen, H., Joutti, A., Vaajasaari, K. & Kauppila, T. Risk assessment of natural and anthropogenic arsenic in the Pirkanmaa region, Finland. Geologian tutkimuskeskus, Erikoisjulkaisut, 126 sivua, 22 kuvaa, 47 taulukkoa ja 4 liitettä.

Arseeni (As) on tunnetusti ihmiselle myrkyllinen aine ja ympäristössä sen haittavaikutukset eliöihin voivat olla moninaiset. Tässä työssä arvioitiin ympäristön arseenista aiheutuvia, ihmisiin kohdistuvia terveysriskejä ja eliöstöön kohdistuvia ekologisia riskejä. Arvioinnissa keskityttiin muutamiin Pirkanmaan alueelta valittuihin tyyppikohteisiin, joissa oli korkeita arseenipitoisuuksia. Kohteina olivat entiset CCA-kyllästämöt, kaivosalueet ja luontaisesti korkeita maaperän ja pohjaveden As-pitoisuuksia sisältävät alueet.

Ekologinen riskinarviointi perustui portaittaiseen menettelytapaan. Ensimmäisessä vaiheessa verrattiin ympäristöstä mitattuja arseenipitoisuuksia ekologisiin viitearvoihin. Näiden perusteella tunnistettiin keskeisimmät kohde-eliöt ja tyyppikohteet, joihin keskityttiin tarkennetussa arvioinnissa. Seuraavaksi arvioitiin kohde-eliöiden altistumista maaperän arseenille käyttäen altistumista ja kertymistä kuvaavia laskentamalleja. Arvioitujen eliöstön arseenipitoisuuksien ja -annosten perusteella laskettiin vaaraosamäärät (HQ) vertaamalla niitä kirjallisuudessa esitettyihin turvallisiin tasoihin. Tarkasteltavina olivat lierot, kasvit, päästäiset ja linnut. Lisäksi tehtiin tilastollinen tarkastelu (probabilistinen arviointi) käyttäen CrystalBall© -tietokoneohjelmaa. Laskennallisen arvioinnin lisäksi tutkittiin maanäytteiden ja niiden vesiuutteiden myrkyllisyyttä ekotoksisuustestein.. Pitoisuuksiin perustuvan laskennallisen arvioinnin tulokset ja toksisuustestien tulokset yhdistettiin erillisiksi riskiluvuiksi ns. TRIAD-menettelyn mukaisesti. Vesistövaikutuksia arvioitiin vertailemalla mitattuja pitoisuuksia vesistön ekologisiin viitearvoihin. Ylöjärven kaivosalueen vaikutusalueella tutkittiin myös järvisedimentin piilevien lajikoostumusta.

Terveysriskien arviointia varten laskettiin ihmisten keskimääräinen altistuminen epäorgaaniselle arseenille eri altistustilanteissa. Arvioinnin ensimmäisessä vaiheessa käytettiin ennen RAMAS-projektia julkaistua tietoa, toisessa vaiheessa näitä täydennettiin RAMAS-projektissa hankituilla lisätiedoilla. Tärkeimmiksi. altistustilanteiksi katsottiin pora- ja kuilukaivojen veden käyttö talousvetenä, entisten kyllästämöalueiden vaikutusalueilla asuminen sekä työperäinen altistuminen hengitysteitse Ylöjärven kaivosalueen pölylle. Saatuja altistusarviota verrattiin edelleen eri tahojen esittämiin sallittuihin saantiarvoihin. Lisäksi määritettiin lisäsyöpäriski kertomalla saantiarvio yksikkösyöpäriskin arvolla. Deterministisen arvioinnin lisäksi tehtiin tilastollinen tarkastelu (probabilistinen arviointi) käyttäen CrystalBall –laskentaohjelmaa. Laskennallista altistusarviota täydennettiin biomonitorointitutkimuksella, jossa määritettiin arseenipitoisuudet ihmisten virtsanäytteistä ja pienepidemiologisella tutkimuksella, jossa määritettiin arseenialtistumiseen liitettyjen syöpäsairauksien esiintymistiheys Pirkanmaalla.

Tulosten perusteella kaikista tarkastelluista arseenin tyyppikohteista aiheutuisi merkittävä riski jollekin eliölajille. Pitoisuuksiin perustuvat, eri maanäytteiden keskiarvoa vastaavat riskiluvut vaihtelivat välillä 0,56 (luontaisesti korkea As) ja 0,98 (Ylöjärven kaivosalue). Ekotoksisuuden perusteella määritellyt riskiluvut olivat vastaavasti 0,37 ja 0,46, ja korkein riskiluku (0,82) saatiin Kautun entisen kyllästämöalueen maaperälle. Ekotoksisuustestien tuloksia voidaan pitää luotettavampina, sillä syynä suuriin kemiallisiin tutkimuksiin perustuviin riskilukuihin olivat etupäässä arseenin kokonaispitoisuuksiin perustuvat laskelmat. Kaivosalueen läheisessä kosteikossa sedimenttieliöille lasketut HQ-arvot vaihtelivat välillä 16 ja 410, mikä osoittaa riskien olevan vähintään kohtalaiset. Kauempana Näsijärvessä riskit jäänevät kuitenkin merkityksettömiksi, sillä suurimmaksi HQ-arvoksi saatiin 20.

Tulosten mukaan arseenialtistuksesta aiheutuu huomattava terveysriski käytettäessä porakaivojen vettä juomavetenä. Kun otettiin mukaan tausta-altistus ravinnostaja maaperästä, saatiin keskimääräiseksi kokonaispäivittäisannokseksi 0,16-55 µg/kg/d keskiarvon ollessa 0,68 µg/kg/d (mediaani 0,27). Turvallisiksi katsotut päivittäisannokset vaihtelevat välillä 0,3 ja 1,0 µg/kg/d, joten sallittu keskimääräinen päivittäisannos ylittyi maksimissaan 180-kertaisesti. Suurimmaksi lisäsyöpäriskitasoksi saatiin $8.3*10^{-2}$. Tilastollisen analyysin perusteella arviolta 5,9 – 46 %:lla väestöstä altistuminen juomaveden arseenille ylittäisi sallitun rajan. Kuilukaivojen vettä käyttävillä riskit olisivat pääsääntöisesti merkityksettömiä. Kyllästämöalueilla lisäsyöpäriski olisi suurimmillaan $2.3*10^{-3}$. Altistuminen kyllästämöalueilla voisi aiheuttaa merkittävän lisäriskin etenkin leikki-ikäisille lapsille. Biomonitorointitutkimus vahvisti altistumisen porakaivoveden arseenille. Virtsan kokonaisarseeni ja epäorgaaninen arseeni korreloivat hyvin veden As-pitoisuuden kanssa (R^2 = 0,95 ja R^2 = 0,83). Pienepidemiologinen tutkimus osoitti etenkin maksasyövän osalta kohonnutta syöpätapausten määrää suhteessa vertailualueeseen. Saatiin myös viitteitä virtsarakon syövän, ihosyövän ja munuaissyövän runsaammasta esiintymisestä, mutta kuitenkin vain osa tuloksista oli tilastollisesti merkitseviä. Syöpärekisteriotannan tuloksia tuleekin tulkita varoen johtuen useista epävarmuustekijöistä.

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PREFACE

RAMAS is a three-year project (2004 - 2007) funded by the participating organizations and the LIFE ENVIRONMENT –programme, by the beneficiary, Geological Survey of Finland (GTK), and by the following partners: Helsinki University of Technology (TKK), Pirkanmaa Regional Environment Centre (PIR), Finnish Environment Institute (SYKE), Agrifood Research Finland (MTT), Esko Rossi Oy(ER) and Kemira Kemwater (Kemira).

The acronym RAMAS comes from the project title "Risk Assessment and risk Management procedure for ArSenic in the Tampere region". Spatially, the project covers the whole Province of Pirkanmaa (also called the Tampere region) comprising 33 municipalities (in 2005), and 455 000 inhabitants within its area. The number of municipalities decreased to 28 in January 2007 while the number of inhabitants reached 469 000. Tampere, Finland's third largest city, is the economic and cultural centre of the region.

The project aims to identify various sources of arsenic in Pirkanmaa, to produce an environmental risk assessment (covering human health risk assessment and ecological risk assessment) for the region and to present recommendations for the management of risks. This project is the first in Finland to create an overall, large-scale risk management strategy for a region that has natural and anthropogenic contamination sources.

The project is divided into logically proceeding tasks having responsible Task Leaders who coordinate the work within their tasks:

1. Natural arsenic sources (GTK) Birgitta Backman
2. Anthropogenic arsenic sources (PIR), Kati Vaajasaari until 30.4.2006; Ämer Bilaletdin since 1.5.2006
3. Risk assessment (SYKE), Eija Schultz
4. Risk management (SYKE), Jaana Sorvari
5. Dissemination of results (TKK), Kirsti Loukola-Ruskeeniemi
6. Project management (GTK), Timo Ruskeeniemi

The project produces a number of Technical Reports, which are published in a special report series by GTK. Each report will be an independent presentation of the topic in concern. The more comprehensive conclusions will be drawn in the Final Report of the RAMAS project which summarizes the project results. Most reports will be published in English with summaries in Finnish. A cumulative list of the reports published so far is presented in the back cover of each report. All documents are also downloadable in the project's home page: www.gtk.fi/projects/ramas.

ABSTRACT
TIIVISTELMÄ
PREFACE

1 INTRODUCTION

Arsenic (As) is an element occurring naturally in the environment. It is widely distributed and present in trace quantities in all environmental compartments. Even meteorites have been reported to contain variable concentrations (0.0005 – 0.1 %) of arsenic and hence, As probably occurs throughout the universe (Merck Index, 1996). Regional and local anomalies with high As concentrations in bedrock and soil have been found around the world.. Human activities have also released arsenic to the environment generating contaminated areas with extremely high As concentrations in different environmental media.

There is no general agreement whether arsenic is essential to organisms or not. Several adverse effects have been described and there are plenty of publications and reviews, particularly concerning human toxicity. The acute toxicity of arsenic has actually been recognized since centuries: "During the Middle Ages, professional poisoners sold their services to royalty and the common populace" and toxic metals such as arsenic was among the most common poisons. The murderous use of arsenic trioxide, "white arsenic", became so widespread that arsenic acquired the name "inheritance powder" (Poklis, 1996).

Development of techniques for ecotoxicological research and chemical analysis has resulted in numerous new aspects relevant from the viewpoint of the As-related effects on biota e.g., toxicity associated with organic arsenicals, long-term exposure to low doses, geochemical and biogeochemical cycles of arsenic in soil etc. Introduction of molecular and cell biological methods has also contributed to the elucidation of toxicity mechanisms (genotoxicity, immunotoxicity etc).

This report presents the results from the risk assessment task carried out within the RAMAS project. The risk assessment covers the assessment of risks to terrestrial and aquatic biota (ecological risk assessment, ERA) and to human beings (health risk assessment, HRA) associated with the specific risk sites in Pirkanmaa, i.e., sites with high levels of anthropogenic or natural arsenic. The identification of such areas was based on the previous work on environmental occurrence documented in other RAMAS reports (Backman *et al*. 2006; Parviainen *et al*. 2006; Mäkelä-Kurtto *et al*. 2007). The results from the ERA and the HRA were further combined with the data on the regional distribution of receptors to be protected and furthermore, in the identification of the high risk areas and of the potential risk management needs within the whole Pirkanmaa. The identification and planning of regional risk management actions belong to a separate task of RAMAS and hence, are reported separately (Lehtinen *et al*. in preparation).

2 ARSENIC IN THE ENVIRONMENT

2.1 Physicochemical properties

Arsenic is an element exhibiting both metallic and non-metallic characteristics with four potential oxidation states (–3, 0, +3 and + 5). In its non-metallic form the chemical behaviour of arsenic resembles that of phosphorus. Under pH 5-7 and oxidizing conditions As appears predominantly as pentavalent (arsenate, As^{5+}) oxyanions, while the trivalent (arsenite, As^{3+}) form is predominant under reducing conditions (e.g., WHO, 2001). However, various factors influence the ratio of As^{5+} to As^{3+} (e.g., Cullen & Reimer, 1989).

The occurrence and the environmental fate of different arsenic compounds depend on several abiotic and biotic factors such as pH, ionic and redox conditions, concentrations of metal sulphides and sulphide ion, temperature, salinity, and distribution and composition of biota. Depending on environmental conditions arsenic may undergo several reactions, e.g., oxidation, reduction, methylation, and demethylation. The pH of aqueous solutions appears to be the major factor in the relative stability of both tri- and pentavalent inorganic arsenic. The trivalent species is more readily oxidized in alkaline solution than at low pH. Pentavalent arsenic is relatively stable in neutral and alkaline conditions but is more readily reduced with decreasing pH (Goyer, 1996).

In soil, arsenic may exist in several geochemical forms depending on the characteristics of soil (Sarkar & Datta, 2004). Some arsenic species tend to adsorb on clay minerals and surfaces of organic material. The extent of adsorption affects the potential availability, which in turn results in different bioavailability and (eco)toxicological effects. Some samples collected from soil at unpolluted areas have shown that arsenic in soil is likely to occur mainly in pentavalent form (Cullen & Reimer, 1989). In marine water 80 % of total arsenic is expected to be in the form of arsenate, 10 % as arsenite and the rest bound in organic compounds. In surface water, besides arsenite and arsenate some methylated ions have been detected (Hanze, 1994). In the atmosphere the main arsenic species is the inorganic trioxide (As_2O_3) (WHO, 2001). Arsenic is released to the athmosphere primarily in the form of As_2O_3 or, less frequently, in the form of various gaseous organic compounds. Arsenic exists in air mainly bound in small particles ($D < 2\ \mu m$).

2.2 Occurrence in air, water and soil

In addition to being a naturally-occurring element, inorganic arsenic is released into the environment from a number of anthropogenic sources. Such sources include metal smelters, chemical manufacturers, sewage sludge, urban areas and traffic, mine tailings and fertilizers and pesticides. Previously, As was also used in the manufacturing of glass and ceramics. Due to restrictions on emissions and limitations and prohibition of the use of arsenic containing chemicals (see Lehtinen & Sorvari, 2006), the load to the environment and consequently, the environmental concentrations even in the vicinity of anthropogenic sources are expected to decrease. At present, the past extensive use of the chromate-copper-arsenate (CCA) chemical in wood preservation is still one of the major causes of As pollution in Finland .

Atmospheric arsenic may originate from both natural sources (volcanic eruption or forest fire) and anthropogenic activities (metal industry, smelters, burning of fossil fuel). In the vicinity of copper smelters As concentrations may exceed 1000 ng/m^3 (Goyer, 1996). In urban areas concentrations vary between of 3 and 200 ng/m^3 while considerably lower concentrations (0.02 - 4 ng/m^3) have been detected in at remote and rural areas (WHO, 2001).

Within Europe, the data on the concentrations of arsenic in air are limited. According to Mukherjee and Bhattacharya (2002) in Finland, the concentration of aerosol-form arsenic ranges from 0.46 to 0.75 ng/m^3. The monitoring studies carried out since 1996 by the Finnish Meteorological Institute show that in the northern part of Finland, the annual average backround concentrations have varied between 0.2 and 0.3 ng/m^3 (Alaviippola *et al.* 2007). At the same time, the concentration has been around 1 ng/m^3 in the capital city area between year 2000 and 2004. Hence, the effect of anthropogenic load is evident. Background concentrations in Pirkanmaa are expected to settle in between these background concentrations. The European Commission has set the lower assessment threshold level of 2.4 ng/m^3 (2004/107/EC) for the annual average As concentration in ambient air.

Equivalent to atmospheric heavy metals, arsenic is also monitored by analysing the concentrations in mosses. Carpet –forming species in particular are suitable indicators since they take nutrients directly from precipitation and dry deposition. A decreasing tendency that is, a change of concentration from 0.26 to 0.19 mg As/kg has been observed when *Hylocomium splendens* and *Pleurozium schreberi* were analysed for arsenic in Finland in 1995 and 2000 (Poikolainen & Piispanen, 2004). Unfortunately, it is not possible to arrive at exact quantitative information on concentrations in air on the basis of moss analyses since several factors such as climatic conditions (e.g., drought, rain, wind) affect the uptake.

Arsenic is widely distributed in freshwaters and typical concentrations in rivers and lakes are below 10 µg/l (WHO, 2001). In the Finnish nationwide survey carried out in 1990 the median concentration in 1160 samples taken from stream waters was 0.36 µgAs/l (range < 0.20 – 6.50 µgAs/l) with 25 % of the samples exhibiting concentrations below the detection limit (Tarvainen *et al*., 1997). According to the same report, lake water contained 0.33µg As/l (range 0.08 – 5.20 µgAs/l, number of samples = 152). In the oceans the concentrations vary between 1 and 2µgAs/l while in Baltic sea, a mean concentration of 0.76 µgAs/l has been measured in 1982-1983 (WHO, 2001). Groundwater concentrations range normally from 1 to 2 µgAs/l but in areas with volcanic rock and sulfide mineral deposits it can range up to 3 mgAs/l (3000 µg/l).

Arsenic is commonly found in the earth's crust included in more than 200 minerals. Depending on the source, concentrations of 1 – 3 mg/kg are considered as a typical average. However, upper background level can reach 40 mg/kg (WHO, 2001; Cullen & Reimer, 1989; Backman *et al*. 2006; Merck Index, 1996). The median concentration of arsenic in Finnish till fine fraction (<0.06 mm) is 2.6 mg/kg (Tarvainen, 2004).

2.3 Concentrations in biota

The chemistry of As in biological systems is not well-known (Langdon *et al.* 2003). A number of microbes (e.g., fungi and bacteria in soil and algae in water) are capable to transform inorganic As to organic forms i.e., methane-arsonic acid and dimethylarsinic acid (HSG 70, 1992). In biological systems arsenic has been analyzed mainly as the total As content. However, the recent development in analytical methods has improved the identification and quantification of different arsenic species in biological material.

Background concentrations in terrestrial biota are usually less than 1 mg As/kg (fresh weight). Arsenic levels are higher in biota collected near anthropogenic sources or in areas with geothermal activity. Although organic arsenic compounds are mainly found in marine organisms, they are also present in terrestrial organisms. (WHO, 2001)

Concentrations in terrestrial plants are usually less than 10 mgAs/kg (Matschullat, 2000). The accumulation of arsenic occurs via root uptake from soil or by adsorption of airborne arsenic deposited on the leaves. Concentrations in Finnish plants generally vary between 0.5 and 1.0 mg/kg (Kuusisto, 2004). In a Finnish long-term (56 d) laboratory experiment using Scots pine (*Pinus sylvestris*), birch (*Betula pendula*) and fescue (*Festuca ovina*) the As concentrations in roots, stems and leaves were less than 3 mg/kg in plants cultivated in uncontaminated soil, while growing in soil containing 5000 mg As/kg resulted in 773, 353 and 863 mg As/kg in roots, respectively (Turpeinen, 2002). In fact, plants growing in areas of very high concentrations of arsenic can develop resistance to it. This can lead to very high As concentrations in plant tissues and hence, pose a risk to herbivores feeding on them (HSG 70, 1992). Specialized plant communities tolerant to arsenic have developed on severely contaminated mine wastes. Some tolerant plants grow on wastes with total arsenic levels of several percent by weight. For example, Porter and Peterson (1975) reported mean concentrations 3000 - 6000 mg As/kg in plant tissue depending on plant species at arsenical mine sites. The biodiversity of plants is presumably low at such high environmental As concentrations.

Aquatic organisms bioaccumulate arsenic, but bioaccumulation factors are generally small compared with persistent and bioaccumulative toxicants (e.g., PCBs, methylmercury). Particularly the unicellular algae take up arsenate from the growth medium actively and consequently, act as the major source of arsenic for higher organisms in the aquatic food web. Algae is also used in soil amendment which may lead to As exposure of terrestrial organisms. Bioconcentration factors (BCF) of 3000 in algae have been reported (HSG 70,19929, while the values for fresh water invertebrates vary between 100 and 200 Nikunen *et al*. 2000). At contaminated sites, aquatic plants take up more arsenic than terrestrial plants (e.g. Mahimaijara *et al.* 2005). This might be due to the higher bioavailability compared with the bioavailability in soil rizosphere. The United States Environmental Protection Agency (USEPA) proposes the use the BSF value of 17 for arsenic (in the form of arsenite) when moving to trophic levels 3 and 4 while the food chain multiplier (FCM) of 1 should be applied (USEPA 1984, ref. in Sample *et al*. 1996). The low FCM indicates that arsenic does not biomagnify in foodwebs.

2.4 Human exposure to environmental arsenic

Human exposure to arsenic may include exposure to the inorganic or organic forms of arsenic or to both of these. Exposure may originate from air, soil, water and food items containing elevated concentrations of arsenic. Exposure routes comprise inhalation, ingestion and dermal intake (exposure through skin). In the general population, the primary route of exposure to

arsenic compounds is through ingestion. Food is considered the main contributor to total arsenic intake while in places where drinking water contains relatively high levels of arsenic, drinking water can be a significant source of arsenic intake. Other routes of exposure such as inhalation and dermal absorption are often considered to have only a minor or negligible contribution, except in the case of occupational exposure.

2.4.1 Arsenic in human diet

Uptake of arsenic by terrestrial plants may result in contamination of food items consumed by humans. Helgesen & Larsen (1998) studied the transfer of arsenic to carrots from soils with different level of contamination. The concentrations reached over 1 mg/kg dw. In carrots cultivated in uncontaminated soil (total arsenic in soil 6.5 ± 0.3 mg/kg dw) concentrations were <0.098 mg As/kg dw. Arsenic was present as inorganic As^{3+} and As^{5+}, no methylated forms were detected. The availability of arsenic to carrots was 0.47 ± 0.06 % (expressed as the ratio of total As in carrot to arsenic in soil) and 580 ± 150% (expressed as the ratio of As in carrot to extractable arsenic in soil). Arsenic concentrations were more than twice as high in the skin than in the core. The studies of Muñoz *et al.* (2002) with vegetables supported these findings showing decreasing As concentrations from root to shoots and further to fruits and 2-5 times higher As concentration on the skin compared to the core. In the study of Muñoz *et al.* (2002) over 90 % of the arsenic in vegetables was in inorganic form. Organic arsenicals (methylarsonic acid, dimethylarsinic acid) showed higher accumulation into shoots than inorganic arsenicals. Similar results have been reported in the case of tomato (Burló *et al.* 1999).

According to the study of Juhasz *et al.* (2006) rice and rice products can contain 0.07-0.76 mg As/kg in fresh weight. A few recent studies report high fractions, i.e., 85–95 %, of inorganic arsenic in rice and vegetables but differences between the products of different geographical origin are substantial. Williams *et al*. (2005) reported 64 % (+/- 1%, n = 7) of arsenic to be inorganic in European rice, while in Bangladesh or Indian rice the proportion is some 80 %. According to the study of Schoof *et al.* (1999), in the rice sold in the USA, approximately 24 % of As is in inorganic form.

Accumulation of elements by fungi has been studied in Sweden in the Forsmark area, where environmental conditions resemble those of our study area. The As concentrations in soil were low, i.e. 0.6 – 1.8 mg/kg dw (Johanson *et al*. 2004). As concentrations in fruitbodies were 0.2 – 7.8 (mean 1.6, median 0.88). Overall, the concentrations in mycelium and fruit bodies were only slightly higher compared to concentrations in bulk soil samples. Hence, arsenic was only moderately accumulated by mycelium (median CR =1.4, CR = ratio of concentration in fungi to concentration in soil, both given as mgAs/kg dw) as well as by fruit bodies (median CR = 1.3). No correlation was found between concentration of arsenic in soil and fungi.

In an Hungarian study, concentrations ranging from 0.05 to 146.9 mgAs/kg-dw were detected in the fruitbodies of mushrooms (Vetter *et al*. 2004). In As accumulating mushrooms As is mainly in organic form, but the chemical species vary between the taxa (Byrne *et al*. 1995)**.** The consumption of As-accumulating mushrooms only poses a low health risk owing to the facts that the consumed fresh fruit bodies contain about a tenth of the As level of the dried mushrooms, the majority of arsenic occurs in organic forms and the frequency of consumption is generally low.

Marine organisms can contain considerable amounts of arsenic, mainly in (harmless or less toxic) organic form i.e., as organoarsenicals (arsenobetaine, arsenocholine,

tetramethylarsonium salts, arsenosugars and arsenic-containing lipids), and typically in the form of arsenobetaine. In the recent UK study (FSA, 2005) the proportion of inorganic arsenic in fish varied from 0 (undetected) to 2.7 %, the average being 0.8 %. In shellfish inorganic arsenic proportion was higher: 0.3 – 13 % with the average of 5 %. The information on the fraction of inorganic arsenic was in good agreement with the study of Schoof *et al*. (1999).

The concentrations in marine fish and crustaceans generally vary between 2 and 20 mgAs/kg fw, arsenic being in organic form (HSG 70, 1992). However, concentrations ranging from 50 to 100 mg/kg have also been reported. In the environmental monitoring carried out in 2002 by the Finnish Environment Institute (SYKE), the average As concentration in the white muscle tissue of pike (*Esox lucius*) from Lake Pyhäjärvi in Tampere was 0.22 mg As/kg dw (n = 10) (Nakari *et al*., 2004. At the same time, concentrations in fish in Finnish lakes ranged from 0.01 to 1.3 mg/kg.)

Finnish foodstuffs typically contain only low concentrations of arsenic with the exception of Baltic fish species (Table 1). The concentration in milk is generally at the same level compared with other European countries. In the SCOOP report (Directorate-General Health and Consumer Protection 2004) mean arsenic content of milk in EU member states were: <0.005 mg/kg in Finland, 0.003 mg/kg in Gemany and 0.0004 mg/kg in the United Kingdom. Most of the arsenic in meat and poultry seem to be in less toxic organic form (Schoof *et al.* 1999) but some contradictory views have also been presented.

Table 1. Concentrations of arsenic in some food items reported in the literature studied.

	As, mg/kg fw	Range, mg/kg fw	Reference
cereal grains and grain products	< 0.05	0.005 - 0.285	Ref. in Mäkelä-Kurtto *et al*., 2006
cereal grains and grain products	< 0.05	0.03 – 0 20	Varo *et al*., 1980
wheat and rye flour	<0.02		
wheat flakes	< 0.05		
mushroooms, mycorrhizal species		< 0.02 – 0.11	Liukkonen-Lilja, 1996
mushroooms, mycorrhizal species[a]		< 0.03 – 0.22	Liukkonen-Lilja, 1996
mushroooms, saprotrophic species		< 0.02 – 0.52	Liukkonen-Lilja, 1996
milk powder	< 0.05		Varo *et al*.,1980; Liukkonen-Lilja, 1993
bovine and porcine meat	< 0.02		Nuurtamo etal., 1980
fresh-water fish		<0.03 - 0.53	Liukkonen-Lilja, 1993
Baltic salmon	0.072		Venäläinen *et al*., 2004
Baltic herring	0.39		Venäläinen *et al*., 2004
commercial fish products		1- 5.2	Liukkonen-Lilja, 1996
shrimps		11 - 19	Liukkonen-Lilja, 1996
potatoes		< 0.01 - 0.12	Varo *et al*., 1980
carrots	< 0.01		Liukkonen-Lilja, 1993
	0.06		Varo et al., 1980
	< 0.01		Liukkonen-Lilja, 1993
mushrooms[b]	0.42	0.14 – 5.11	Pelkonen *et al*., 2006

[a]mushrooms growing in the vicinity of a copper smelter
[b]12 edible species

Arsenic has also been found in breast milk, although only at low levels (Concha *et al*. 1998).

2.4.2 Intake estimates

Environment Canada has estimated the intake of inorganic arsenic from air, water, food, and soil for various age groups of the general population and for those living near point sources (Table 2) Even for inorganic arsenic, food represented the main source of exposure for all age groups, followed by ingestion of soil/dirt for infants and children, and intake of water and air.

Table 2. Estimated mean daily intake of inorganic arsenic (Environment Canada, 1993).

	Estimated daily intake (µg/kg body weight per day)				
Medium	0–0.5 years[a]	0.5–4 years[b]	5–11 years[c]	12–19 years[d]	Adult[e]
Water[f]	0.08	0.3	0.2	0.1	0.1
Food[g]	<0.04–2.4	<0.05–2.0	<0.03–1.9	<0.02–1.2	0.02–0.6
Air[h]	0.0003	0.0004	0.0004	0.0004	0.0003
Soil/dirt[i]	0.03–0.08	0.02–0.05	0.006–0.02	0.002–0.005	0.001–0.004
Total	0.1–2.6	0.3–2.4	0.2–2.1	0.1–1.3	0.1–0.7

[a]Weight 6 kg; inhalation 2 m^3 air/day; ingestion 0.1 L water/day and 35 mg soil/day
[b]Weight 13 kg; inhalation 5 m^3 air/day; ingestion 0.8 L water/day and 50 mg soil/day
[c]Weight 27 kg; inhalation 12 m^3 air/day; ingestion 1.1 L water/day and 35 mg soil/day
[d]Weight 55 kg; inhalation 21 m^3 air/day; ingestion 1.1 L water/day and 20 mg soil/day
[e]Weight 70 kg; inhalation 20 m^3 air/day; ingestion 1.5 L water/day and 20 mg soil/day
[f]Concentration in water estimated to be 5 µgAs/L in non-point source areas.
[g]Based on the assumption that 37% of intake from food is inorganic. No data available for the assessment of intake from breast milk.
[h]Concentration in air assumed to be 0.001 µg/m3 in non-point source areas.
[i]Range of inorganic arsenic in Canadian soil types is 4.8–13.6 mg/kg.

In the United States, food intake of arsenic has been estimated to range from 2 µg/day (infants) to 92 µg/day (60–65-year-old men) (Tao & Bolger, 1999). The average intake of inorganic arsenic ranges from 1.34 µg/day to 12.54 µg/day, respectively. The main dietary contribution to total arsenic comes from seafood (76–96%), except in the case of infants. In the diet of infants, the contribution of seafood and rice products is 42 and 31%, respectively. It is also noteworthy that in seafood, As is mainly present as arsenobetaine which is a non-toxic organic form of arsenic (see also chapter 2.4.1).

Adult dietary arsenic intakes reported for various countries range from 11.7 to 280 µg/day (Tao & Bolger, 1999). In some cases, other food items than those identified generally as important As sources may also have a significant contribution to the total exposure to arsenic. In the USA, Lasky *et al*. (2004) reported exceptionally high, i.e., 3- to 4-fold higher concentrations (mean 0.39 mg/kg) of total arsenic in young chickens compared with other poultry and meat, and arrived at human intake estimates of 1.38–5.24 µgAs/day (inorganic As) through the consumption of chicken alone.

Meliker *et al*. (2006) studied the intake of inorganic arsenic in south-eastern Michigan, USA, and concluded that arsenic in drinking water constitutes the major part of total intake and accounts for 55 % of the variance in the intake estimates. Food estimates explained 37 % of the variance, rice being the largest contributor. Water used for cooking and arsenic from smoking had only minimal contribution to the total intake.

In the Europe, estimates for the intake of arsenic from food items vary significantly between the member states of the European Union (Table 3). Owing to the scarcity of data it is difficult to derive exact intake values representative to Finland.

Table 3. Estimates of total daily arsenic intake from diet of the mean adult population in Denmark, Germany and United Kingdom (Directorate-General Health and Consumer Protection, 2004).

Food	Estimates of daily intake (µg/day)			
	Denmark	Germany	UK	Mean
Milk and dairy products	3.9	0.16	0.11	1.39
Fruits and vegetable	6.2	7.6	0.95	0.46
Cereals and bakery wares	8.3	9.4	2.0	6.57
Meat and offal	2.6	3.52	0.29	2.14
Fish	32.7	11.2	61	34.9
Bivalves, cephalopods, crustaceans		1.1		1.1
Eggs	0.2	0.17	0.01	0.13
Sweeteners		0.08	0.32	0.2
Salts and spice		0.17		0.17
Beverages	9.7[a]	3.3	1.0	4.67
Sum (rounded)	64	37	66	52

[a]Including drinking water

The studies (Table 3) show that in Denmark, Germany and UK the main As source in human diet is fish, followed by cereals and bakery wares. The data from Denmark and the UK indicate that fish and other seafood contribute more than 50% of the total arsenic to the daily diet. The estimate for the UK is in line with the results of the Food Standards Agency (FSA, 2004) which arrived at the estimates of 50 µg/d of total As and 0.9 – 5 µg/d of inorganic As.

No comprehensive study on the exposure to arsenic has been conducted in Finland. The National Public Health Institute has estimated that total intake of inorganic arsenic compounds from food is about 10 – 20 µg/d (KTL, 2006). On the other hand, in 2004 Finnish experts presented the mean estimate of 15 µg/d for arsenic intake from fish and milk with the majority (14 µg/d) originating from fish (Directorate-General Health and Consumer Protection, 2004). The rather low intake from fish is probably due to the primary consumption of fish from fresh and brackish water.

3 TOXIC ACTIONS OF ARSENIC

3.1 Bioavailability

The actual intake and uptake of arsenic from environmental media by humans and biota is governed by bioavailability that is, **the individual physical, chemical, and biological interactions that determine the exposure.** Because of the enormous diversity of organisms and differences in their physiology, the actual process of contaminant transfer into a cell - or factors that may impede or facilitate it - varies depending on the type of receptor. (National Research Council, 2003)

The key determinant of potential bioavailability of arsenic in both humans and biota is its capacity to be released from environmental matrices (e.g., Caussy, 2003). Most of the studies on bioavailability seem to have focused on the availability in human body and on the potential availability from environmental matrices. Hence, data on the bioavailability to wildlife is very

scarce. In addition to the speciation and environmental media (soil, water, sediment, air, food) involved, the route of exposure and individual propertied of the receptor affect the final bioavailability. Hence, it is quite obvious that the information on the total As content is not sufficient to evaluate the risks caused by environmental arsenic.

Turpeinen *et al*. (2003) measured bioaccessibility of arsenic using luminescent bacteria. They found a significant positive correlation between the concentration of water-soluble arsenic and the bioavailability to bacteria although it varied between sampling sites. Compared to the concentration of total water-soluble As, the bioavailable fraction varied from 3 to 77 % of the water-soluble As in soil.

Abundant absorption of pentavalent and trivalent soluble forms of ingested arsenic has been described with empirical animal models and verified in humans (Pomroy *et al*. 1980; Freeman *et al*. 1995). In humans, the absorption of 80-90 % from the gastrointestinal tract has been documented. Bioavailability of some inorganic species from food may even reach 100 %. Organic arsenic is less bioavailable than inorganic arsenic (e.g., Juhasz *et al*. 2006).

Two important factors are involved in human digestion of As-contaminated soil: solubility of the chemical in the digestive juices, and its absorption across the intestinal membrane (Rodriguez & Basta, 1999). Studies on the absorption from soil indicate that oral bioavailability of arsenic from soil or dust is often lower than that of pure soluble salts typically used in toxicity studies. However, the availability is substantially dependent on the soil type. Hamel *et al*. (1998) measured arsenic bioaccessibility from soils in two hazardous waste sites using synthetic gastric juice. The results showed that the bioaccessibility varies between 5-56 % depending on the soil type and liquid to solid ratio used in the test. In the study of Sarkar & Datta (2004) the bioavailability from spiked soils ranged from 46 % to 88 % when *in vitro* gastrointestinal method after four months equilibration were used for different soil types. Roberts *et al.* (2002) studied arsenic bioavailability from five different anthropogenically polluted soils representing various soil types, including soil originating from a site contaminated by CCA-chemical. The results showed bioavailability ranging from ca. 11 % to some 25 %. In fact, several studies have shown that natural or aged anthropogenic arsenic in soil is less bioavailable due to aging and sequestration.

Arsenic in air exists on particulate matter and hence, respiratory absorption of arsenic is a two-stage process, involving deposition of the particles on airway and lung surfaces, followed by absorption of arsenic from deposited particulates. Absorption of arsenic from inhaled airborne particles is highly dependent on the solubility and the size of particles. In workers in the smelting industry exposed to arsenic trioxide, 40-60 % of the inhaled As dose has been estimated to be excreted in the urine. This indicates good absorption from arsenic containing particles. However, in the same study the autopsy data representing former smelter workers showed eight-fold higher levels of arsenic in the lungs compared with the control group. Hence, under some circumstances inhaled arsenic can accumulate in lung tissue. (WHO, 2001)

The systemic dermal absorption of arsenic appears to be low. In the study of Wester *et al*. (1993), after 24 hours 6.4 % of arsenic as arsenic acid, and 4.5 % of arsenic mixed with soil was absorbed systemically in Rhesus monkeys. It was suggested that dermal exposure initially leads to arsenic binding to skin, and that the bound arsenic may slowly be taken up into the blood, even after exposure.

There is no scientific knowledge whether absorption of arsenic from the gut in children differs from adults. There is some information suggesting that children may be less efficient in converting inorganic arsenic to the less harmful organic forms. For this reason, children may be more susceptible to health effects from inorganic arsenic than adults. There is some evidence

that inhaled or ingested arsenic can damage pregnant women or fetuses, although the studies are not definitive. Studies on animals show that large doses of arsenic cause illness in pregnant females and can also cause low birth weight, foetal malformations, and even foetal death. Arsenic can cross the placenta and has been found in foetal tissues. (WHO, 2001)

3.2 Kinetics and metabolism

In many species, arsenic metabolism is characterized by two main types of reactions: (1) reduction of pentavalent to trivalent arsenic, and (2) oxidative methylation in which trivalent forms of arsenic are sequentially methylated to form mono-, di- and trimethylated products (WHO 2001).

Once absorbed into human body, arsenic is rapidly distributed throughout the body with skin, nails and hair encompassing the highest concentrations (WHO, 2001). Arsenic appears to be able to transfer from maternal to foetal blood. Metabolic conversion of arsenite to arsenate is considered to be an important intoxication mechanism. Metabolism of arsenate to arsenite can also occur. Mono- and di- methylation of arsenite occurs in several tissues. Methylation of inorganic arsenic facilitates the excretion of inorganic arsenic from the body, as the end-products are readily excreted in urine. Smaller amounts of arsenic are excreted in feces. Some arsenic may remain bound to tissues, depending inversely on the rate and extent of methylation. The methylated arsenic acids are generally regarded as being less toxic and less well retained by the body than the inorganic forms of arsenic. However, recent evidence suggests that toxicity and/or carcinogenicity may also be enhanced by methylation reactions (Duker *et al*. 2005).

Factors such as dose, age, gender and smoking contribute only minimally to the large inter-individual variation in arsenic methylation observed in humans. Animal and human studies suggest that arsenic methylation may be inhibited at high acute exposures. The metabolism and disposition of inorganic arsenic may be influenced by its valence state, particularly at high dose levels. Studies in laboratory animals indicate that administration of trivalent inorganic arsenic initially results in higher levels in most tissues than does the administration of pentavalent arsenic. However, the trivalent form is more extensively methylated, leading to similar long-term excretion. Ingested organoarsenicals are much less extensively metabolized and more rapidly eliminated in urine than inorganic arsenic. (WHO 2001)

Levels of arsenic or its metabolites in blood, hair, nails and urine are used as biomarkers of arsenic exposure (e.g., Hughes, 2006). Since As is rapidly cleared from blood, blood arsenic is a useful biomarker only in the case of acute arsenic poisoning or stable chronic high-level exposure. Urinary As levels represent exposure during the last 4-5 days. Because hair and nails grow slowly, their analysis may give an indication of past exposure to arsenic. The increased arsenic exposure is best indicated by increased concentrations of arsenic species (inorganic arsenic, methylarsonic acid (MMA), dimethylarsinic acid (DMA)) (e.g., Mäki-Paakkanen *et al*. 1998). In the study population in southwest Finland exposed to drinking water with high As concentrations (max. 980 µg/L), the mean concentration of arsenic in the urine was 58 µg/L (N = 17) compared with the mean concentration of 5 µg/L in the reference group exposed to drinking water containing less than 1 µg As/L (N = 9) (Kurttio *et al*., 1998). Another Finnish study showed total arsenic concentration of 180 µg/L in urine and 1.3 mg/kg in hair for the users (N = 32) of drinking water from wells with median concentration of 410 µg As/L (Mäki-Paakkanen *et al*. 1998). Background levels of arsenic in hair are typically below 1 mg/kg and in nails between < 1.5 and 7.7 mg/kg. At the same time, nail clippings from a patient with acute polyneuritis caused by arsenic poisoning have been reported to contain 20-130 mg As/kg (NRC, 1977).

In mammals, arsenic has an antagonistic[1] toxicity relationship with selenium (Se). The mechanism of this effects is not known, but it may be associated with the formation of a complex that is excreted more rapidly than either arsenic or selenium alone (e.g. Gailer *et al*. 2000; Levander, 1977) or due to selenium-induced changes in As methylation (Styblo & Thomas, 2001; Walton *et al*. 2003). Miyazaki *et al*. (2003) found a negative correlation between urinary As and Se concentrations and concluded that excessive As intake may change Se metabolism in humans and enhance fecal Se excretion. On the other hand, Gailer *et al*. (2000) and Spallholz *et al*.(2004) have proposed that because body's As detoxification mechanisms enhance selenium excretion, this could lead to selenium-deficiency. The symptoms of chronic arsenic toxicity and selenium deficiency show significant similarities and might arise confusion.

3.2 Ecotoxicity

The opinions on the essentiality of arsenic to organisms vary to some extent. However, arsenic seems to behave more like an environmental contaminant than as a nutritionally essential mineral. On the other hand, low doses (<2 µg/day) of arsenic have been documented to have positive effects on some animals e.g., on silkworms. Negative effects associated with As deficiency have been observed at least in rats, goats and pigs. The symptoms include low growth rate, decreased hematocrit, increased fragility of red cells, impaired reproduction performance, increased neonatal mortality and lower birth weight. (NAS, 1977)

The ecotoxicity of arsenic has been reported in many studies. Most arsenic investigators agree that the toxicity of arsenicals conforms to the following order, from the highest to the lowest toxicity: arsines > inorganic arsenites > organic trivalent compounds (arsenoxides) > inorganic arsenates > organic pentavalent compounds > arsonium compounds > elemental arsenic (Eisler, 1988). In general, inorganic arsenicals are more toxic than organoarsenicals and arsenite is more toxic than arsenate. The modes of toxicity to organisms differ as does the mechanisms of uptake. The primary mechanism of toxic action has been considered to result from the binding of arsenic (As^{3+}) to sulfhydryl groups of proteins. Arsenate (As^{5+}) is known to affect oxidative phosphorylation by competition with phosphate. Arsenic can cause toxic effects by interacting with sulfhydryl groups of proteins and enzymes, and by increasing the number of reactive oxygen species (Duker *et al*., 2005). Inhibition of enzyme functions has been demonstrated in over 200 enzymes (Abernathy, 1999). Many toxicity features and mechanisms have been studied in mammals, but since some ubiquitous functions are affected (control of DNA repair, enzymes involved in production of cellular energy) the same effects on other species may be revealed in the course of time. The elucidation of all the mechanisms of arsenic toxicity and genotoxicity is still not resolved.

[1] The response to exposure to multiple substances is less than would be expected if the known effects of the individual substances were added together (additive effect).

3.2.1 Terrestrial organisms

Terrestrial biota shows a wide range of sensitivities to different arsenic species. Generally, plants are more sensitive to arsenic than animals (see Table 4). However, highly tolerant plant species and hyperaccumulators have also been identified. The sensitivity of biota and consequently, the magnitude of toxic response, is modified by biological and abiotic factors e.g., temperature, pH, redox-potential, organic content, phosphate concentration, adsorption to solid matrices, the presence of other substances and toxicants, duration of exposure and arsenic species present. In plants, the water-soluble forms of arsenic are the most phytotoxic, arsenite being more phytotoxic than arsenate. Both of these inorganic forms are much more phytotoxic than monosodium methane arsenic acid (MSMA) (Ref. in Patra *et al*. 2004).

Inorganic and organic forms of arsenic previously used as pesticides, plant defoliants, and herbicides accumulate in soils and in plants. Chemical behavior of arsenic is largely similar to that of phosphorus in soils. In all plant species tested so far, arsenate is taken up via the phosphate transport systems. Phytotoxicity appears e.g., as limited species abundance and diversity (WHO, 2001). Plants take up arsenic as arsenite and arsenate, the major forms of arsenic, which is greatly influenced by soil texture and the presence of phosphates. The effects include wilting, chlorosis, browning, dehydration, mortality, and inhibition of light activation (Eisler, 1988). Retardation of root growth and reduced biomass production of the aboveground plant parts are also typical symptoms of phytotoxicity. In agricultural and horticultural crops, the inhibition of physiological and biochemical processes results in reduction in morphological characteristics and yield (Sheppard *et al*. 1992). Major factors affected are tillers (in cereals), plant height, leaf number and area, pod number and length (in legumes), and dry matter production. Phytotoxic effects are generally seen at soil concentrations of 5 – 20 mgAs/kg (Eisler, 1988). An average toxicity threshold of 40 mgAs/kg has been established for crops (Sheppard *et al*. 1992).

Arsenic ions are toxic to most microorganisms, but due to the capability of adapting to extreme environmental conditions and developing resistance mechanisms, a wide range of microorganisms can thrive in environments heavily contaminated by arsenic (Cervantes *et al*. 1994). Microbial transformations of inorganic arsenic to methylated compounds occur both in aerobic and anaerobic conditions (HSG 70, 1992). Microbial communities can also adapt to As-containing environments in a site-specific manner (Turpeinen, 2002). Therefore, it is important to measure the effects of contaminants case-by-case. Interactions of microbes with arsenic species in soil have also consequences regarding the mobility and hence, environmental fate of arsenic (Turpeinen *et al*. 1999, Duket *et al*. 2005).

The data on the toxicity of arsenic to soil invertebrates is very limited, i.e., only data on the toxicity to earthworms is readily available. In birds, the tolerance to arsenic varies among species. The toxic effects include destruction of gut blood vessels, blood-cell damage, muscular incoordination, debility, slowness, jerkiness, falling, hyperactivity, fluffed feathers, drooped eyelids, immobility, seizures, and systemic, growth, behavioral, and reproductive problems (e.g., Stanley *et al*. 1994; Whitworth *et al*. 1991; Camardese *et al*. 1990).

An overview of toxicity reference values related to the effects of As to terrestrial wildlife is presented in Table 4. On the basis of these toxicity reference values, arsenic appears to be toxic to earthworms with long lasting effects (chronic toxicity, category 2[2]). The chronic toxicity to birds and mammals varies between very toxic (category 1[1]) and harmful (category 3[1]) with long lasting effects depending on the species. When interpreting the reference values it has to

[2] The toxicity categories refer to the classification proposed by the United Nations Sub-Committee of Experts on the Globally Harmonized System of Classification and Labelling of Chemicals (UNSCEGHS, 2006).

be kept in mind that several organism-specific and physico-chemical factors affect the toxicity of As to the receptor. These include e.g., age, gender, sex, life stage, nutritional status of the organism, arsenic speciation, and the size, number and frequency of doses. In many cases, not all this data has been reported, which makes the comparison of different studies difficult.

Table 4. Toxicity of arsenic and different As species to terrestrial wildlife, some domestic plants and animals. LOEC = Lowest Observed Effect Concentration, LCT = lowest concentration tested, NOAEL/NOAEC = No Observed Adverse Effect Level/Concentration, LOAEL = Lowest Observed Adverse Effect Level, LD50 = Lethal Dose 50%. In the case of animals, all doses refer to oral exposure.

Target organism and species	**Concentration / Dose**	**Explanation**	**Source**
Earthworm (*Eisenia fetida*)	68 mg/kg-dw in soil	LOEC (56 d), AsV, cocoon/adult, LCT	Fischer & Koszorus, 1992, ref. in Efroymson *et al*., 1997b
Soil microbes	187 / 1675 mg/kg-dw in soil	LOEC (0.1 d, varied organic carbon content), enzyme activity (various), LCT	"
Terrestrial plants - ryegrass (*Lolium perenne*) - blueberry (*Vaccinum angustifolium*) - spruce - potato (*Solanum tuberosum*) - barley (*Hordeum vulgare*)	 22 mg/kg-dw in soil 55 mg/kg-dw in soil¨ 1000 mg/kg-dw in soil 97 mg/kg-dw in soil 22 mg/kg-dw in soil	 geometric mean of LOAEC and NOAEC, growth " LOEC (335 d), As_2O_3, height geometric mean of LOAEC and NOAEC, growth "	 Jiang & Singh, 1994[a] Anastasia & Kender, 1973[a] Rosehart & Lee, 1973[b] Jacobs *et al*., 1970[a] Jiang & Singh, 1994[a]
Birds - mallard duck (*Anas platyrhynchos*) - pheasant (*Phasianus colchicus*) - various species	 3.72 / 17.3 mg/kg-bw/d 0.410 mg/kg-bw/d 1.49 mg/kg-bw/d 386 mg/kg 17.4…3,300	 NOAEL (10 w / 4 w), mortality, juveniles LOAEL (10 w), enzyme activity (acetylcholine-esterase), juveniles LOAEL (2 w), growth, juveniles LD_{50} (single dose), $NaAsO_2$ LC_{50}, arsenic compounds	 Camardese *et al*., 1990[a] / Hoffman *et al*., 1992[a] Camardese *et al*., 1990[a] Camardese *et al*., 1990[a] Sample *et al*., 1996 Eisler, 1988
Terrestrial mammals - rat (*Rattus norvegicus*) - rabbit (*Oryctolagus cuniculus*) - mouse (species not specified) - meadow vole (species not specified) - short-tailed shrew (species not specified)	 1.39...32 mg/kg-bw/d 5.0…20.6 mg/kg-bw/d 0.447...10.3 mg/kg-bw/d 1.2 mg/kg-bw/d 0.750 / 3.0 mg/kg-bw/d 0.44...19 0.114 mg/kg-bw/d 0.150 mg/kg-bw/d	 NOAEL (varying exposure time), mortality, juveniles LOAEL (varying exposure time), growth, juveniles NOAEL (varying exposure time), growth, juveniles LOAEL (6 w), enzyme activity (general changes), juveniles NOAEL / LOAEL (12 d), mortality, gestation LOAEL (lifetime), varying exposure dose and toxic effect, As III NOAEL, arsenite (derived from test species: mouse) NOAEL, arsenite (derived from test species: mouse)	 studies reviewed in USEPA, 2005 " " Wood & Fowler, 1978[a] Nemec *et al*., 1998[a] Sample *et al*., 1996 Opresko, 1994 Sample *et al*. 1996 Sample *et al*. 1996
Domestic animals - dog (*Canis familiaris*) - mouse (*Mus musculus*)	 2.25 / 5.62 mg/kg-bw/d 1.04 / 1.66 mg/kg-bw/d 24.0 / 48.0 mg/kg-bw/d 2.84...7.69 /5.69…32.4mg/kg-bw/d 0.00650; 0.5484mg/kg-bw/d	 NOAEL / LOAEL (2 yr), $NaAsO_2$, mortality, juveniles NOAEL / LOAEL, (8 w), growth, juveniles NOAEL / LOAEL (9 d), mortality, gestation NOAEL /LOAEL (varying exposure time), growth, juveniles LOAEL (91 d; 6 mo), reproduction, juveniles	 Byron *et al*., 1967[a] Neiger & Osweiler, 1989[a] Nemec *et al*., 1998[a] studies reviewed in USEPA, 2005 Healy *et al*., 1998[a]; Schroeder & Mitchener, 1971[a]

[a] Ref. in USEPA, 2005
[b] Ref. in Will & Suter, 1995

3.2.2 Aquatic organisms

In general, inorganic arsenic is not acutely very toxic to aquatic organisms and acute LC50 concentrations vary generally between 10 and 100 mg/l. The data on the toxicity, however, are somewhat contradictory. Moreover, there are differences between marine and fresh water biota. In chronic exposure, juvenile fish and *Daphnia magna* have been reported to show toxic response at concentrations of 4 of mg As/l (fish) and 0,5 mg/l (Daphnia). Arsenic has also been claimed to be one of the most toxic elements to fish. Acute exposures can result in immediate death due to As-induced increases in mucus production, which causes suffocation, or direct detrimental effects on the gill epithelium. Chronic exposures can result in the accumulation of arsenic to toxic levels. The detoxification role of the liver places it at considerable risk and may result in morphological and neoplastic changes. In addition, the toxic responses detected in aquatic organisms include behavioral impairments, growth reduction, loss of appetite, and metabolic failure. Aquatic bottom feeders are more susceptible to arsenic. (HSG 70, 1992)

The reference values associated with chronic toxicity seem to verify the high aquatic toxicity in long-term exposure (Table 5). Algae seem to be more sensitive to arsenate and some species grow poorly already at the concentration of 75 µg/l. Some marine macroalgae may suffer severely at the concentration around 10µg/l and even below. (HSG 70. 1992)

Table 5. Toxicity of arsenic and different As species to aquatic wildlife. LCV = lowest chronic value, PEC = probable effect concentration, TEC = threshold effect concentration

Target organism and species	**Concentration**	**Explanation**	**Source**
Midge, larva (*Chironomus riparius*) (*Tanytarsus dissimilis*)	22 / 54 mg/kg-dw in sediment 97.0 mg/l in water	TEC / PEC (14 d) LC_{50} (48 h)	Jones *et al*., 1997 Holcombe *et al*., 1983 ref in Sample *et al*. 1997b
Amphipod, crustacean (*Hyalella azteca*)	11 / 33 mg/kg-dw in sediment	TEC / PEC (14 d)	Jones *et al*., 1997
Aquatic plants (species not specified)	1 µg/l in water	LCV	Suter and Tsao, 1996
Algae	61,000 µg/l in water	EC50 (12 d), *Scenedesmus quadricauda*	Ecotox database, 2007
Water flea (*Daphnia* sp., *Ceriodaphnia* sp., *Simo cephalus*)	0.23 µg/l in water 2,850…7,400 µg/l in water 520 / 1400 µg/l in water 1500…4300 µg/l in water	LCV LC_{50}, *Daphnia magna* EC_{50}, reproduction LC_{50} (48 h), *D. magna*, *D. pulex*, *Ceriodaphnia reticulate*	Suter and Tsao, 1996 Nikunen *et al*., 2000 “ Ecotox database, 2007
Invertebrates (species not specified)	6.1 µg/l in water	LCV	Suter and Tsao, 1996
Fish - species not specified - different species - salmon (*Salmo gairdnerii)* - rainbow trout (*Oncorhynchus mykiss*)	 3.8 µg/l in water 10,000…18,000 µg/l in water 550 µg/l in water 23,300…26,600 µg/l in water	 LCV LC_{50} (2-4 d), As_2O_5 LC_{50} (24 h), As_2O_5 LC_{50} (96 h), As III	Suter and Tsao, 1996 Nikunen *et al*., 2000 “ Spehar *et al*. 1980 ref in Sample *et al*. 1997b

3.3 Human toxicity

3.3.1 Non-cancer toxicity

Arsenic-containing compounds vary in toxicity to mammals according to the valence state, form (inorganic or organic), physical state (gas, solution, or powder) and factors such as solubility, particle size, rates of absorption and elimination, and presence of impurities. The data available from studies on humans show skin to be the most sensitive target of non-cancer effects associated with long-term oral exposure to arsenic (WHO, 2001). Typical dermal effects include hyperkeratinization (especially on the palms and soles), formation of multiple hyperkeratinized corns or warts, and hyperpigmentation of the skin with interspersed spots of hypopigmentation. At oral exposure levels of about 0.002–0.02 mg As/kg/day, peripheral vascular effects are also commonly noted, including cyanosis (bluish coloration of the skin due to the deoxygenated hemoglobin) and gangrene (skin decay) (Chen *et al*. 1994). Other cardiovascular effects of oral exposure to inorganic arsenic include increased incidences of high blood pressure and circulatory problems. Muscle cramps, mainly in the legs, have been reported in people who have been drinking water with high arsenic contents (max. 980 μg/L) in southwest Finland (Kurttio *et al*., 1998). Moreover, chronic arsenic exposure in Taiwan has been shown to cause blackfoot disease (BFD), a severe form of peripheral vascular disease which leads to gangrenous changes (e.g., Chen *et al*., 1994). This disease has not been documented in other parts of the world, and the findings in Taiwan may depend upon other contributing factors. No reliable dose response data for these effects has been found but oral exposure data from studies in humans indicate that increased risks of As-associated skin lesions are manifested with ingestion of drinking water even with concentrations ≤50 μg As/l corresponding an exposure of approximately 2 μg As/kgBW/day (WHO, 2001). Skin lesions appear to be uncommon in the case of exposure through respiratory organs.

There are only a few quantitative data on non-cancer effects in humans exposed to inorganic arsenic by the inhalation route. However, it appears that such effects are improbable below a concentration of about 0.1–1.0 mg As/m^3. Arsenic has been shown to cause peripheral neuritis following inhalation exposure. Peripheral neuritis (inflammation) has been found in workers inhaling around 50μg As/m^3. (WHO, 2001)

Arsenic has reported to induce different chromosomal disturbances, down-regulation of DNA repair and oxidative DNA damage (Snow *et al*. 2005). Arsenic has also shown immunotoxic effects in *in vivo* and *in vitro* tests (WHO, 2001). Equivalent to several metals it appears to produce immunostimulation at low exposure levels but immuno-suppression at higher exposures. In addition, it has been associated with non-insulin diabetes mellitus.

Although the data on the effects of the organic forms of arsenic to human health are sparse, it is generally considered that organic arsenicals are substantially less toxic than the inorganic forms. However, data available mainly from animal studies imply that adequate doses of the methyl and phenyl arsenates can produce adverse health effects that resemble those of the inorganic arsenicals. Thus, the possibility of health risks from the organic arsenicals should not be disregarded. (WHO, 2001).

3.3.2 Genotoxicity and carcinogeneity

Arsenic is both genotoxic and a well-known human carcinogen (CSTEE, 2001). The International Agency for Research on Cancer (IARC, 1988 and 2004) has classified arsenic and arsenic compounds to class 1 (known human carcinogens). However, this evaluation does not

necessarily apply to all individual compounds within the group. USEPA has stated that inorganic arsenic reaching the body through inhalation and oral intake is a human carcinogen and has assigned it the cancer classification, Group A (USEPA, 1998). Arsenite is considered to be a more potent carcinogen (perhaps up to ten fold) than arsenate. However, the *in vivo* relevance of this data is questionable since arsenite and arsenate are interchangeable in the body.

The principal target organs of carcinogenic response to arsenic in humans are the lungs, skin and bladder. There is evidence that the colon, liver and kidney may also be targets. Lung cancer appears to be a critical effect following chronic inhalation exposure to arsenic (WHO, 2001). However, there has been much controversy concerning the shape of the response curve, particularly at low doses. This controversy has been exacerbated by the fact that the mechanisms of arsenic carcinogenesis are still unclear, possibly due to arsenic tolerance found in a number of experimental animals but not in humans. European scientific committee concluded that although there are some reasons for considering that there may be a threshold for carcinogenicity, the direct evidence to support this is poor (CSTEE, 2001). In the absence of such data it is considered as appropriate to assume that no threshold exists.

Kurttio *et al.* (1999) assessed the levels of arsenic in drilled wells in Finland and studied the association of arsenic exposure with the risk of bladder and kidney cancers. Water samples were obtained from the wells used by the study population during 1967-1980. The total As concentrations in the wells of the reference study population were low (median = 0.1 μg/L; maximum = 64 μg/L), and 1% exceeded 10 μg/L. Despite of the very low exposure levels, some evidence of the association between arsenic in well water and the incidence of bladder cancer was found. Bladder cancer tended to be associated with As concentration and daily dose during the third to ninth years prior to the cancer diagnosis while there was no association between arsenic and incidences of kidney cancer and skin cancer. On the contrary to this study, some epidemiological studies have shown that the relative cancer risk among populations exposed to moderate concentrations to arsenic in their drinking water is often lower than the risk for the unexposed control population (Lamm *et al*. 2004, Mahata *et al*. 2004). This low dose adaptive (protective) response by a toxic agent is characteristic of many agents that induce oxidative stress (Snow *et al*. 2005).

4 STUDY MATERIALS AND METHODS

4.1 Description of the study problem

The aim of the risk assessment task was to find out the magnitude and scope of risks associated with environmental arsenic in the Pirkanmaa region. These risks originate from elevated As levels in soil, water, sediment, air and diet and may convergence in human beings (e.g., residents in the vicinity of contaminated sites or consuming contaminated groundwater) or to biota dwelling (or feeding) on or in contaminated media. Both naturally occurring arsenic and anthropogenic arsenic are involved in the formation of risks to human health and biota in Pirkanmaa. Due to the lack of comprehensive data from the whole region, the risk assessment was focused on certain identified hot spot areas, i.e., specific areas found to include high environmental concentrations of arsenic.

4.1.1 Study data

Pirkanmaa can be divided into three geologically distinct units based on the dominant rock types encountered in the region. This division is crucial in the identification of natural As anomaly areas. The main geological subdivisions in the study area are (Fig. 1): the Central Finland Granitoid Complex (CFGC) in the north, the Tampere Belt (TB) in the centre, and the Pirkanmaa Belt (PB) in the south (Nironen *et al.*, 2002). From the bedrock, arsenic has been migrated to groundwater posing a potential risk to human health in the case of using water with elevated As concentrations as drinking water.

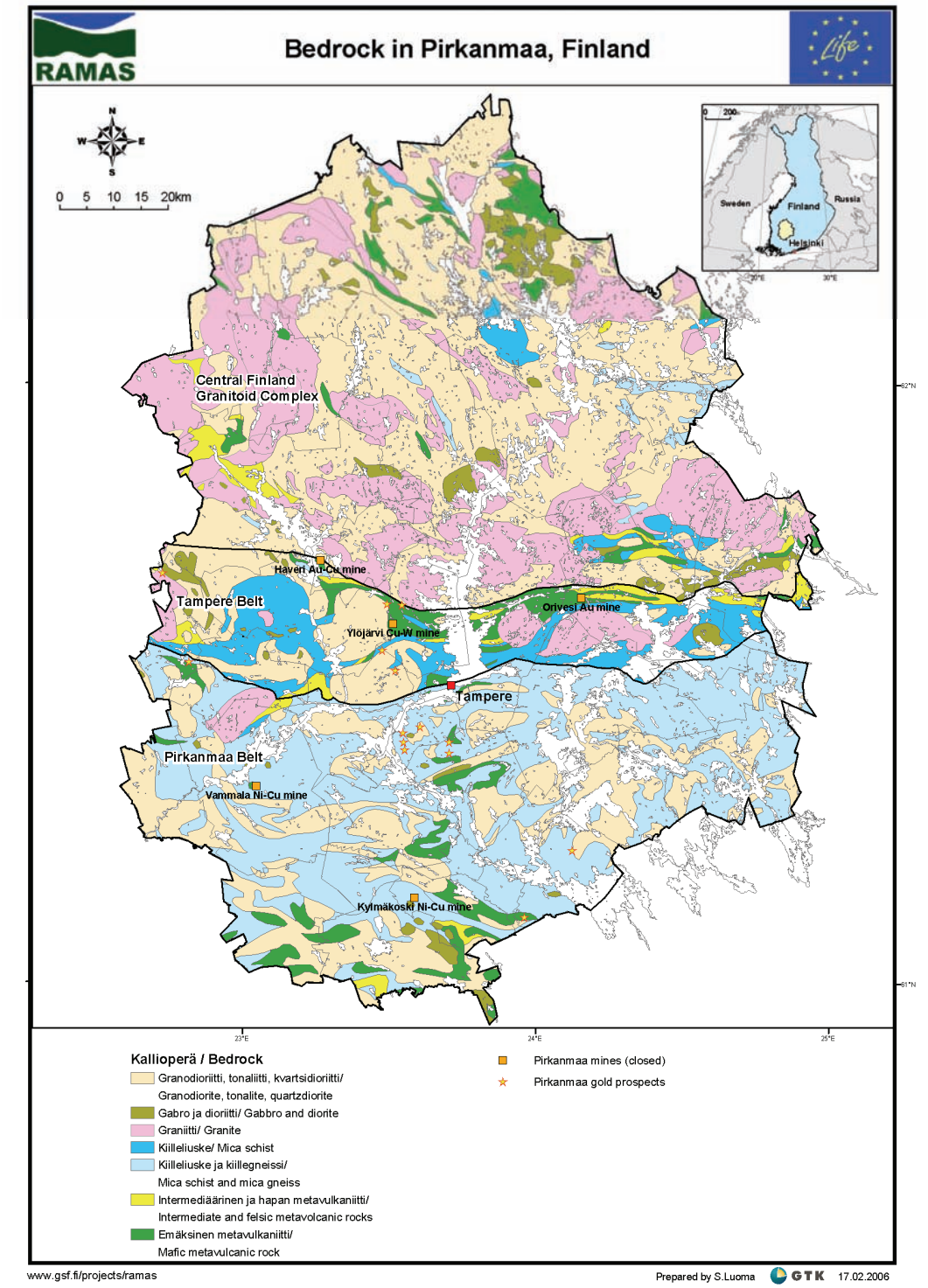

Figure 1. Bedrock in the Pirkanmaa region. Processed from the GTK data (Geological mapping data © Geological Survey of Finland, Base map data © National Land Survey of Finland) (from Backman *et al.*, 2006). The mines acting as potential anthropogenic As sources are also shown.

The majority of the data on As concentrations in soils within Pirkanmaa relates to till fines. According to this nation-wide data, the As concentrations in till are the highest within the Pirkanmaa belt and lowest within the CFCG belt. In addition to this nation-wide survey on till, data associated with ore exploration has been collected from a relatively small, geochemically anomalous area. (Backman *et al*. 2006)

The previous data on arsenic in the Pirkanmaa area well waters consisted of results from the analysis of 1183 samples from drilled rock wells and 269 results from dug wells taken between 1991 and 2002. During the RAMAS project, supplementary sampling of groundwater and till soils was carried out in 2005. Of the 103 new groundwater samples, 89 were from drilled wells and 14 from shallow dug wells. (Backman *et al*. 2006)

To complement the soil and groundwater data, forest soil, crops, surface waters, sap and mushrooms were studied in selected areas during RAMAS. The studies on forest soils consisted of samplings of different soil profiles, the majority of which were taken from areas with clay and fine sand. Each of the 11 profiles was composed of four different sampling depths representing humus (11 samples), clay (27 samples), and fine sand (6 samples). Along with the studies on the crops, arable soils (N = 15) in the areas of potentially high As concentrations were investigated (documented in Mäkelä-Kurtto *et al*. 2007). The data on the concentrations in crops were used in the health risk assessment and the data on the concentrations in arable soils in ecological risk assessment.

The potential anthropogenic sources of arsenic include old wood preservation plants which might have used CCA chemical. Within Pirkanmaa there are 14 such plants, at the majority (8) of these some remediation actions have been carried out. Another type of the main As hot spot areas are mining sites, which may affect vast areas through air and particularly through surface waters. In RAMAS, data from four old CCA-treatment plants (three of these had been previously remediated) were reviewed and supplementary investigations were carried out at one site (Ruovesi I, not remediated). In addition, one mine site (Ylöjärvi) was studied for As contamination. In 2005, five composite samples (depth 0.05-0.3 m) were taken from the southern tailings area of Ylöjärvi mine to determine the total concentrations of As and heavy metals (using strong nitric acid as a solvent). In addition, the amount of potentially available arsenic was determined from the selected samples from the mine site and CCA-plant using a laboratory-scale leaching test (two-stage batch test EN-12457-3). Both the soil samples and the eluates from leaching tests were further tested for toxicity using luminescent bacteria test and reverse electron transport assay (RET) test. (Parviainen *et al*. 2006, Schultz & Joutti, 2007)

For the assessment of risks to terrestrial biota, soil samples from sites with elevated natural or anthropogenic arsenic were also studied using ecotoxicity tests with plants and soil animals (see chapter 4.2.5 and Schultz & Joutti, 2007). For the assessment of risks to aquatic biota, the monitoring data collected previously in the water system affected by the effluents from the Ylöjärvi mine and from some water systems within Pirkanmaa both with and without anthropogenic sources were compiled. To study the long-term effects in the lake ecosystem, we also investigated the abundance and diversity of diatoms in the sediment in one part of the water system. In addition, environmental fate of arsenic in the drainage area was modeled (Bilaletdin *et al*. 2007). To study the immobilization of arsenic from bedrock owing to human activities, few surface water samples were collected in the sites in which quarrying was practiced. All this data generated within other RAMAS tasks was used as a basis for the risk assessment.

4.1.2 Arsenic concentrations in the environment

The data on the As levels in well waters collected between 1991 and 2002 were used to calculate the mean value of As concentration. In the case of multiple samplings representing the same drilled well, only the first concentration measured was included in the calculation. We arrived at the average concentration of 29.7 µgAs/l representing 1151 samples from drilled wells. The median concentration was 2.5 µgAs/l, maximum 2230 µgAs/l and standard deviation (sd) 137 µgAs/l. The big difference between the median and the mean indicates that only few samples had very high As concentrations while in the majority of the samples, the concentrations were low. Within the separate geological belts, the average concentrations were the following: (µgAs/l): CFCG 1.4 (sd 2.0, maximum 10), TB 51.8 (sd 182, max. 2230) and PB 13.9 (sd 86.7, max. 1560). In the dug wells the mean arsenic concentration was 0.65 µg/l (sd of 3.3 µg/l) with a median of 0.19 µg/l and the maximum of 45 µg/l. This *preliminary data* on the As concentrations in well waters was used in the Phase 1 health risk assessment (Section 4.4.1 and 6.1).

When the preliminary data and new well water data collected in RAMAS were combined, the recalculation resulted in the average concentration of 28.1 µgAs/l in drilled well waters with sd = 132 µgAs/l. The median concentration was 2.5 µgAs/l and the maximum concentration 2230 µgAs/l. In the dug wells, the mean arsenic concentration was 0.75 µg/l (sd = 3.6 µg/l) with a median of 0.20 µg/l and a maximum of 45 µg/l. In the final aggregate data, the average arsenic concentrations in drilled well waters by the geological units were (µg/l): CFCG 1.36 (sd 2.0, maximum 10), TB 48.2 (sd 176, maximum 2230) and PB 13.6 (sd 83, maximum 1560). In the dug well waters the average concentrations were (µg/l): CFCG 1.11 (sd 4.3, maximum 29.2), TB 0.37 (sd 0.56, maximum 2.51) and PB 0.61 (sd 3.4, maximum 45). This aggregate data was used in the Phase 2 health risk assessment (Section 4.4.1 and 6.2).

According to the the nation-wide till data the median arsenic concentration in Pirkanmaa is 5.35 mg/kg, which is slightly higher than that of the whole country (2.57 mg/kg) (Backman *et al*. 2006). The results from ore exploration areas show much higher median values compared to this data. Moreover, arsenic content tends to increase with depth and maximum values are very high. In the soil samples representing the mine site and CCA-plant, As concentrations varied between 1000 and 2200 mg/kg (Parviainen *et al*. 2006).

As concentrations in the crop samples studied were at the typical national level. Mean concentration in wheat grains was 0.005 mgAs/kg-dw (n=5, range <0.004-0.005), in peeled potatoes 0.004 mgAs/kg-dw (n = 5, range 0.002-0.006) and in unpeeled potatoes 0.008 mgAs/kg-dw (n = 5, range <0.006-0.011) (Mäkelä-Kurtto *et al*. 2006). Arsenic had one of the lowest soil-to-plant uptake factors among the 13 elements studied. It was concluded that the contents of arsenic - like other elements - in plants are mainly genetically determined, but are also influenced by soil, atmosphere, weather, and climate factors and by cultivation practices. No elevated concentrations of As were found in sap and mushrooms (unpublished data).

Contents of arsenic (and other elements) in the arable soils were of the same low level as found in other regions in Finland, the average value being 4.06 mgAs/kg (sd 1.03 mgAs/kg, max. 6.8 mgAs/kg) in the plough layer. Concentration was generally slightly higher in the plough layer than in the subsoil (mean 3.72 mgAs/kg, sd 0.58 mgAs/kg, max. 4.82 mgAs/kg). The correlations of As concentrations with the characteristics of soil were weak, humus and clay content showing the strongest positive correlations. The low concentration of acid ammonium acetate – EDTA extractable As concentration also suggested a low availability to plants. (Mäkelä-Kurtto *et al*. 2006)

In forest soil, As content in the humus layer varied from 2.17 to 8.58 mg/kg with the median value of 4.67 mg/kg. The average As content was higher in clay samples than in the samples representing fine sand. Samples taken from the areas of fine sand contained low concentrations (less than 5 mgAs/kg) for all layers. The average arsenic content in the combined data representing mineral soil was 5.07 mgAs/kg with the median value of 3.84 mgAs/kg and the maximum of 14.2 mgAs/kg. (Mäkelä-Kurtto *et al*. 2006)

The data on the concentration of arsenic in surface waters show a clear effect of anthropogenic sources. The concentrations of arsenic in the surface water affected by effluents from the Ylöjärvi mine site were more than 100-fold higher, at the maximum, compared with 'normal' background. Slightly elevated As concentrations were also detected in the surface waters affected by quarrying activities.

The concentration data available for the risk assessment is summarized in Appendix 1.

4.2. Ecological risk assessment

4.2.1 Goals and methodology

The ecological risk assessment (ERA) in RAMAS was focused on the study of the identified hot spot areas with high concentrations of arsenic originating from anthropogenic sources or natural occurrence. The purpose of ERA was to give preliminary information on the possible ecological effects related to elevated arsenic levels in such hot spots within the Pirkanmaa area. Due to the lack of data, the detailed ERA was focused on terrestrial environment while it was possible to carry out only a screening level ERA on aquatic biota.

In the regional level ERA, the aim is generally to study the risks related to all stressors taking into account the spatial heterogeneity of landscape (e.g., Hunsaker *et al*. 1990). Such a study would have necessitated data on the specific landscape forms, protected areas and receptors and valued resources within the whole region and data on the importance of other stressors than As. This detailed data was not available to us and could not be generated within RAMAS. Also, the focus of RAMAS –project was on arsenic only. In practice, the inclusion of other concurrent contaminants could not be avoided due to their presence in the environmental samples. Hence, the aim of the ERA was to produce the following outcomes:

- risk estimates for some terrestrial key organisms which might grow or dwell at the identified As hot spot areas; various methods were used to increase the reliability of the results
- screening-level risk estimates for some key aquatic organisms within the Ylöjärvi water system.

In order to identify the targets for risk management actions, the results were further combined with the data on the locations of valued resources within Pirkanmaa (reported separately in the context of Task 4, Lehtinen *et al*., in preparation).

ERA was based on the use of the following methods:

- comparison of environmental concentrations with the ecological benchmarks issued for different environmental media (soil, water, sediment) (tier 0):
- determination of toxic responses using laboratory-scale ecotoxicity tests and soil samples collected from the hot spot areas (tier 2);
- modelling of the exposure of some key terrestrial organisms and comparison of the estimated dose or concentration with the toxicity reference values (tier 1 and tier 2);

- studies on the abundance of diatoms in different sediment layers in the water system receiving runoffs from a mine site (tier 1).

We followed a tiered approach recommended both on international and national level (see Fig. 2). In a tiered approach, ERA starts with a screening level assessment (tier 0) generally based on the comparison of environmental concentrations of contaminants with the ecological benchmarks indicating possible risks to a specific species or biota in general. Exceeding of the benchmarks normally indicates the need for a more detailed i.e., baseline assessment (tier 1). Here, we used some uptake and intake models to derive risk estimates. In tiers 0 and 1, we used all data on concentrations of arsenic in different environmental media (soil, water, air, sediment) compiled and produced in RAMAS. Hence, unlike in health risk assessment (see Section 4.4.1), we did not study separately the chemical data collected before the start of RAMAS project (i.e., preliminary data). Such study was not meaningful in the case of ecological risks since the additional chemical data was focused on well water. Hence, we did not expect the results of ERA to differ from those derived on the basis of preliminary data. In tier 2 we included the data from specific laboratory-scale ecotoxicity test (see Section 4.2.5) in the assessment. In addition, we elaborated the assessment based on utake and intake modeling by the inclusion of a statistical analysis.

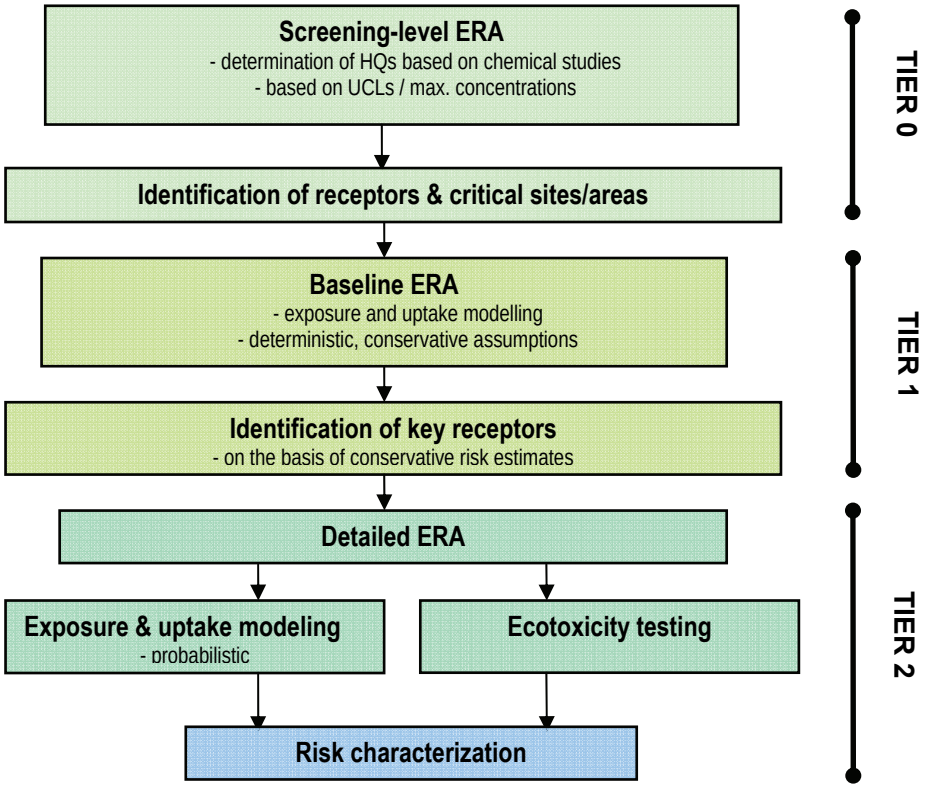

Figure 2. Tiered approach followed in the ecological risk assessment (terrestrial ecosystems). UCL = Upper Confidence Limit (95 %) of the mean value

Moving from tier 0 to higher tiers means collecting additional data. This way, the resources needed for ERA are optimized i.e., only such additional data are gathered which is necessary in order to arrive at a decision on risk management or to verify the need for further assessment. However, in our case owing to the fact that RAMAS was a demonstration project, the ecotoxicity tests were run accorging to the study plan and simultaneously with other studies. Hence, the results were already available during the realization of the entire ERA. So, rather than using the tiered approach as a mean to optimize regional and site-specific studies we followed the approach in order to identify the key hot spot areas and key organisms on which

we should focus in the next tier. This way, the resources put on the actual assessment work are optimized.

Due to the lack of data on the habitat types and sizes, the diversity of species, and abundance within species, it was not possible to carry out a detailed ecological risk assessment on community and ecosystem level. This would have required ecological studies (studies on the abundance and biodiversity of biota). The need for a more detailed ERA was to be assessed on the basis of the results from this preliminary ERA.

4.2.2 Preliminary conceptual models

The hot spot areas included in the ERA and the environmental media involved are presented in Table 6.

Table 6. Areas included in the ecological risk assessment. SW = surface water, GW = groundwater

Site	Environmental media studied	Source of arsenic
Former wood treatment plant	soil, SW, GW	CCA chemical (anthropogenic)
Ylöjärvi mining site	SW, soil, air	Cu-W-As mine (anthropogenic)
Farm	soil, GW, food crops	natural
Forest area	soil, biota (mushrooms), sap	natural

Arsenic pathways, transport and exposure mechanisms and receptors vary depending on the site. These are described in a conceptual model. Fig. 3. presents a generic conceptual model for a former wood treatment plant and a mining site. The conceptual models for risks associated with naturally occurring arsenic are principally equivalent in the case there is a water system involved.

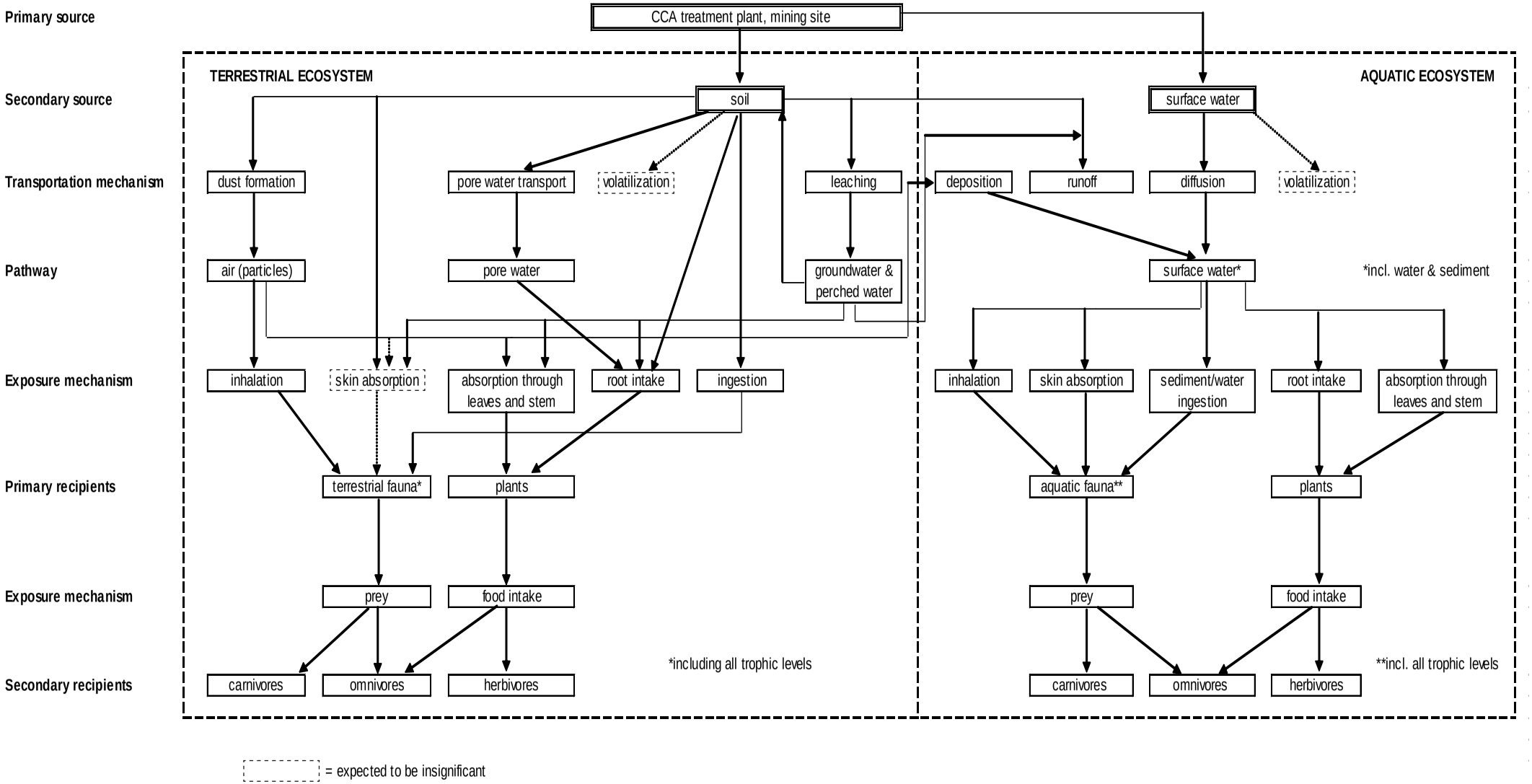

Figure 3. Preliminary conceptual model describing the formation of ecological risks owing to anthropogenic arsenic at a former CCA wood treatment plant and the Ylöjärvi mining site.

Normally, the definition of the key receptors and assessment endpoints should be based on site-specific data on the local biota dwelling on the site or on the biota typical to equivalent sites in non-polluted areas or in areas without natural contaminant anomalies, environmental conditions and land use. These data are used to define which species are to be protected at a particular site. In our study case, there was no such data available since no ecological studies were carried out. Moreover, our 'site' covered the entire province (ca. 15,000 km^2). In some countries, e.g., in the Netherlands and USA, guidelines for the definition of generic assessment endpoints have been issued (Faber, 1998; USEPA, 2003a). Since such guidelines do not exist in Finland, the receptors were selected on the basis of the general data on the prevalence and importance of different organisms in the Finnish landscape types and the size and the land use scenarios of the specific hot spot areas studied within RAMAS. These hot spot areas, the land use scenarios related to them and the key receptors to be protected are presented in Table 7.

Table 7. Terrestrial receptors to be protected in different land use scenarios when only the most sensitive activities in different land use categories are considered.

Type of the hot spot	**Relevant land use scenarios**	**Important ecological factors[a]**
former CCA wood treatment plant (anthropogenic As)	- natural area - residential area with gardens	all species, interactions and processes the most sensitive crops, ornamental plants and grasses, nutrient cycles and symbiotic interactions, soil recovery potential, domestic animals and eusynantrophic biota
former mining area (anthropogenic As)	- natural, forested area - recreational amenities, parks	all species, interactions and processes insensitive plant species, grasses, trees, shrubs, nutrient cycles, avifauna
agricultural area (natural As)	- agricultural - natural area (meadows) - residential area with gardens - recreational amenities, parks	the most sensitive crops and cattle, soil recovery potential all species, interactions and processes the most sensitive crops, ornamental plants and grasses, nutrient cycles and symbiotic interactions, soil recovery potential, domestic animals and eusynantrophic biota insensitive plant species, grasses, trees, shrubs, nutrient cycles, avifauna
forest area (natural As)	- natural area - recreational amenities, parks	all species, interactions and processes insensitive plant species, grasses, trees, shrubs, nutrient cycles, avifauna

[a]adapted from Faber, 1998 and van Hesteren *et al.*, 1999

When specifying the species representing each type of receptors, the following criteria are generally applied:

- probability of existence at the study site;
- availability of toxicity data;
- sensitivity to the key contaminants;
- ecological importance of the species in ecosystem level;
- suitability of scale i.e., the size of habitat range in relation to the size of the contaminated area.

4.2.3 Comparison with ecological benchmarks (Tier 0)

The preliminary, tier 0 level ecological risk assessment was based on the use of ecological benchmarks (Tables 8 and 9). Benchmarks are helpful in determining whether contaminants warrant

further assessment or if they are at a level that requires no further attention. Benchmarks are generally used in the identification of contaminants of potential concern (COPC). If the concentration of a contaminant falls below the lowest benchmark value, the contaminant may be eliminated from further studies. Concentrations exceeding an upper screening benchmark indicate that the contaminant is clearly of concern and that remedial actions are likely to be needed. The benchmark values vary considerably since they are based on the toxicity to different receptors. The methodology for the derivation (e.g. the level of protection, safety factors etc.) and the toxicity data used as basis may also vary. Moreover, the status of benchmarks in decision-making (e.g., regulatory vs. advisory) may be different. Therefore, the use of multiple benchmarks is generally recommended to indicate the likelihood and nature of effects. For example, exceedance of only one conservatively estimated benchmark may provide weak evidence of real effects, whereas exceedance of multiple benchmarks of varying conservatism may provide strong evidence of real effects (e.g., Jones *et al.* 1997).

Table 8. Ecological benchmarks for arsenic in soil. SSL = soil screening level, SRC = serious risk concentration

Benchmark mg/kg-dw	**Explanation**	**Source**
20 100	phytotoxicity, gardens and allotments phytotoxicity, public parks, gardens, recreational areas	van Hesteren *et al*. 1999; van de Leemkule *et al*. 1999
10	Phytotoxicity	Efroymson *et al*. 1997c
18 43 /67 /1,100 46 /170	Eco-SSL, plants Eco-SSL, avifauna: insectivores/herbivores/carnivores Eco-SSL, mammals: insectivores/herbivores& carnivores	USEPA 2005
0.9 / 25 56 / 160	HC_5, soil species / microbial processes HC_{50}, soil species / microbial processes	Swartjes 1999
60	community-level BM, low confidence (based on a single study)	Efroymson *et al*. 1997b
9.9	preliminary remediation goal (PRG), shrew	Efroymson *et al*. 1997a
85	SRC, all species and processes in soil	Verbruggen *et al*. 2001

The Eco-SSLs issued by USEPA are concentrations of contaminants in soil that are protective of ecological receptors that commonly come into contact with soil or ingest biota that live in or on soil. They are conservative and presumed to provide adequate protection of terrestrial ecosystems. The Eco-SSLs are to be used at the screening stage of ERA, i.e., to identify whether a further site-specific ERA is needed. The Preliminary Remediation Goal (PRG) of soil is the lowest of the benchmarks for wildlife, plants and soil invertebrates (Efroymson *et al*. 1997a). Generally, PRGs (also those for aquatic environment) correspond to minimal effects on individual organisms and in turn, minimal effects on populations and communities. It is noteworthy that PRGs may not be sufficiently protective of species of special concern, e.g. protected species.

The most recent Finnish soil guideline values for arsenic in soil are the following: 5 mg/kg-dw (benchmark), 50 mg/kg-dw (lower guideline value), 100 mg/kg-dw (upper guideline value). These are based on ecological risks (lower and higher guideline values) and background concentrations (benchmark value). The derivation of the guideline values has been equivalent to the Dutch methodology, i.e., they have been derived from the concentration levels which are expected to cause adverse effects to 50% of soil species or processes (Hazardous Concentration, HC50).

In Finland, no generic quality standards for arsenic in aquatic ecosystems have been issued. However, several organizations abroad have presented various ecological benchmarks suitable for screening-level ERA in aquatic ecosystems (Table 9).

Table 9. Ecological benchmarks for arsenic in surface water.

Benchmark µg/L	Explanation	Source
190	As^{3+}, aquatic biota, chronic NAWQC	Suter & Tsao, 1996
48	As^{5+}, aquatic biota, lowest CV	"
3.1	As^{5+}, aquatic biota, SCV, Tier 2	"
50	As^{5+}, aquatic biota, EQG	CCME, 2002
5.0	total As, aquatic biota	CCME, 1999
31 / 190	As^{5+} / As^{3+}, PRG based on the lowest toxicity reference value	Efroymson *et al*., 1997
150 / 850	CCC As total / CMC As^{5+}	NOAA, 2004

NAWQC = National Ambient Water Quality Criteria; CV = Chronic Value; SCV = Secondary Chronic Value; EQG = Environmental Quality Guideline;
PRG = Preliminary Remediation Goal; CCC = Criteria Continuous Concentration (chronic value, concentration corresponding the maximum 4-d average exposure level not to be exceeded more than once every three years) ; CMC = Criteria Maximum Concentration (acute value, concentration corresponding the maximum 1-h average exposure level not to be exceeded more than once every three years); NOAA = National Oceanic and Atmospheric Administration (USA)

Generally, the chronic benchmarks for aquatic biota are to be used as lower screening benchmarks (Suter & Tsao, 1996). Each of the alternative benchmarks has a different interpretation. In the USA, the National Ambient Water Quality Criteria (NAWQCs) are regulatory values indicating the need of actions. Lowest chronic values have been presented by the USEPA in place of NAWQC, but they are not criteria. The Tier 2 values are more conceptually consistent with the NAWQCs than lowest Chronic Values (CVs) but they are only USEPA's proposals. Exceeding of the Tier II value implies a greater than 20% chance that the NAWQC, if their value were known, would be exceeded. Exceeding of any of the other benchmark indicates a risk of real effects that should lead to additional data collection and assessment. However, these inferences all depend on comparison of the benchmarks to appropriate water concentrations. They merely indicate that the USEPA believes toxic effects may occur at that concentration. In the identification of risks to aquatic biota, Suter and Tsao recommend to consider all benchmarks presented.

In the case of , e.g., microbes in the sediment, plants, bottom-dwelling invertebrates and bottom-feeding fish, benchmarks for sediment should be applied in the identification of risks. No national guidelines exist for the evaluation of contamination of sediments while several organizations abroad have issued benchmarks based on different levels of protection (Table 10). The Finnish Ministry of the Environment has only issued guidelines for the replacement of dredged sediments in the sea: 15 mgAs/kg-dw (level 1) and 60 mgAs/kg-dw (level 2) (Ministry of the Environment, 2004). If the concentration is below level 1, sediment is considered as harmless while the exceeding of level 2 indicates that sediment is contaminated. Between the levels 1 and 2, sediment is considered as potentially contaminated and a site-specific investigation is needed. These guideline values have been derived for marine environments and they are not straightforwardly applicable to surface waters.

Table 10. Ecological benchmarks for arsenic in fresh water sediment.

Benchmark mg/kg	Explanation	Source
7.24	U.S. Region IV, TEL	Jones *et al*. 1997
8.2	OSWER (USA), ER-L	"
12.1	ARCS (USEPA), TEC	"
6	MOE (Canada), Low	"
5.9	NOAA, TEL CCME, ISQG	NOAA 2004 CCME 2002

TEL = Threshold Effects Level; OSWER = Office of Solid Waste and Emergency Response; ER-L = Effects Range – Low; ARCS = Assessment and Remediation of Contaminated Sediments Program; TEC = Threshold Effect Concentration, MOE = Ontario Ministry of the Environment; Low = lowest effect level i.e., the 5th percentile of the screening level concentration; NOAA = National Oceanic and Atmospheric Administration (USA); CCME = Canadian Council of Ministers of the Environment; ISQG = Interim freshwater sediment quality guideline

The sediment benchmarks presented in Jones *et al*. (1997) are meant to be used as screening values at some sites governed by the U.S. Department of Energy (DOE). They indicate the nature and extent of contamination and the need for additional site-specific studies. These benchmarks can also be used for baseline ERAs but they must be used merely in the identification of contaminants which are most likely causing the toxicity and not as the sole measure of sediment toxicity. The quality guideline issued by the Canadian Council of Ministers of the Environment (CCME) corresponds to the 5 % incidence of adverse biological effects in aquatic biota. Unlike the Effect Range value (ER), the Threshold Effects Level (TEL) also incorporates chemical concentrations observed or predicted to be associated with no adverse biological effects (no effects data). Specifically, the TEL is the geometric mean of the 15th percentile in the effects data set and the 50th percentile in the no effects data set. Therefore, the TEL represents the upper limit of the range of sediment contaminant concentrations dominated by no effects data

In tier 0, the above-presented ecological benchmarks (BM) were used to calculate risk estimates (Eq. 1):

$$HQ_{ib} = EC_j / BM_{ib} \quad (1)$$

where HQ_{ib} = hazard quotient indicating the risk to an organism b living in environmental compartment i (soil, water, sediment); EC_j = concentration of arsenic in environmental compartment j; BM_{ib} = benchmark issued for organism b living in the environmental compartment i.

In the characterization of risks, the following rules of thumb can be applied:
EC < BM → low risk
EC = 1-10 x BM → moderate risk
EC = 10-100 x BM → high risk
EC >100 x BM → extremely high risk.

4.2.4 Modelling (Tier 1 and 2)

The modelling of exposure of selected key terrestrial organisms was based on the validated uptake models and models for the determination of the habitat size presented in the literature (Table 11).

Table 11. Models used in the quantitative ERA (terrestrial biota).

Organism	Model	Source
- animals on soil	$E_j = \frac{A}{HR}\sum_{i=1}^{m}(I_i \times C_{ij})$	adapted from Sample *et al*., 1997a
- earthworm	As: 1) $C_{earthworm} = 0{,}523 * C_{soil}$ [a] As: 2) $\ln(C_{earthworm}) = (0.706 \pm 0.169) * \ln(C_{soil}) + (-1.421 \pm 0.327)$ [b] Cd: $(0.759 \pm 0.037) * \ln(C_{soil}) + (2.114 \pm 0.079)$ Cr: $C_{earthworm} = 3.162 * C_{soil}$ [a] Cu: $\ln(C_{earthworm}) = (0.264 \pm 0.040) * \ln(C_{soil}) + (1.675 \pm 0.141)$ [c] Ni: $C_{earthworm} = 4.730 * C_{soil}$ [a] Pb: $\ln(C_{earthworm}) = (0.807 \pm 0.044) * \ln(C_{soil}) + (-0.218 \pm 0.245)$ [b] Zn: $\ln(C_{earthworm}) = (0.328 \pm 0.024) * \ln(C_{soil}) + (4.449 \pm 0.132)$ [b]	Sample *et al*.. 1998a Sample *et al*., 1999; USEPA, 2003b Sample *et al*., 1998a ” ” ” ” ”
- shrew	HR (acres) = $0.59 * BW^{0.92}$ I_{food} (kg-dw/kg/d) = $(0.0306 * BW^{0.564})/BW$ (rodents) I_{water} (l/kg/d) = $(0.099 * BW^{0.90})/BW$	Sample *et al*., 1997a “ “
-mammals (incl. shrew)	$\ln(C_{mammals}) = 0.8188 * \ln(C_{soil}) - 4.8471$ $HR_{omnivores}$ (acres) = $0.59(BW)^{0.92}$	Sample *et al*., 1998b; USEPA, 2003b Sample *et al*., 1997a
- birds	I_{water} (l/kg/d) = $(0.059 * BW^{0.67})/BW$ 1) I_{food} (kg-dw/d/kg) = $(0.0582 * BW^{0.651})/BW$ all birds 2) I_{food} (g-dw/d) = $0.648 * BW^{0.651}$ (birds) 1) I_{food} (g-dw/d) = $0.398 * BW^{0.850}$ (passerine birds) 2) I_{food} (kg-dw/d/kg) = $(0.0141 * BW^{0.850})/BW$ (passerine birds)	Sample *et al*., 1997a Sample *et al*., 1997a Sample *et al*., 1996 Sample *et al*., 1996 Sample *et al*., 1997a
- plants	1) $C_{plants} = 0.03752 * C_{soil}$ 2) $\ln(C_{plants}) = (-1.992 \pm 0.431) + (0.564 \pm 0.125) * \ln C_{soil}$ [c]	Bechtel Jacobs, 1998; USEPA, 2003b Bechtel Jacobs, 1998

A = area (ha) contaminated, HR = home range size (ha) of endpoint species, E_j = total oral exposure to contaminant (j) (mg/kg/d), m = total number of ingested media (e.g., food, water, or soil), I_i = ingestion rate for medium (i) (kg/kg body weight/d or L/kg body weight/d), C_{ij} = concentration of contaminant (j) in medium (i) (mg/kg or mg/L); BW = body weight (kg)
[a]recommended for conservative assessment; [b]recommended for general estimates; [c]recommended both for conservative and general assessment (Obs. 1 acre = 4,047 m^2).

The results from exposure modelling are combined using similar equation than Eq. 1. i.e.:

$$HQ_{ib} = E_{b,As} / NOAEL_b \qquad (2)$$

where HQ_b = hazard quotient indicating the risk to an organism b living in environmental compartment i (here: soil); E_{As} = organism b's total exposure to arsenic; $NOAEL_b$ = No Observed Adverse Effect Level for organism b.

Since the tier 1 ERA was meant to yield preliminary, reasonable “worst case”, i.e., conservative risk estimates, conservative input values were used in the calculations. Therefore, the bioavailability was assumed to be 100 %. In the assessment of the exposure of biota in higher trophic levels (e.g., mammals, birds) to soil contaminants, the respiratory and dermal exposures were excluded since

generally these give only a minor contribution to the total exposure particularly in the case of non-volatile contaminants bound to soil particles. Firstly, respirable particles (>5 μm) are most likely ingested as a result of mucocilliary clearance rather than being inhaled (Witschi & Last, 1996). Secondly, the contribution of inhalation of contaminants associated with soil dust is expected to be less than 0.1 % of total risk compared to oral exposures (USEPA, 2003b). For most contaminants, the dermal exposure is expected to contribute less than one percent to 11 % of the total risk compared to oral exposures. Moreover, current information is insufficient to evaluate dermal exposure of contaminants in soil or to predict possible absorption for many species.

Shrew was selected as a proper receptor due to its abundance, availability of toxicity data and ecological importance (as a predator of soil animals and food source of predators on higher trophic levels, e.g. birds). Sample *et al*. (1996) have derived a NOAEL value of 0.15 mg/kg/d for the total arsenic intake of shrews. To define the parameter values concerning the characteristics and behaviour of shrews we used the data of the common shrew (*Sorex aureus*) which is the most abundant species in Finland and found commonly in varying habitats. In the literature, the home range of this species has been reported to vary between some 400 m^2 and 600m^2 while the calculation using the equation in Table 11 resulted in a value of ca. 34 m^2 (BW = 10 g). Hence, the home range of *Sorex aureus* is also suitable since it covers only part of the surface area of the study sites/areas and consequently, a single shrew can be expected to dwell entirely within our particular study site (or area). The species *S. aureus* is an insectivore feeding on earthworms, molluscs and different insects. However, since there was no data available to assess the concentration of contaminants in all these food sources, it was assumed that earthworms cover 100 % of the total diet. This can be considered as a conservative starting point owing to the earthworms' tendency to take contaminants from soil. In fact, the generic ecological benchmarks derived for shrews are often based on this assumption (e.g., USEPA, 2003b).

To assess the risks to birds due to secondary and tertiary poisoning (food and water), we used two different species which represent different trophic levels i.e., blackbird (*Turdus merula*, BW = 75.5-110 g) and Tengmalm's owl (*Aegolius funereus*, BW = 120-200 g). Blackbird is a common passerine dwelling even in residential areas and earthworms comprise a significant part of its diet while Tengmalm's owl is the most common owl species in Finland preying mainly small mammals. As a conservative assumption, we assumed owls to feed solely on shrews and blackbirds to feed entirely on earthworms. Soil ingestion was not included in the assessment of exposure since for birds, there was no such data available. Moreover, potential home range was not considered since the size varies considerably depending of various factors, e.g., availability of food, season, life stage and gender of the animal, and competitive species, among others. Hence, due to the lack of data only a screening level, tier 1 ERA based on the potential exposure of an individual bird was carried out. To characterize the risks, we used the general NOAEL value of 5 mg/kg-d presented for several bird species (Sample *et al*. 1996).

In addition to the horizontal factors i.e., the size of the home range (habitat) in relation to the size of the contaminated area, the vertical dimensions of pollution need to be considered in ERA. Different organisms forage in different parts of soil and hence, the depth has to be specified on the basis of the receptor (Table 12). Furthermore, in the selection of concentrations and parameter values, the starting point and the objectives of the ERA need to be taken into account. Generally, in a conservative assessment this means the use of the higher ends of the statistical variables e.g., in the case of normally distributed data the 95 % Upper Confidence Limits (UCLs) of the mean values of concentrations or in the case of other distributions, 90^{th} or 95^{th} percentile values (e.g. USEPA, 1989 and 1992). In those cases in which the number of samples was small (< 30), UCLs were determined based on the t-distribution. If the UCL value exceeded the maximum concentration measured, this

maximum concentration was used in the calculations in tier 1. In the case of naturally occurring As in topsoil, the primary data needed for the calculation of UCLs was not available since the concentration data were mainly produced in previous studies outside RAMAS. The concentrations used in the calculations are summarized in Table 13.

Table 12. Land use-specific soil layers relevant in ecological risk assessment.

Land use category	Receptor	Soil depth, m	Basis
Natural areas (meadows)	plants: grasses, scrub, microbes earthworms *Eisenia fetida* mammals (e.g., shrew), avifauna	0 - 0.5 0 - 2 topsoil topsoil	root depth, scrubs dwelling of all species[a] soil surface dwelling soil surface dwelling, direct contact (ingestion)
Natural areas (forest)	plants: trees and scrub, microbes earthworms mammals (e.g., shrew), avifauna	0 - 1.5 0 – 2 topsoil	root depth, trees dwelling of all species[a] soil surface dwelling, direct contact (ingestion)
Recreational amenities, parks	plants: trees, scrub, microbes, avifauna	0 – 1.5 topsoil	root depth, trees soil surface dwelling, direct contact (ingestion)
Agricultural areas	crop, cattle, soil organisms Eisenia fetida	0 – 0.3 topsoil	root depth, crops soil surface dwelling
Residential areas with gardens	plants: trees, scrub, grasses earthworms Eisenia fetida	0 - 1.5 0 – 2 topsoil	root depth, trees dwelling of all species[a] soil surface dwelling

[a]including soil surface dwelling (e.g., *Eisenia fetida*), topsoil dwelling and subsoil dwelling (e.g., *Lumbricus terrestris*) species

Table 13. Concentrations in soil used in the tier 0 and tier 1 ecological risk assessment. Only the most sensitive potential future land uses were considered.

Study site	Land use scenario	Concentration in soil, mg/kg	Receptors	Basis
CCA-plant, Ruovesi	- natural area - residential area with gardens	2500[a] (UCL)	all	only analysis from the sampling depth of 0.05 m available
Ylöjärvi mine site	- natural, forested area - recreational amenities, parks	2400 (max. conc.[b])	all all	only analysis from the sampling depth of 0.05 m available, tailings area
farms	- agricultural - natural area (meadows) - residential area with gardens - recreational amenities, parks	4.6 (UCL) 4.3 (UCL) 4.6 (UCL) 4.3/6.8[c] (UCL) 4.6/6.8[c] (UCL) 4.3/6.8[c] (UCL) 4.6/6.5[c] (UCL)	crops, *Eisenia fetida* plants, microbes, earthworms shrews, avifauna plants, earthworms *Eisenia fetida* microbes, plants, avifauna	plough layer all depths (plough layer and subsoil) topsoil (plough layer) all depths topsoil all depths topsoil
forest area	- natural area - recreational amenities, parks	44 (98%)[d1] / 198 (98%)[d2] 105 (98%)[d3] 44 (98%)[d1] / 198 (98%)[d2] 105 (98%)[d3]	plants, microbes, earthworms shrews, avifauna microbes, plants avifauna	all depths, whole Pirkanmaa topsoil all depths, whole Pirkanmaa topsoil

[a] max 4200, min 66, mean 1152, median 440 (Parviainen *et al*.. 2006); [b]UCL > max. concentration; [c]concentration based on the sampling on the field area (topsoil = plough layer) / forest area within the study farms (topsoil = 0…max. 0.35 m); [d]Backman *et al*. 2006: 1 = data from national till data (tier 1 ERA), 2 = data from ore prospect data (deeper soil layers, tier 0 ERA), 3 = data from ore prospect data (upper soil layers, tier 1 ERA)

Concentrations in surface water (Table 14) were assigned on the basis of previous monitoring studies in some areas with no anthropogenic As sources and around the Ylöjärvi mining area, and on the basis of the data collected within RAMAS (studies around the Ruovesi CCA plant, additional studies at the Ylöjärvi mine site). Following the principles of conservative risk assessment, we used the UCL values also in the ERA associated with aquatic ecosystems.

Table 14. Concentration of arsenic in surface waters around the study sites. sd = Standard Deviation, UCL = Upper Confidence Limit of the mean value, 95 %.

Concentration, µg/l	Mining area	CCA plant[a]	Natural areas
mean	75	49	1.6
max	315	49	3.6
sd	97	-	0.9
UCL	150	49	2.4

[a]only one measurement result available, sample taken from the stream flowing outwards from the site (Parviainen *et al.*, 2006)

In the tier 2 we included a statistical analysis in the assessment based on uptake and exposure models (plants, earthworms, shrews). This uncertainty analysis was carried out using Monte Carlo simulation technique and Crystal Ball© software. The number of simulations (n) was selected on the basis of the preferred certainty of the individual percentiles. Here, the following target was set: the median value should be within ± 2 percentile with 95 % confidence (Eq. 3).

$$n = 0.5 \times (1 - 0.5) \times (2 / 0.02)^2 = 2{,}500 \qquad (3)$$

The statistical data concerning the parameters in the models and characteristics of the study organisms were collected from the literature (for sources, see Table 11). The concentration data were fitted to log-normal distribution.

4.2.5 Ecotoxicological tests (Tier 2)

Ecotoxicological studies were used to collect complementary data to chemical analyses for ecological risk assessment (Table 15). The tests selected for these studies are described in detail in Appendix 2. Ecotoxicological laboratory tests measure harmful effects on test species at controlled standard conditions. For practical reasons, only a very limited number of species can be used in a single study. Therefore, test species serve as surrogates for a wide variety of species in the target environment. Observed responses are direct evidence of bioavailability and uptake of harmful agents. Recordings of observations are made after a certain period of time either on life threatening (lethal) effects, effects on growth, reproduction, behavior or biochemical changes. These different responses may be used as measurement endpoints for potential hazard. Effects may be presented for example, as percentage inhibition or as EC50- values (median effective concentration causing a defined adverse effect).

To study the phytotoxicity of metals, the root-elongation test is the most widely used method. In addition, determination of various parameters, such as germination, growth of seedlings, plant height, leaf number and area, pod number and length (in legumes), biomass production, dry matter production and reproduction have been used as indicators of phytotoxicity. A very specific effect is the phytotoxicity owing to inactivation of photosynthesis by heavy metals.

Table 15. Selection of measurement endpoints and test species and their connection to the ecological risks to be assessed.

Risks to be assessed	Assessment endpoint	Measurement endpoint	Test species
risks of soil-bound As to terrestrial plants	effect on the viability of 1) ornamental and natural plants (grasses) 2) edible plants	seed germination	1) ryegrass (*Lolium multiflorum*) 2) lettuce (L*actuca sativa*)
risk of soil-bound As to soil invertebrates	effect on the abundance of 1) earthworms 2) enchytraeids	mortality, reproduction (number of offsprings)	1) *Eisenia fetida* 2) *Enchytraeus albidus*
risk of soluble As (in pore water or leachate) to plants	effect on the viability of plants	inhibition of growth	duckweed (*Lemna minor*)
risks to microbes	general toxicity	inhibition of luminescence	*Vibrio fischeri*
risks to soil biota	general toxicity to biota	inhibition of enzyme activity *in vitro*	RET
risks of soil-bound As to soft-bodied soil invertebrates and organisms feeding on them	1) bioavailability to earthworm 2) exposure of shrew through food intake	concentration of As in earthworms	*Eisenia fetida*

Samples for ecotoxicity studies were collected at the specific hot spot areas containing naturally occurring or anthropogenic arsenic (see Section 4.2.2). The samples from these hot spot areas were analyzed for chemical concentrations of arsenic and metals using ammonium acetate and aqua regia digestions. Results of the chemical analyses of the soil samples are shown in Appendix 1. Ammonium acetate is assumed to reflect the easily leachable, i.e., potentially bioavailable, fraction of metals which is mainly responsible for biological effects.

Besides toxicity of contaminants their environmental fate is of concern when assessing the factual risks. Hence, the combination of leaching tests and ecotoxicity test with soil samples allows the derivation of some estimates of possible environmental risks in the future. The results from the batch leaching tests (documented in Parviainen *et al*. 2006) were studied in order to have an estimate of the temporal scale of leaching, and hence of future risks.

4.2.6 Study on algal species

The tier 0 level risk assessment concerning the aquatic ecosystem in the watercourse receiving effluents from the Ylöjärvi mine site was complemented with a study on the abundance of different diatom species in the sediment.

Diatoms (*Bacillariophyceae*) are microscopical, single-celled photosynthetic algae. They have long been used in paleolimnological research to indicate eutrofication level (e.g., Alhonen, 1972). The facts that diatoms are naturally abundant in all aquatic environments, their shell is well preserved in geological formations and the different species typical to specific envionmental conditions are usually recognizable, make diatoms as good indicators in environmental studies related to anthropogenic emissions. When environmental conditions change e.g., owing to eutrophication, acidic emissions or heavy metal load, the composition of diatom taxa changes. This change can be observed by studying the shells deposited in the sediment.

In the RAMAS project, the purpose was to study whether there are changes in the composition of diatom (algal) species which could be linked with some differences in the chemical composition such as the concentration of arsenic and nutrients. For this purpose, a sediment core was retrieved from lake Vähä-Vahantajärvi located 3 km downstream from the mine site (see e.g., Parviainen *et al.* 2006). This core was studied for both elemental chemistry and remains of diatoms (*Bacillariophyceae*). The core covered a time span from the pre-mining period till the day of sampling. Hence, the study material included also samples that were deposited during the active mining period with presumably higher As loading than at present. This means that the relationship between the composition of algal species and environmental factors was derived for the whole time period instead of only the situation during sampling (i.e., present situation).

4.2.7 Derivation of risk scores and risk characterization

The tier 0 and tier 1 ERA generated risk estimates which can readily be compared with the acceptable concentration and intake levels. Generally, in the case a single contaminant a value of HQ $\leq$ 1 is considered as acceptable i.e., to indicate insignificant risks. If several contaminants are present the sum of contaminant-specific HQs (i.e. hazard index, HI) should be equal or below 1. To take into account possible risks owing to other, non-quantifiable contaminants, HQ = 0.1 has also been used in some instances (e.g., State of Maryland, 2001).

Since besides arsenic, there are several concurrent contaminants present in the environment, it was necessary to consider the possibility that these other contaminants may also pose ecological risks. Particularly in the case of a CCA wood treatment plant the presence of other metals, e.g., copper and chromium, can not be ignored in the risk assessment. To study the toxic potency, i.e., the contribution of these to the overall ecological risks, HQs were calculated for these, too, using the benchmarks presented in table 16.

Table 16. Ecological benchmarks (mg/kg) used in the study of toxic potency of different contaminants. BM = Benchmark, EIV = Serious Risk Concentration, RIVM = Rijksinstituut voor Volksgezondheid en Milieu (National Institute of Public Health and the Environment, the Netherlands), CCME = Canadian Council of Ministers of the Environment, RC = Remediation Criterion, PRG = Preliminary Remediation Goal, HC5 = Hazardous Concentration, i.e., concentration which is expected to cause adverse effects to 5% of the organisms. NA = not available

Receptor	Description	Al	As	Cd	Cu	Co	Cr	Fe	Ni	Pb	Zn
Microbes	BM[a], USA	600	100	20	100	1,000	10	200	90	900	100
Earthworm	BM[a], USA	NA	60	20	50	NA	0.4[h]	NA	200	500	200
Terr. plants	BM[b], USA RC[c], CCME	50 NA	10 20	4 3	100 150	20 40	1 750	NA NA	30 150	50 375	50 600
Terr. biota	PRG[d]	NA	9.9	-	60	20	0.4[h]	NA	30	40.5	8.5
Soil biota	SRC[e], RIVM HC5[f], RIVM	NA NA	85 0.9	13 0.79	96 3.4	180 2.4	220 0.38[i]	NA NA	100 0.26	580 55	350 16
Mouse	PRG[g]	NA	149	63	10,100	NA	880	NA	1,830	6,250	35,000
Shrew	PRG[g]	NA	9.9	6	370	NA	110	NA	246	740	1,600

[a]Efroymsson *et al.*, 1997b; [b]Efroymsson *et al.*, 1997c; [e]Verbruggen *et al.*, 2001; [c]agricultural area (Efroymsson *et al.*, 1997b); [d]including secondary poisoning (Efroymsson *et al.*, 1997a); [f]Swartjes, 1999; [g]Efroymsson *et al.*, 1997a; [h]low confidence; [i]Cr(III)

To determine the contribution of arsenic in relation to the concomitant contaminants, we assumed that the combined toxic effects are strictly additive and calculated hazard indexes (HI) (Eq. 4).

$$HI = \sum_{j=1}^{n} HQ_j \qquad (4)$$

This methodology is equivalent to the determination of Toxicity Units (TUs) for the identification of Contaminants of Potential Concern (COPCs) (documented e.g., in Suter, 1996). Instead of using the standard test end point concentrations recommended for the definition of TUs, we used benchmarks (BMs) referring rather to effect levels than to probable no-effect concentrations (PNECs), e.g., NOEC (No Observed Effect Concentration) values, in the calculation of HIs (and HQs on which they are based). The primary reason for this was that the information on the toxicity varies significantly in different literature sources making the selection of the toxicity values difficult and reducing their comparability with each other. This problem was minimized by using sets of BMs in which the basis and method of derivation was consistent, and hence would include values comparable with each other. Since our interest was on the comparison of the contribution of each relevant contaminant to the overall ecological risk and not on the determination of risk estimates, i.e., absolute values of HQs/HIs, this was seen as a suitable methodology.

In addition to the HQ methodology, we adopted an alternative methodology to produce risk estimates which consider all COPCs. This PAF (Potentially Affected Fraction of species) methodology is based on the calculation of the substance-specific Toxic Pressure (TP_j) values (Eq. 5) which are further combined to give a TP_{combi} value (Eq. 6) (e.g., Rutgers *et al*. 2005; Jensen & Mesman, 2006).

$$TP = \frac{1}{1 + e^{(\log HC50 - \log C_j)/\beta}} \qquad (5)$$

$$TP_{combi} = 1 - ((1 - TP_1) \times (1 - TP_2) \times \ldots \times (1 - TPj)) \qquad (6)$$

where HC50 (Hazardous Concentration) is the concentration which is expected to cause adverse effects on 50 % of the species in soil and β is slope factor of the Species Sensitivity Distribution (SSD) curve, i.e., the response curve representing all species. In the case of metals, the value of 0.4 can be used as an assumption for β (e.g., Jensen & Mesman, 2006). In the calculation of the TPs we used the lowest of the HC50 values reported by the Dutch RIVM (Swartjes, 1999).

The effect of concomitant contaminants also appears when the test organisms are exposed to field samples in ecotoxicity tests. Therefore, the actual causal relationships between the concentrations of arsenic and the concomitant contaminants and measurement endpoints were studied in more detail. The As-specific effects on toxicity were determined using an elaboration model based on covariance analysis parameters (SPSS software, version 11). The primary aim was to find out whether arsenic was actually the main stressor causing the toxic response.

In the case of ecotoxicological tests, the interpretation of the results is not straightforward due to the variability of different test results and the lack of clear definition for the acceptable risk level. To give an idea of the possible overall ecological effects rising from the toxicity of environmental media including high levels of arsenic (and possibly other contaminants), the results from different ecotoxicological test can be combined using scoring to give an integrated risk estimate (e.g., Jensen & Messman, 2006). The scoring method is selected on the basis of the type of the risk assessment (Eq. 7a&b) or measurement endpoint (8a-c):

<u>Risk scores derived from the hazard quotients for shrews</u>

$$R_{HQ} = 1 - \frac{1}{(1 + HQ)} \quad (7a)$$

$$Score_{HQ} = \frac{R_{HQ} - R_{background}}{1 - R_{background}} \quad (7b)$$

where $R_{background}$ is the R_{HQ} corresponding the natural background concentration of arsenic. The concentration was defined on the basis of the median concentration in till fines reported for the whole country (1 mg/kg, source: Ministry of the Environment, 2007).

In the case of ecotoxicity tests, the method to calculate risk scores depends on the toxic response (R_t). Results from toxicity tests, negative response (e.g., inhibition of growth):

$$Score_{tox} = \frac{R_t}{100} \quad (8a)$$

Results from toxicity tests, positive response (e.g., survival of earthworms):

$$R_{tox} = \frac{(100 - R_t)}{100} \quad (8b)$$

$$Score_{tox} = \frac{R_{tox} - R_{tox.ref}}{1 - R_{tox,ref}} \quad (8c)$$

where ’ref’ refers to the reference soil (contains no arsenic). The risk scores based on chemical studies (TP, $Score_{HQ}$) and the scores from the ecotoxicity tests ($Score_{tox}$) were combined to total risk scores as follows:

$X = \log(1\text{-Score})$ or $X = \log(1\text{-TP})$ (9a)

Total risk score $= 1 - 10^{(\Sigma X)/n}$ (9b)

where n = the total number of different risk scores (i.e. TPs and $Score_{HQ}$ –values or $Score_{tox}$ -values).

The uncertainty of the integrated risk estimates was studied using the same method as in the probabilistic assessment based on uptake and exposure models (see Section 4.2.4. and Eq. 3). In the case of the results from ecotoxicity tests, we used triangular distributions with the high and low ends set according to the highest and lowest response values detected while the center points were fixed with the mean values calculated from the results representing parallel samples. The determination of the distribution of the concentrations and parameter values involved in the calculation of risk estimates based on uptake and exposure models is described in Section 4.2.4.

Since no ecological studies were carried out within RAMAS, it was not possible to follow the TRIAD approach (described e.g., in Jensen & Messman, 2006). Consequently, no integrated risk estimates were generated from the results representing different ERA methods (i.e., assessment based on chemical studies and ecotoxicity tests). Instead, separate risk scores were produced and compared with each other. Moreover, the results from modelling using uptake models were compared with the results from the determination of bioavailable arsenic in earthworms to find out the suitability of the models.

The exposure estimates for shrews derived using modelling were also converted to population level assuming a distinct population on the site so that the exposure of the population is represented by the exposure of all of the individuals. In this extrapolation method, all individuals at the site are assumed to experience equivalent exposure. This assumption is appropriate for small organisms with limited home ranges on large sites, particularly if the site constitutes a distinct habitat that is surrounded by inappropriate habitat (e.g., Sample *et al*. 1997a). For example, a grassy site surrounded by forest or industrial development might support a distinct population of voles. The risks to that population can be estimated directly from the exposures of the individual organisms. There are several more sophisticated methods available to assess population-level risks, however, these methods assume site-specific data on the size of the suitable habitat within the contaminated area or on the wildlife exposure from multiple, spatially disjunct areas and data to weight the potential exposure at each area or the preference of the available habitats (e.g., based on the availability of food).

4.3 Human health risk assessment

4.3.1 Methodology and methods

The assessment of risks to human health was based on different methods. Firstly, we assessed the potential daily As intake in different exposure scenarios. Here we used generic models depicting contaminant intake from different environmental media. The estimates of daily dose were further compared with reference values i.e., acceptable daily doses (ADDs), to produce quantitative risk estimates. This risk assessment based on exposure models was carried out in two phases. In Phase 1 the daily dose from drinking water was calculated using the preliminary results (see Section 4.1.2) from analyses of arsenic in well water samples. Background exposure, i.e., exposure from other than site-specific sources, e.g. food, was estimated from national level data. In Phase 2, the risk assessment was refined with inclusion of the new concentration data gathered in RAMAS project. Here, the potential exposure arising from the key anthropogenic hot spot areas i.e., mine sites and CCA wood treatment plants, was also considered. The primary calculations were based on the highest As levels in order to cover the "worst case" exposure scenarios.

To complement the data on the health risks assessed by modelling and to verify potential exposure and risks in population scale, we carried out a separate biomonitoring study and an epidemiological study. The biomonitoring study was run by the Finnish Institute of Occupational Health and the Finnish Environment Institute. The Environmental Epidemiology Unit of the National Public Health Institute was responsible for the realization of the epidemiological study.

The biomonitoring study was carried out by monitoring the As concentration in the urine of selected residents in Pirkanmaa. The study population was selected from the households who had permitted environmental sampling in their property during RAMAS project. This material was complemented

with other types of households. At the end, the total study population comprised both households which used their own wells as a source of drinking water including varying concentrations of arsenic, but also households which were connected to a public water supply system or had another alternative source of drinking water (for example bottled water). The residents were contacted separately to ask for their willingness to participate in our biomonitoring study. The study population covered finally 40 persons representing 15 households. The methods and results of the biomonitoring study are described in more detail in a separate report in Appendix 3.

The epidemiological study was based on the spatial analyses of As-related cancer risk incidence within the whole Pirkanmaa region. The argument was that in long term elevated As concentrations in household water are reflected as a higher incidence of certain cancer types compared with the population not exposed to arsenic through household water. Therefore, the cohort, i.e. the study population fixed into a certain study year, has to be selected so that the development of cancer could appear in statistics. Moreover, the probability of the use of private wells had to be considered since we did not have exact data on the number of wells used as a source of household water nor the information on the households/persons who had used private well water during the time period studied. For the analyses, the data on the As concentration in ground water collected within RAMAS were mapped using grids suitable for the spatial epidemiological analyses. The methods and results of the epidemiological study are described in more detail in a separate report in Appendix 4.

4.3.2 Preliminary conceptual models

In the case of local arsenic sources, the intake profile may be significantly different from the mean human population (see Section 4.3.4). In addition to food, drinking water, soil ingestion and inhalation may be significant contributors to the total exposure. In Pirkanmaa area, arsenic in drinking water is expected to be the dominating source of human exposure to inorganic arsenic. In addition, the ingestion of contaminated plants (potatoes, tuberous vegetables, mushrooms) was considered a possible exposure route. Arsenic is typically not transported from soil to above ground plant parts in remarkable quantities, but significant contamination of cattle fodder was considered possible. Due to the the contaminated fodder, also meat and milk products could act as secondary pathways of human exposure to soil arsenic. A preliminary conceptual model for a farm located at the area with high concentration of natural arsenic is presented in Fig. 4. In addition to the exposure routes included in Fig. 4, background exposure e.g., from rice and marine fish, must be taken into account when estimating the total intake.

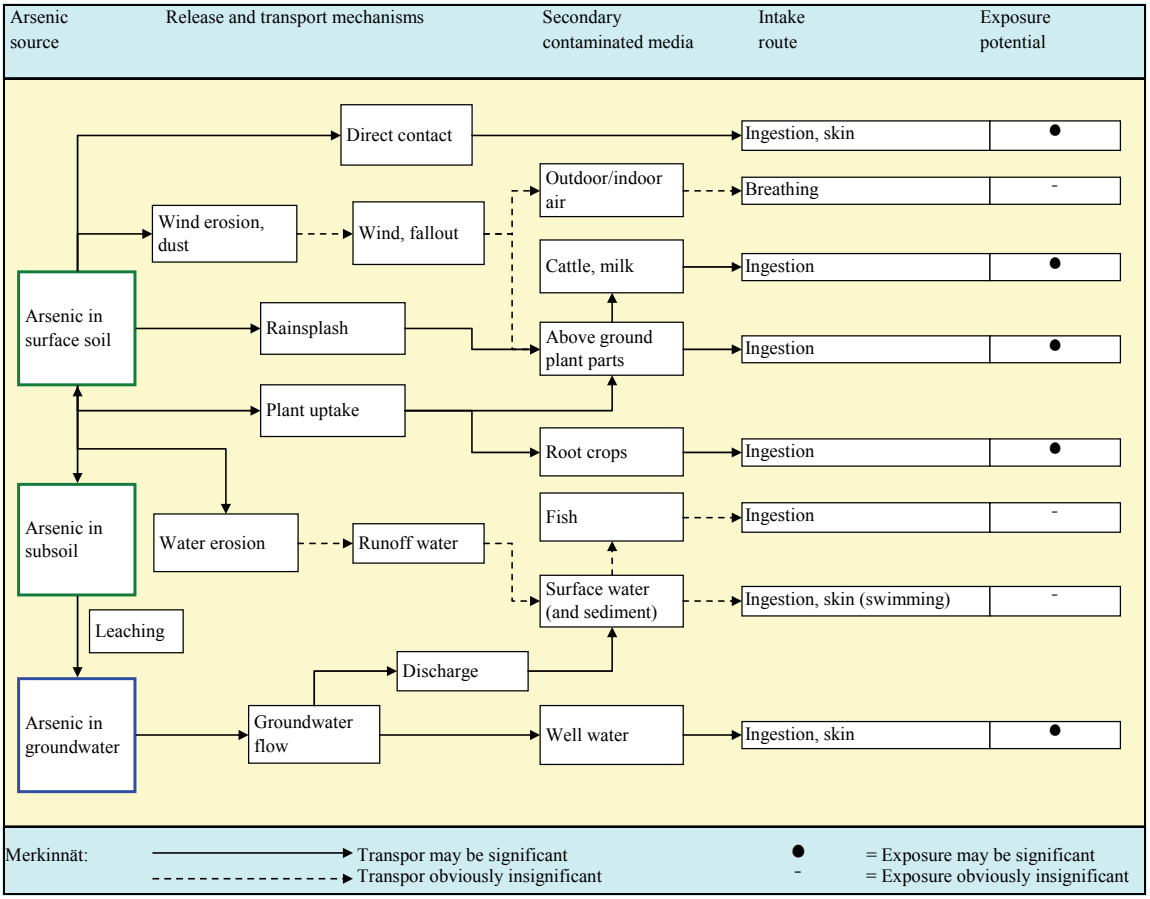

Figure 4. A preliminary conceptual model describing the potential exposure of a farm resident to local, naturally occurring environmental arsenic.

Since children generally consume less food and beverages than adults, ingestion of contaminated food or juice or infant formula containing As-contaminated water may represent a significant source of exposure. In addition, due to the unintentional ingestion of soil/dirt related to hand-to-mouth behaviour of small children (age 1-6 years), ingestion of contaminated soil may be a more important source of As exposure compared with that of adults.

Since former wood treatment sites are typically spatially limited, they are not expected to pose a risk of contaminating crops or cattle fodder. On the other hand, direct contact to arsenic in surface soil was anticipated to be particularly important at anthropogenically contaminated sites, in which the concentrations of As in surface soil may be high. Particularly living in residential areas built at such sites may pose considerable risks to human health if no remedial actions are carried out. At anthropogenically contaminated sites, the concentrations in soil and groundwater may be unbalanced and may therefore change with time.

The old mine sites studied in the RAMAS project cover rather large areas extending up to adjacent water systems. The open tailing areas are susceptible to wind erosion and may cause contamination of air. Currently, the former Ylöjärvi mine site is used daily by the Finnish army to destroy expolosives and materials used in fireworks. These activities enhance the distribution of soil dust in air. Hence, it is important to consider possible occupational risks.

4.3.3 Dose-response modelling

In the assessment of average daily As doses, we considered all relevant exposure scenarios. These included the following:

- exposure associated with living in the countryside,
- exposure of people living at a former CCA site,
- occupational exposure at the old mine site (Ylöjärvi).

Intake routes considered in the Phase 2 assessment included:

- ingestion of groundwater,
- general background exposure from food,
- soil ingestion,
- dermal intake through skin contact with surface soil and
- inhalation (only occuptational exposure at the mine site).

The daily doses associated with the above-mentioned exposure routes were calculated by using generic equations presented e.g., by USEPA (1989):

$$\text{Ingestion and inhalation, dose (µg/kg/d)} = \frac{C \times CR \times ABS \times EF}{BW}$$

$$\text{Dermal contact, dose (µg/kg/d)} = \frac{C \times A \times SA \times ABS \times EF}{BW}$$

As concentration in the env. medium	C	µg/g, µg/m^3
Ingestion rate, inhalation rate	CR	0,05 g/d, 20 m^3/d
Absorbed fraction	ABS	medium-specific
Exposure frequency	EF	d/365 d
Body weight	BW	70 kg
Soil adherence to skin	A	5×10^{-4} mg/cm^2
Skin contact area	SA	1700 cm^2

The calculated daily doses were compared with the toxicity-based acceptable daily doses (ADDs) issued by different organizations (see Section 4.4.5). ADDs represent an estimate of the daily human exposure to a substance that is assumed to be safe, i.e., without an appreciable risk of adverse health effects over a specified duration of exposure. For noncancer effects, the default assumption is that the dose-response model has a threshold below which no adverse health effects are expected to occur. Noncancer risks were expressed as hazard quotients (HQ):

$$\text{Non-cancer-based hazard quotient HQ} = \frac{CDI}{ADD}$$

Chronic daily intake of arsenic	CDI	mg/kg/d
Acceptable daily dose	ADD	mg/kg/d

The cancer risks (skin and liver cancer) associated with As intake were further determined on the basis of USEPA's linear model. The cancer slope factor estimates the excess upper-bound lifetime probability of an individual developing cancer owing to a lifetime (70 year) exposure. In the case of the Ylöjärvi mine site risk for lung cancer was calculated from the measured arsenic concentration

in the air and the unit risk value[3] (UR) for As concentration in air. The UR value represents the excess cancer risk over background associated with continuous lifetime exposure to a pollutant and is typically expressed as risk or probability of cancer from lifetime exposure per 1 µg pollutant/m^3 air (EPA, 1986). The excess cancer risk is calculated as follows:

Individual cancer risk from chronic arsenic intake $= CDI \times SF_o$		
Individual cancer risk from exposure to As in air $= C_A \times UR_{inh}$		
Oral slope factor	SF_o	$(mg/kg/d)^{-1}$
Average As concentration in air	C_A	$\mu g/m^3$
Unit risk value for inhalation	UR_{inh}	$(\mu g/m^3)^{-1}$

In addition to deterministic calculations, we carried out a probabilistic assessment of the As intake using Crystal Ball® software (Decisioneering, Inc., Denver, CO, U.S.A.). Here we used 20,000 runs of Monte Carlo simulation. Crystal Ball® calculations were executed to estimate distributions of As concentration in water. Average values and standard deviations of water use metrics were taken from the national diet study (Männistö *et al*. 2003). Distribution of water intake was assumed to be lognormal (e.g., Roseberry & Burmaster, 1992) and fully characterized by the mean and variance. The exposure parameters were assumed independent, i.e., possible correlations we ignored due to lack of data.

In the case of the CCA treatment plant site, the As concentration detected in the groundwater does not necessarily represent the contamination level because migration of As to the groundwater is a slow phenomena and measurement points may not be situated at centerline of the plume. Therefore, the measured As concentration was compared to the theoretical maximum of As concentration in groundwater. The potential leaching of arsenic from soil to groundwater was calculated by applying the so-called RBCA standard principles (ASTM, 1995) as follows:

Leaching of arsenic to groundwater (g/d) $= \frac{C_s \rho_s}{[\theta_{ws} + K_d \rho_s]} \times AI$		
Arsenic concentration in unsaturated soil	C_s	mg/kg
Soil-water partition coefficient	K_d	l/kg
Water-filled soil porosity	θ_w	vol/vol (default 0,2)
Dry soil bulk density	ρ_s	kg/l (default 1,6)
Size of the contaminated area	A	m^2
Infiltration rate	I	m/d (default $8{,}2 \times 10^{-4}$)

Because As compounds present in soil are virtually non-volatile, the component ‘soil air’ was omitted from the equation. The key parameter in the model is the soil-water partition coefficient which was calculated from the leaching test results (documented in Parviainen *et al.* 2006) as a ratio of solid to eluate concentrations.

[3] risk estimate for a lifetime exposure to a concentration of 1 µg/m^3

4.3.4 Input data for dose-response modelling

In the Phase 1 health risk assessement the main focus was on the ingestion of As-containing water. The main components of ingested water are plain drinking water, beverages made with water (coffee, tea etc.) and water in foods (e.g. soup, oatmeal). Average daily consumption of water and different food items among the adults has been studied in Finland in 2002 (Männistö *et al*. 2003) (Table 17). Average body weight was set to 70 kg which according to this dietary study, is somewhat less than the true average.

Table 17. Average intake of water from different fluids and foods in Finland in 2002 (Männistö *et al*., 2003). Water in foods was estimated from the volume of consumed soups and oatmeals.

Intake route	Average consumption l/d	Standard deviation l/d
Drinking water (plain water)	0.63	0.57
Coffee	0.47	0.37
Tea	0.12	0.22
Home made juices	0.09	0.19
Water in foods	0.2	0.39
Total water consumption	1.51	

Food is typically the most important additional sources of arsenic. The National Public Health Institute has estimated that the total dietary intake of inorganic arsenic in Finland is 10 – 20 µg/d (KTL, 2006). This corresponds to 0.14 – 0.29 µg/kg/d in the of the average body weight of 70 kg. In Phase 1 we used the upper estimate of the background intake (0.29 µg/kg/d).

In the Phase 2 HRA, more detailed data was used to calculate the background exposure related to food. In this calculation, we took into account the intake of grain cereals, fish and shellfish, root crops, meat and poultry, milk and dairy products, vegetables, fruit and berries, eggs and mushrooms. Since the studies carried out in RAMAS on crops, sap, berries and mushrooms revealed no differences between the concentrations in the study area and other parts of the country, we used the nationwide data on As concentrations to calculate background exposure to inorganic arsenic originating from food items (Table 18).

Table 18. Average inorganic arsenic intake from different foods in Finland. Food consumption data from Männistö *et al*. (2003). Arsenic bioavailability (fraction absorbed) from food was assumed to be 100 %.

Food item	As concentration µg/kg Fw	Fraction of inorganic As	Consumption g/d	Inorganic As intake µg/d
Cereals (exl. rice)	0.02	0.65	315	3.93
Rice	0.24	0.65	12	1.80
Fish and shellfish	0.5	0.05	28	0.67
Root crops	0.01	1	200	1.92
Meat and poultry	0.01	0.5	200	0.96
Milk and dairy products	0.0012	0.5	430	0.25
Vegetables	0.01	0.5	100	0.48
Fruit & berries	0.01	0.5	150	0.72
Local wild berries	0.01	0.5	50	0.24
Egg	0.001	1	18	0.02
Mushroom	0.05	0.5	1	0.02
Total				11.01

Besides water and food, in some cases soil can be a significant medium contributing to the total exposure to contaminants. Soil ingestion was taken into account in both the assessment of health risks owing to naturally occurring arsenic (Table 19) and anthropogenic arsenic originating from the CCA-chemical (Table 20).

Table 19. Estimate of inorganic arsenic intake from other media but water.

Intake route	As concentration mg/kg Dw	Soil quantity g/d	Fraction absorbed	Exposure frequency d/a	Average As intake g/d
Soil ingestion	6	0.05	0.25	255	0.06
Soil, dermal uptake	6	0.85	0.01	255	0.04
Food					11.0[a]
Total excluding water					11.1

[a]see Table 18 for the calculation of the value

Table 20. Calculation of arsenic intake from direct contact with contaminated soil at the CCA sites studied.

	Site			
	Ruovesi I	**Ruovesi 2**	**Vilppula**	**Virrat**
Average As concentration in soil mg/kg	1152	1474	82	220
Ingested soil quantity g/d	0.05	0.05	0.05	0.05
Fraction absorbed of ingested soil	0.25	0.25	0.25	0.25
Soil adhered to skin g/d	0.85	0.85	0.85	0.85
Fraction absorbed of soil on skin	0.01	0.01	0.01	0.01
Exposure frequency d/a	225	225	225	225
Average As intake g/d	0.21	0.27	0.02	0.04

The contribution of inorganic arsenic in different media and the proportions of bioavailable As used in the exposure calculations in Phase 2 are summarized in Table 21.

Table 21. Share of the inorganic arsenic and bioavailability values (fractions absorbed) used in the calculation of daily As doses.

Exposure medium	**Fraction of inorganic As**	**Basis**	**Bioavailability**	**Basis**
Grain products	65 %	Williams *et al*, 2005; Schoof *et al.* 1999	100 %	Pomroy *et al*. 1980; Freeman *et al*. 1995
Potatoes and root vegetables	100 %	Burló *et al*. 1999, Muñoz *et al*. 2002; Helgesen & Larsen 1998	100 %	Pomroy *et al*. 1980; Freeman et al. 1995
Fish, shellfish and fish products	5 %	FSA 2005; Schoof *et al*. 1999	100 %	Pomroy *et al*. 1980; Freeman *et al*. 1995
Mushrooms	50 %	literature e.g. Byrne *et al*., 1995: arsenic species vary depending on the taxa	100 %	Pomroy *et al*. 1980; Freeman *et al*. 1995
Animal crops	50 %	scarce data, see eg. Schoof *et al*. 1999	100 %	Pomroy *et al*. 1980; Freeman *et al*. 1995
Ingested soil	100 %	Backman *et al* 2006; Parviainen *et al*. 2006	25 %	literature, e.g. Roberts *et al*. 2002
Soil adhered to skin	100 %	Backman *et al* 2006; Parviainen *et al*. 2006	1 %	low solubility + e.g. Wester *et al*. 1993
Soil dust from the air	100 %	Backman *et al* 2006; Parviainen *et al*. 2006	50 %	literature, e.g., WHO, 2001
Drinking water	100 %	Backman *et al* 2006	100 %	literature, e.g., WHO, 2001

4.3.5 Risk characterization

Acceptable daily doses used in the derivation of risk estimates (for equations, see previous Section 4.4.3) vary in different countries and it is not possible to define a definitely proper value (see e.g., Provoost *et al*. 2006). As there are many sources of arsenic in food items with very different toxicity, it is important to differentiate between the species in order to evaluate the impact of arsenic on human health. The necessity of speciation inorganic/organic As is already reflected in some limit values. The "Joint Expert Committee on Food Additives and Contaminants" has suggested a PTDI-value (Provisional Tolerable Daily Intake) of 2 µg inorganic As/kg body weight (JECFA, 1983). This PTDI was derived from estimated LOAEL[4] value for chronic intake of 100 µg arsenic/l in drinking water, assuming a daily intake of drinking water of 1.5 litres.

In the IRIS-database maintained by USEPA, the acceptable daily dose (i.e., reference dose, RfD) for chronic oral exposure has been set to 3 $x10^{-4}$ mg/kg-day (EPA, 2006). The critical effects considered cover hyperpigmentation, keratosis and possible vascular complications, for which a NOAEL of 0.0008 mg/kg-day was derived. An uncertainty factor of 3 was used to account for both the lack of data to preclude reproductive toxicity as a critical effect and the potential uncertainty in the coverage of all sensitive individuals in the NOAEL value. Confidence of the RfD was considered as medium. The revision of the oral RfD is currently ongoing.

The Dutch Institute for Public Health and the Environment (RIVM) derived a tolerable daily intake (TDI) of 1 $x10^{-3}$ mg/kg-day for critical skin effects upon humans (Baars *et al*. 2001). This value was based on a NOAEL of 0.0021 mg/kg-day used in the derivation of the provisional maximum tolerable weekly intake (PTWI) by the World Health Organization. RIVM used an uncertainty factor of 2 to compensate for observation errors in an epidemiological study.

The World Health Organization (WHO) has estimated that the unit risk for As-induced lung cancer is 1.5×10^{-3} (WHO, 2000). It is difficult to translate such data into a limit value because the European Union has not established criteria for the 'acceptable risk'. EU has neither presented any data on dose-response relationship for arsenic.

USEPA has derived an oral cancer slope factor of 1.5 $(mg/kg/d)^{-1}$ and a drinking water unit risk of 5×10^{-5} $(\mu g/l)^{-1}$ for inorganic arsenic based on human dose-response data. The unit risk for cancer associated with inhalation is 4.3×10^{-3} $(\mu g/m^3)^{-1}$. These values are based on extrapolation method which is time- and dose-related formulation of the multistage model (USEPA, 1988). USEPA is currently revising the assessment for inorganic arsenic.

Limit values for total arsenic have been established only for drinking water (0.01 mg/l, WHO, 1993). This guideline value is based on the PTWI-value (Provisional Tolerable Weekly Intake) of 15 µg/kg assuming a 20 % allocation to drinking water. In the last update of the guideline, WHO (2006) commented that: “In view of the significant uncertainties surrounding the risk assessment for arsenic carcinogenicity, the practical quantification limit in the region of 1–10 mg/l and the practical difficulties in removing arsenic from drinking-water, the guideline value of 10 mg/l is retained. In view of the scientific uncertainties, the guideline value is designated as provisional.” No limit values exist for food products within the European Union.

Since there is no definite ‘right’ acceptable daily dose for human toxicity to arsenic, all the values described above were used in the determination of risk estimates.

[4] Lowest Observed Adverse Effect Level

5 RESULTS – ECOLOGICAL RISKS

5.1 Risks to terrestrial biota

5.1.1 Risk estimates from Tier 0

The risk estimates from tier 0 ERA are shown in Table 22. To determine these we used the maximum concentrations and the calculated Upper Confidence Limit values (UCLs) of the concentrations for each site type. The HQs were produced using the benchmarks presented in Table 16 (Section 4.2.7).

Table 22. Variation of the tier 0 risk estimates for ecological risks in different site types: Hazard Quotients (HQs) calculated on the basis of different benchmarks. HQ1 = HQ based on the Upper Confidence Limit of As concentration (95%), HQ2 = HQ based on the maximum As concentration.

Site/area studied	HQ1	HQ2
CCA plant	2-2700	4-4700
Ylöjärvi mine site	-	2-2600
farm, fields	0.004-5	0.006-8
farm, forest	0.006-8	0.01-16
natural As	0.18-220[a]	8-10,000

[a]based on the 98 % UCL, 95 % UCL was not available

Table 22 shows that the lowest benchmarks presented in the literature are exceeded in all site types representing the identified hot spot areas. The highest risk estimates (HQs) refer to the benchmark of 0.9 mg/kg representing the HC_5 value for soil species. The derivation of the HC_5 value includes high uncertainty and hence, it cannot be considered as a good indicator of overall ecological risks. At farm areas, ignoring of this benchmark results in the maximum HQs of 0.7 and 1.2 when calculated on the basis of UCL and maximum concentration, respectively. Hence, ecological risks associated with arsenic are expected to remain insignificant in farm areas. In other site types, ecological risks may be significant and hence a more detailed assessment is needed.

5.1.2 Risk estimates from Tier 1

The tier 1 ERA based on conservative (see Section 4.2.4) assumptions resulted in very high risk estimates (HQs) and uptake and exposure estimates in the case of the former wood treatment plant and Ylöjärvi mine site (Table 23).

Table 23. Results from tier 1 ecological risk assessment associated with environmental arsenic: risk estimates (Hazard Quotients, HQs), concentrations (C) and daily intakes (E) of selected target receptors. Subscripts 1 and 2 refer to the results from different uptake and exposure models while subscript max refers to the highest of the two exposure estimates based on different exposure models.

	Shrew	**Plants**	**Earthworm**		**Shrew**	**Blackbird**		**Owl**	
Site	HQ	C mg/kg	C_1 mg/kg	C_2 mg/kg	C mg/kg	E_1 max mg/kg/d	E_2 max mg/kg/d	E_1 mg/kg/d	E_2 mg/kg/d
CCA plant	910	14	210	440	470	1.6	0.058	0.036	0.40
mine site	1,600	13	200	430	460	1.6	0.056	0.034	0.38
forest area[a]	19	0.85	3.7	13	8.5	0.048	0.0017	0.00064	0.0071
forest area 2[b]	21	1.1	5.1	17	12	0.064	0.0023	0.00088	0.0098
farm, forest area	4.6	0.24	0.57	2.6	1.3	0.0094	0.00033	0.000099	0.0011
farm, meadow	3.8	0.17	0.36	1.7	0.82	0.0062	0.00022	0.000062	0.00069

[a]calculation based on the concentrations from the national till data (see Table 13 for concentration)
[b]calculation based on the concentrations of soil samples taken for ecotoxicity tests (see Table 6 in Appendix 1 for concentration)

The use of the generic reference value of 5 mg/kg/d (NOAEL) for birds leads to risk estimates below 1 and hence, risks related to secondary poisoning of birds are expected to be minimal. However, it has to be noted that the NOAEL value refers to total exposure from all possible intake routes and therefore, with the contribution of other possible exposure routes such as, soil ingestion, the actual total intake can be higher. In the case of owls, the benchmark values vary from 9.2 to 43.9 mgAs/kg in food and 32.8-78.4 mgAs/l in water depending on the owl species (Sample *et al.* 1996). The calculated concentration in shrews dwelling at the CCA plant or Ylöjärvi mine site clearly exceed even the highest reference value for diet. Water is not expected to contribute significantly in the total exposure since the concentration of As in surface water is clearly below the highest reference value at all our study sites/areas. It is noteworthy that birds are expected to have a wide home range and consequently, to feed only partially within a limited area such as a CCA plant. Consequently, the assumption that owls would feed solely on shrews and blackbird solely on earthworms living at a particular contaminated site is very conservative. In addition, uncertainty is involved in the reference values, too, since they have been derived from the test animal data using different conversion coefficients and there are significant differences in the feeding habits and physiology of different birds. Competition and availability of other food sources also affect the actual exposure to contaminated food. Finally, the concentration in shrews was assessed by using a regression model based on empirical studies on the correlation between the concentration in soil and concentration in shrews. Such a model can involve significant uncertainties.

The use of uptake models in the assessment of As concentrations in plants resulted in concentrations which imply risks to herbivores. Sample *et al*. (1996) have presented the following benchmarks for the food consumed by small herbivores: 0.881 mg/kg (mouse) and 1.008 mg/kg (vole). The respective reference values for water are 0.454 and 0.84 mg/l. The estimated concentrations in plants growing at the CCA plant or mine site clearly exceed the reference values

for food whereas the concentrations in surface water are below the reference values for water at all study sites/areas. Hence, food is expected to be a major source of exposure to arsenic.

Considering all the uncertainties and the conservative starting point of tier 1, from the target receptors considered only shrews and other small mammals are expected to experience significant risks. The estimated concentrations in earthworms are also high indicating possible adverse effects in some soil invertebrates. Birds can be omitted from further studies while a more realistic assessment (tier 2) needs to be carried out in the case of shrews (and earthworms belonging to their diet). Plants also deserve a more detailed assessment particularly owing to the significant uncertainties involved in uptake models and differences in uptake between plant species.

5.1.3 Risk estimates from Tier 2

In tier 2, the results from the ecotoxicity tests (Table 24) were combined with the results from refined modelling of uptake and exposure of the selected key organisms.

Table 24. Summary of the results from the ecotoxicity tests.

	% inhibition of germination		Soil invertebrate acute and reproduction toxicity				Tests on water and salt extracts			Leaching test ealuates, 1-stage	
sample	Rye grass	Lettuce	Ench. Acute, EC 50(%)	Ench. Reprod. EC50 (%)	Eisenia mortality % (test conc)	Eisenia reprod. EC50 (%)	Lumin. bacteria, EC50(%)	Lemna, Inh%	RET, EC50 (%)	Bacteria, EC50 (%)	RET, EC50 (%)
Natural 1	2	2	nt	23.0	67 (100)	EC<100	nt	2.6	6.45		>80
Natural 2	7	15	nt	22.0	nd	nd		0.0	1.45		nd
Natural 3	9	48	nd	nd	nd	nd		0.0	18		nd
Natural 4	0	70	nt	62.0	100 (100)	EC<100		11.1	>80		>80
Natural 5	0	10	nt	55.0	0 (100)	75<EC<100		0.0	>80		nd
Natural 6	1	0	nt	54.0	0 (!00)	EC<100		23.8	>80		nd
Natural7	5	6	nd	nd	nd	nd		20.9	>80		nd
CCA plant 1	9	3	nd	nd	nd	nd	>50	72.6	18.8		nd
CCA plant 2	14	18	nt	23.0	0 (100)	17	3.5	96.8	11.2		nd
CCA plant 3	2	29	nt	42.0	3 (100)	EC<50	8.8	100.0	6.48	nt	43
CCA plant 4	12	26	nd	nd	nd	nd	1.8	96.8	10.7		nd
CCA plant 5	4	13	nt	19.0	0 (50)	3.5	2.4	98.4	0.92	nt	11
CCA plant 6	4	56	nd	nd	nd	nd	0.9	99.2	8.13		nd
CCA plant 7	0	0	nt	36.0	0 (100)	<100	5.9	68.0	12.13	nt	>80
Mine 1	0	11	nt	40.0	42 (50)	0<EC<15		83.1	69.4		Nd
Mine 2	6	0	nt	29.0	nd	nd		94.4	24.9		14
Mine 3	0	2	nt	25.0	nd	nd		33.9	>80		>80
Mine 4	0	0	nd	nd	nd	nd		66,9	>80		Nd
Mine 5	2	3	nd	nd	nd	nd		49,2	>80		>80

nd, not determined
nt, not toxic

The ecotoxicity tests showed that soil samples from the CCA plant and Ylöjärvi mine site were phytotoxic. Fig. 5 illustrates the observed toxic response in *Lemna minor*.

Figure 5. The final stage of the growth inhibition test using *Lemna minor*, tests with soil samples from the CCA plant site and control soil (on the left). The test plants were grown one week in a vessel containing 50 g of soil sample and 100 ml nutrient solution. At the start of the test, there were 10 leaves in each vessel.

In addition to the soil samples, the eluates from the leaching test with the soil from the Ylöjärvi mine site and CCA plant site were tested with luminescent bacteria and RET. The former did not show any toxicity to eluates from the samples while some samples gave toxic response in the RET test. In the case of the Ylöjärvi mine site, the toxicity detected in one single sample was possibly related to the high concentration of sulphate (Parviainen *et al*. 2006). These results imply possible risks to surface waters and hence, to aquatic biota in the case of transport due to runoff. In fact, high concentrations of arsenic have been detected at least in the streams running from the mine site (see Section 5.2.).

The luminescent bacteria also showed toxicity to CCA plant samples (extracts). The reproduction of Enchytraeds was decreased in all sample types while in the case of earthworms, the soil from the CCA plant was clearly more toxic than the soils containing natural As. The effective concentrations of the soil samples from the Ylöjärvi mine site could not be determined owing to the fact that the soil was unsuitable as a habitat for earthworms. The results from the ecotoxicity tests are presented in more detail in a separate report (Schultz & Joutti, 2007).

To combine the results from the assessment based on chemical studies with those from the ecotoxicity tests, we first compared the calculated and measured concentrations in earthworms were with each other (Table 25).

Table 25. Comparison of the calculated and measured concentrations of arsenic in earthworms. Measured results refer to the mean value of parallel samples in which the concentration of As-containing test soil is 100 %.

Sample	Calculated, tier 1	Measured
Natural 1	1.8	0.98/1.4[a]
Natural 6	2.6	2.0/2.1[a]
CCA plant 3	137	370/335[a]

[a]depurated

The calculated concentrations (tier 1) are very near the concentrations measured in earthworms. This is surprising since in tier 1 we used a conservative model which is not recommended for general assessment. Additional samples would have made it possible to verify whether there is a true correlation between the calculated and measured concentration. Since the calculated concentrations are almost equivalent to the measured ones there is no need to elaborate the risk estimates of shrews (Table 23).

Tier 1 ERA showed that in addition to omnivores like shrew, herbivores may also experience some adverse effects. The tier 2 assessment shows low risks at forest areas and high risks at CCA plant site and Ylöjärvi mine site (Table 26) since the concentration in plants clearly exceed the reference values for the food consumption of small herbivores (0.881-1.008 mg/kg).

Table 26. Concentrations in plants (mg/kg-dw), percentiles from the Monte Carlo simulation (N = 2,500).

Percentile	CCA plant	Mine	Forest	Forest 2	Farm, forest	Farm, meadow
5%	1.8	3.5	0.06	0.26	0.20	0.19
10%	2.6	4.4	0.09	0.36	0.23	0.21
25%	4.1	6.3	0.19	0.56	0.29	0.25
50%	6.9	9.9	0.38	0.84	0.36	0.31
75%	11	15	0.84	1.2	0.45	0.37
95%	22	29	2.6	2.0	0.63	0.50
100%	81	93	24	4.9	1.3	1.0
point estimate	46	45	2.8	3.6	0.79	0.57

When the results from different chemical methods used in ERA are combined, the mine site appears to pose the highest ecological risks (indicated by the highest total scores) compared with other study sites/areas (Table 27). However, the difference between the toxicity scores of the CCA plant are minimal.

Table 27. Results from the ecological risk assessment based on chemical studies: average risk scores and scores representing the 5th and 95th fractile in the statistical distribution curve.. TP_{total} = toxic potency determined on the basis of acid leachable (total) concentrations in soil, TP_{NH4Ac} = toxic potency determined on the basis of concentrations lechable in ammonium acetate (NH4Ac) solution, HQ = Hazard Quotient

Sample	TP_{total}	TP_{NH4Ac}	$Score_{HQ}$ (shrew)	Total score	Total score 5% / 95%
Natural 1	0.70	0.05	0.70	0.56	0.53 / 0.59
Natural 2	0.83	0.07	0.77	0.65	0.64 / 0.69
Natural 3	0.86	0.05	0.97	0.83	0.82 / 0.85
Natural 4	0.76	0.09	0.31	0.47	0.44 / 0.49
Natural 5	0.92	0.09	0.05	0.59	0.57 / 0.61
Natural 6	0.48	0.04	0.65	0.44	0.42 / 0.47
Natural 7	0.65	0.05	0.20	0.36	0.33 / 0.39
CCA plant 1	0.99	0.67	0.03	0.88	0.86 / 0.89
CCA plant 2	0.99	0.61	0.49	0.87	0.85 / 0.88
CCA plant 3	0.98	0.57	0.72	0.87	0.86 / 0.89
CCA plant 4	1.00	0.81	1.00	0.99	0.99 / 0.99
CCA plant 5	1.00	0.98	1.00	1.00	1.00 / 1.00
CCA plant 6	1.00	0.98	1.00	1.00	1.00 / 1.00
CCA plant 7	0.79	0.19	1.00	0.90	0.89 / 0.91
Mine 1	1.00	0.97	0.88	0.98	0.98 / 0.99
Mine 2	1.00	0.95	0.26	0.96	0.95 / 0.96
Mine 3	0.99	0.81	0.97	0.97	0.96 / 0.97
Mine 4	1.00	0.96	1.00	1.00	1.00 / 1.00
Mine 5	1.00	0.93	1.00	1.00	1.00 / 1.00

The toxicity scores calculated on the basis of ecotoxicological studies (Table 28) differ from those determined by chemical studies. On the basis of ecotoxicity tests, soil in the CCA plant poses the highest risks while the scores of most of the mine site samples are rather low.

Table 28. Results from the ecological risk assessment based on ecotoxicological studies: test-specific average risk scores ($Score_{tox}$) and scores representing the 5th and 95th fractile in the statistical distribution curve.

Sample	$Score_{tox}$ Lemna	$Score_{tox}$ Rye-grass	$Score_{tox}$ Lettuce	$Score_{tox}$ Earth-worm	$Score_{tox}$ Enchytr.	$Score_{tox}$ Bacteria	$Score_{tox}$ RET	$Score_{tox}$ Total score	$Score_{tox}$ Total score 5% / 95%
Natural 1	0.03	0.02	0.02	na	0.77	na	0.94	0.58	0.55 / 0.58
Natural 2	0.00	0.07	0.15	na	0.78	na	0.99	0.70	0.67 / 0.73
Natural 3	0.00	0.09	0.48	na	na	na	0.82	0.46	0.41 / 0.48
Natural 4	0.11	0.00	0.70	na	0.38	na	0.20	0.33	0.30 / 0.42
Natural 5	0.00	0.00	0.10	0.39	0.45	na	0.20	0.21	0.15 / 0.23
Natural 6	0.24	0.01	0.00	na	0.46	na	0.20	0.20	0.13 / 0.26
Natural 7	0.21	0.05	0.06	na	na	na	0.20	0.13	0.070 / 0.23
CCA plant 1	0.73	0.09	0.03	na	na	0.50	0.81	0.53	0.50 / 0.56
CCA plant 2	0.97	0.14	0.18	0.99	0.77	0.97	0.89	0.89	0.84 / 0.93
CCA plant 3	1.00	0.02	0.29	na	0.58	0.91	0.94	0.89	0.87 / 0.90
CCA plant 4	0.97	0.12	0.26	na	na	0.98	0.89	0.87	0.79 / 0.90
CCA plant 5	0.98	0.04	0.13	1.00	0.81	0.98	0.99	0.95	0.92 / 0.96
CCA plant 6	0.99	0.04	0.56	na	na	0.99	0.92	0.88	0.85 / 0.94
CCA plant 7	0.68	0.00	0.00	na	0.64	0.94	0.88	0.69	0.67 / 73
Mine 1	0.83	0.00	0.10	1.00	0.60	na	0.31	0.81	0.76 / 0.82
Mine 2	0.94	0.06	0.00	na	0.71	na	0.75	0.67	0.51 / 0.76
Mine 3	0.34	0.00	0.02	na	0.75	Na	0.20	0.33	0.26 / 0.44
Mine 4	0.67	0.00	0.00	na	na	Na	0.20	0.28	0.21 / 0.34
Mine 5	0.49	0.02	0.03	na	na	Na	0.20	0.21	0.14 / 0.30

It has to be noted that the results from the ecotoxicity tests were inadequate in some extent that is, not all effects were measured or they could not be measured owing to the difficulties in the realization of the tests (for further information see Schultz & Joutti, 2007). This may cause some bias in the final results. It is also noteworthy that the deviation calculated for the ecotoxicity score underestimates the true diversity of the toxicity responses since some of the results (bacteria, RET, Echytraeids) are based on individual samples only, i.e., no concurrent samples were tested.

When we combine the scores from different samples representing the same site/area we can get a better idea of the potential population-scale ecological effects (Table 29).

Table 29. Average total scores from the different lines of evidence (chemistry, ecotoxicology) for ecological risks and their statistical variation expressed as the 5th and 95th percentile in the distribution curves defined by Monte Carlo analysis. Scores determined as the mean value of the single samples representing different types of site/area studied.

	Chemistry			Ecotoxicology		
Area/site	**Mean**	**5%**	**95%**	**Mean**	**5%**	**95%**
Natural As	0.56	0.54	0.58	0.36	0.32	0.42
CCA plant	0.93	0.92	0.94	0.82	0.78	0.85
Mine site	0.98	0.98	0.98	0.46	0.38	0.53

On the basis of chemical studies, all study sites/areas pose moderate or high risks (score > 0.75) to biota whereas the ecotoxicological studies show high risk only in the case of the CCA plant and low risks (< 0.5) in the case of the mine site and areas with high natural As in till. Generally, ecotoxicological studies give more reliable information on the actual risks since they consider several factors which are ignored in the assessment based on chemical studies. Such factors include particularly bioavailability, excretion and metabolia, adaptation and joint toxic actions (owing to simultaneous contaminants). The low scores of TP values based on ammonium acetate extraction support the assumption of low bioavailability and hence, low toxicity of soils with high levels of natural As. However, in the case of the mine site and CCA plant the potential availability seems to be rather high. Moreover, elevated concentrations (up to some 400 mg/kg) of As were detected in the earthworms grown in the CCA plant soil. This indicates that a significant part of the As in soil can be bioavailable.

It has to be noted that the TP –values cover all relevant contaminants whereas the HQ values for shrew only consider arsenic. This causes some sample-specific differences between the TP_{total} and $Score_{HQ}$ –values, particularly in the case of soil with naturally high concentration of arsenic. The contribution of other contaminants is discussed in Section 5.1.4. In addition, differences arise from the different fcalculation methods.

5.1.4 Risk characterization

Although our study on the risks was focused on arsenic, it was clear that the possible toxic effects originating from other contaminants present in our environmental samples could not be ignored. In the ecotoxicity tests the effects of such simultaneous contaminants are automatically present while in the case of modelling-based ERA, other contaminants have to be studied separately. In our case, the most relevant simultaneous contaminants included chromium and copper in the CCA plant. In the areas with high levels of natural As, various other elements may be present. The possible contribution of chromium in the realization of ecological risks at our study sites/areas determined on the basis of chemical studies is presented in Table 30.

Table 30. Contribution of the major contaminants As and Cr to the total risk estimate calculated on the basis of ecological benchmarks protective of different species: the variation of the ratio (%) between the contaminant-specific Hazard Quotient (HQ) and the Hazard Index (HI, sum of contaminant-specific HQs) in samples representing different study sites/areas. Samples refer to samples studied in the ecotoxicity tests.

	Natural As		**CCA plant**		**Mine site**	
Receptor	**As**	**Cr**	**As**	**Cr**	**As**	**Cr**
Microbes	0 – 1[a]	2 – 3[a]	1 – 14	10 – 68	1 – 4	0 – 1
Earthworm	0 – 1	98 – 99	1 – 2	97 – 99	17 – 34	63 – 68
Plants	0 – 2[b]	7 – 13[b]	3 – 16	30 – 79	9 – 21[b]	2 – 3[b]
Plants	15 – 85	1 – 5	82 – 94	1 – 3	97 – 98	0
Terr. biota	0 – 7	91 – 98	3 – 10	89 – 96	56 – 75	23 – 43
Soil biota	4 – 53	10 - 19	45 – 70	9 – 20	90 – 94	0 – 1
Soil biota	2 – 33	39 – 55	23 – 49	42 – 63	88 – 94	3 – 7
Mouse	26 – 90	8 – 51	81 – 93	6 – 16	99	0 – 1
Shrew	36 – 93	4 – 39	88 – 95	3 – 9	99	0

[a] Except for two CCA plant samples, the prevailing contaminant is Fe with the contribution of HQ ranging from 44 % to 86 % of HI

[b] The prevailing contaminant is Al with the contribution of HQ ranging from 76 % to 92 % of HI

The results (Table 30) show that the possible toxic response is not associated with arsenic alone and that the contribution of other contaminants, particularly chromium is significant in some cases. The relative contribution of a single contaminant depends on the receptor as indicated by the variation of the relative HQs. Generally, arsenic seems to be the major toxicant to organisms in higher trophic levels e.g., small mammals, whereas soil invertebrates and microbes seem to be more insensitive to As compared with other contaminants. In the case of plants, the contribution of arsenic in the overall toxicity is unclear since the benchmarks used in the determination of risk estimates vary considerably.

Our preliminary findings of the statistical analysis on the ecotoxicity tests' results using the elaboration model verify the fact that the toxic responses detected in the test organisms are not solely associated with the elevated arsenic concentration but also with other concurrent contaminants, particularly copper in the CCA -soils. The results are studied further and the final outcomes of the statistical analyses are documented elsewhere.

The results from the batch leaching test (L/S = 2 and L/S = 10) can be used as rough estimates of the mobility and potential availability of arsenic. Leachability of arsenic from the soil at both the CCA-plant and mine site was low, i.e., 2.0 % and 0.5 % (L/S = 10) at the maximum. This suggests that the leachability and hence, the mobility is also low. However, due to the extremely high concentrations in soil, the amount of contaminants in leachates may be significant. If the environmental conditions, pH being one of the key factors, remain the same, arsenic is expected to stay mostly bound in the soil particles. However, radical changes in the physico-chemical characteristics of soil might chance this situation.

Contrary to the batch leaching test, ammoniumacetate-EDTA dissolved less arsenic from the CCA-soils (6 % from the total As concentration) than from the mine tailings (25 % from the total As concentration). The ammoniumacetate-EDTA extraction has often been considered as a method to assess the bioavailability of contaminants. However, bioavailability to both animals and plants can hardly been described by one and the same extraction method. In fact, the preliminary results from

the statistical analysis indicate that this extraction method is a poor predictor of bioavailability in the case of soil animals.

The fact that some soil samples representing the areas with naturally occuring arsenic were taken from the deeper layers of soil causes some bias in the results particularly in the case of organisms foraging only in upper soil layers or on the soil surface. Such organisms include shrews, the exposure of which was estimated on the basis of a generic exposure model. In this model, soil ingestion related to topsoil is also considered. The calculations showed that the contribution of soil ingestion in the total exposure was around 90 % at the minimum. In fact, this was expected since arsenic does not accumulate in earthworms (i.e. the concentration in earthworms is lower than in soil) which were assumed to be the main food source and since soil ingestion was estimated to be 13 % of the food consumption (Sample *et al*. 1997a). Hence, depending on the actual concentrations of arsenic in topsoil, the use of concentrations detected in deeper soil layer may either over- or underestimate the potential exposure of shrews to arsenic.

Taking into account the surface area of the mine sites, ecological risks are expected to be the most significant in the environment affected by Ylöjärvi mine site. Hence, the factual spatial dimensions of the risks, i.e., the size of the impact area is unknown since it was not studied. The ecological risks at former CCA plants Ruovesi 1 being one of these, can be expected to be rather small in population level due to the limited surface areas of the sites. Since even the soils with elevated concentrations of naturally occurring arsenic showed low to moderate risks to biota, effects to the most sensitive species along with some natural selection towards less sensitive species has probably occurred at areas with very high natural arsenic.

5.2 Risks to aquatic biota

The monitoring data from the Ylöjärvi mine site indicates releases of anthropogenic arsenic to aquatic ecosystems since the concentrations are clearly higher compared with the concentration level in non-effected areas. According to the monitoring data starting from 1975 of several water systems within Pirkanmaa region, in the areas with no anthropogenic sources, the concentration of arsenic in water phase lies generally below 1 µg/l and there are only single cases in which the level of 5 µg/l has been exceeded. Generally, these exceedances are related to the old data from the 1970s' when the accuracy of the methods of chemical analysis were not as good as at present. On the average, the level of 1 µg/l has been exceeded in ca. 30 % of the samplings while at the same time in some parts of the Vahantajoki river basin connected with the Ylöjärvi mine site, this level is exceed in nearly all samples taken. Furthermore, on the average some 90 % of the samples in the Ylöjärvi water system have exceeded the level of 5 µg/l.

The risk estimates (HQs) show that arsenic may pose at least a moderate risk to the aquatic biota in Lake Parosjärvi (Table 31). In the case of other water systems, risks are expected to remain insignificant. However, since there were no data available on the biota in the streams nor in the lake Parosjärvi, it is not possible to draw any definite conclusions on the risks to aquatic ecosystems in these water systems. Due to the small size, it is presumable that the ditch running outwards from the tailings area does not permanently inhabit fish or any other species in higher trophic levels.

Table 31. Risk estimates (HQs) for surface water calculated on the basis of the mean concentration and median concentration (HQ_1*) of As detected in the water system at different sampling points in different times. The HQ_1…HQ_4 refer to the use of different toxicity-based ecological benchmarks (BM_1…BM_4) presented in the literature. Samples taken from all depths have been combined and the final HQs rounded to the maximum of two significant numbers.

Sampling point	HQ_1	HQ_1*	HQ_2	HQ_3	HQ_4
Ditch from tailings to the Lake Parosjärvi	83	81	8.4	5.4	1.4
Lake Parosjärvi	30	32	3.0	1.9	0.5
Stream Parosjärven oja	16	10	1.7	1.1	0.3
Stream Vähä-Vahantajärven oja	19	4.5	1.9	1.2	0.3
Stream Vahantajoki downstream	5.2	2.0	0.5	0.3	0.08
Lake Näsijärvi	2.3	2.7	0.2	0.2	0.04

BM_1 = 3.1 µg/l, BM_2 = 31 µg/l, BM_3 = 48 µg/l, BM_4 = 190 µg/l

The magnitude of ecological risks is clearly reduced when distance from the mine site increases, i.e., along the water route: ditch – Lake Parosjärvi – streams – Lake Näsijärvi. The results from the sediment studies support this observation. The calculated HQs for sediments vary between 0.8 and 410 depending on the sampling site (and, i.e, on the distance from the mine site) and sampling depth (Table 32)

Table 32. Estimates for the sediment-associated risks in the area of Ylöjärvi mine site.

Sampling point	HQ_1		HQ_2	
	Max	min	max	min
Wetland, 0 – 0.5 m	410	240	200	120
Wetland, 0.51 - 1.71 m	150	100	72	51
Wetland, 1.07 - 1.78 m	340	160	170	80
Wetland, 1.8 - 1.83 m	16	16	8.1	8.1
Lake Vähä-Vahantajärvi, < 0.1 m	53	18	27	9.1
Lake Vähä-Vahantajärvi, 0.1 - 0.3 m	22	4.9	11	2.5
Lake Näsijärvi, 0.05 m	3.4	3.4	1.7	1.7
Lake Näsijärvi, 0.2 m	21	21	11	11
Lake Näsijärvi, 0.3 m	1.5	1.5	0.8	0.8
Stream Parosjärven oja	21	2.3	10	1.1

HQ_1/ HQ_2 = calculation based on the ecological benchmark of 11 mg/kg / 22 mg/kg

In the survey on the stream sediments in Finland the average concentration has been 9.58 mgAs/kg with a median value of 6.0 mgAs/kg. Hence, in the sediments of Ylöjärvi area, the concentrations are clearly elevated.

In addition to As, the studies on the sediments show increased concentrations of Ag, Mo, Cu and Se. In the sediment core taken from lake Vähä-Vahantajärvi, the concentrations of As and these metals were the highest at 8 cm and diatom species compositions changed correspondingly. However, indications of concomitant increases in nutrient inputs were also present as diatom-inferred concentrations of total phosphorus (DI-TP) increase in lake water. This increase is not mine-related but presumably caused by nutrient load generated by agriculture since the DI-TP curve follows the content of fine-grained mineral matter in sediment. The ecological effects of As and other mining inputs were studied with the multivariate redundancy analysis (RDA) method. In RDA, no statistically significant effect of As on diatom species composition was found. This was

true for mineral matter content as well, which was used as a surrogate variable for agricultural nutrient inputs to the lake. Thus, statistically significant effects of As on the diatom algal population in Lake Vähä-Vahantajärvi could not be detected despite the changes in diatoms in the mine water-impacted sediment layers.

The monitoring data concerning the surface waters in the watercourse around Ylöjärvi mine site show a temporal tendency of slightly increased concentration of arsenic in the streams leading outwards from the mine site (Fig. 6.). The same tendency can be seen in the Lake Parosjärvi (Fig. 7).

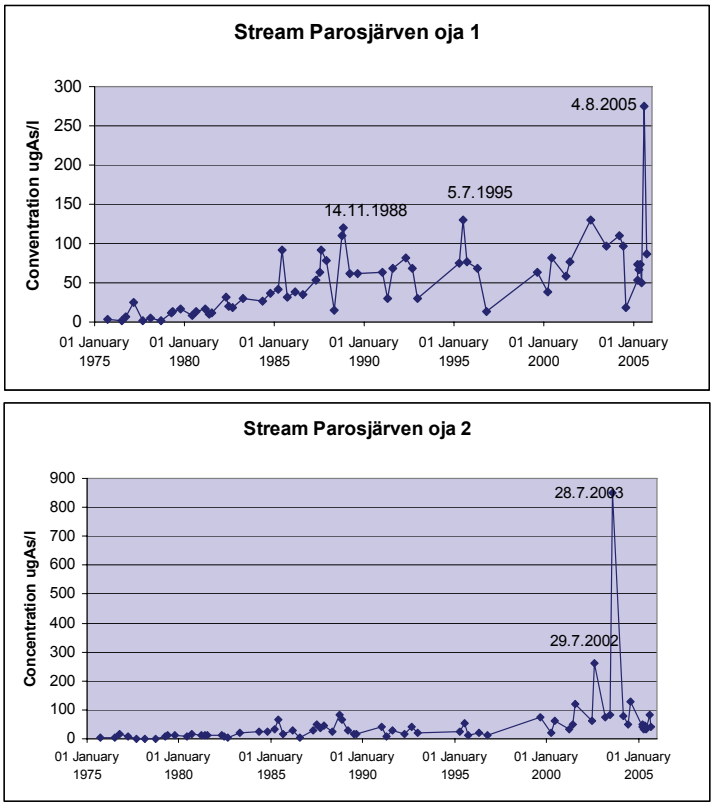

Figure 6. The concentration of As in the stream waters flowing from the Ylöjärvi mine site as a function of time.

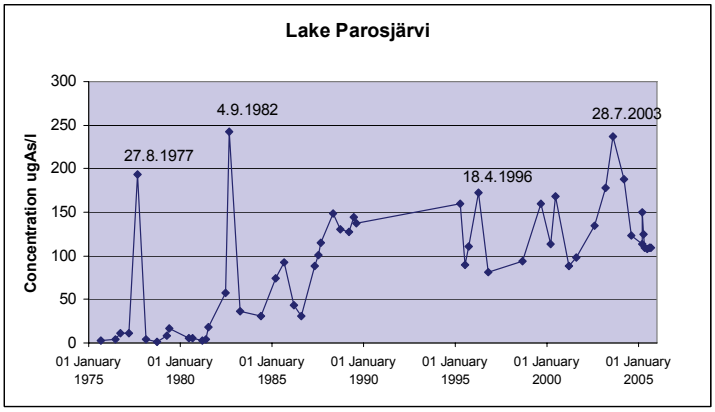

Figure 7. The concentration of As in the Lake Parosjärvi receiving the streams flowing outwards from the Ylöjärvi mine site as a function of time.

The interpretation of the figures 6 and 7 are hampered by the fact, that methods to analyze arsenic have changed. That is, the accuracy and sensitivity of previous methods was lower compared with the present situation causing inaccuracy particularly in the results from the 1970s'-1980s' versus those from the start of 1990s'.

As can be concluded from figures 6 and 7, the release of arsenic from the mine area is not diminishing. If anything, in case no risk management actions will be carried out, the emissions are expected to continue at present scale at least for several decades since the tailings form a substantial As reserve. Consequently, the ecological risks will stay and their magnitude may even increase due to the potential increase in the concentration level in aqueous phase and accumulation of arsenic in the sediment.

The benchmarks for water used in the screening-level assessment are based on toxicity tests conducted in the laboratory. Therefore, they should be compared to water concentrations that are as equivalent as possible to concentrations in test water which is nearly all dissolved. On the other hand it is common to use acid soluble concentrations rather than dissolved concentrations in the risk assessment. Also in RAMAS, the measurements of arsenic in water were based on filtrated, acid (HNO_3) soluble fraction (Bilaletdin *et al.* 2007). However, acid soluble concentrations of metals typically include 30 to 95% particle bound material (HECD, 1992) and consequently, use of them leads to conservative risk estimates. Dissolved concentrations give a more realistic estimate of risks.

Since no data was available on the biodiversity and abundance of aquatic biota, it was not possible to assess the possible population-level, community-level, or ecosystem-level ecological risks in Lake Parosjärvi. It is also noteworthy that other concomitant contaminants are also present and these might add to ecological risks. The contribution of these in the aquatic environment was not studied. Hence, additional site studies are necessary to verify the overall risks to aquatic biota.

6 RESULTS - HEALTH RISKS

6.1 Phase 1: HRA based on preliminary data

In Phase 1 health risk assessment focus was on the As exposure from household water. In addition, we considered the contribution of food intake and exposure to contaminated media at a former CCA wood treatment site. The input values used as a basis for the calculations have been presented in Section 4.4.4.

6.1.1 Intake estimates of natural and anthropogenic arsenic

The adjustment of different distributions to the concentration data showed that the statistical distribution of As concentration in drilled well waters was best characterized by lognormal distribution, even though the fit was not very close (Fig. 8). Concentration data comprised more low-end figures and some very high concentrations than the lognormal model. Also the effect of detection limits (5 and 10 µg/l) could be seen in the frequency data that is part of the concentration data was given as < 5 or < 10 µg/l.

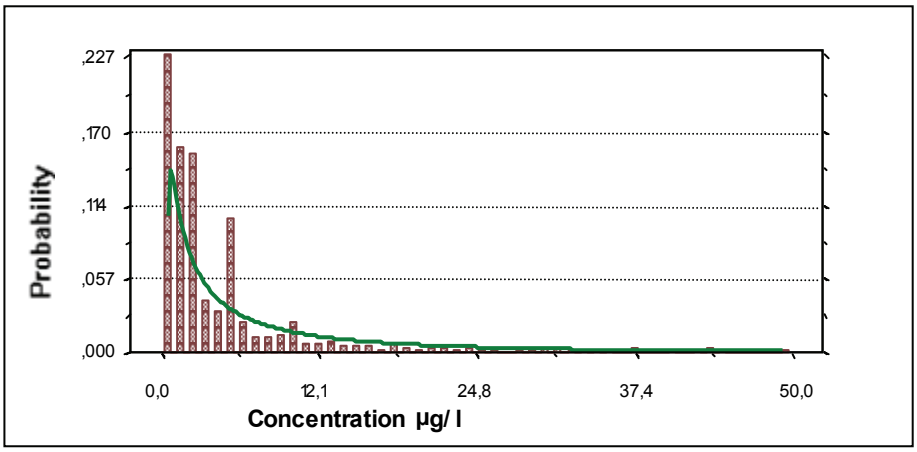

Figure 8. Frequency data illustrating the As concentration in drilled well waters and the lognormal distribution fitted by using the statistical Crystal Ball® software.

Arsenic content in the dug well waters was typically below 1 µg/L and hence, apparently does not pose any significant risk for people consuming the water. On the other hand, the maximum concentrations exceeded the guideline value of 10 µg/L. Statistical distribution of the concentration data was close to lognormal.

On the basis of the statistical distribution of total water intake, the average water intake was estimated to be 1.5 l/d (median 1.4 l/d) with the overall range 0.33-4.0 l/d (Fig. 9).

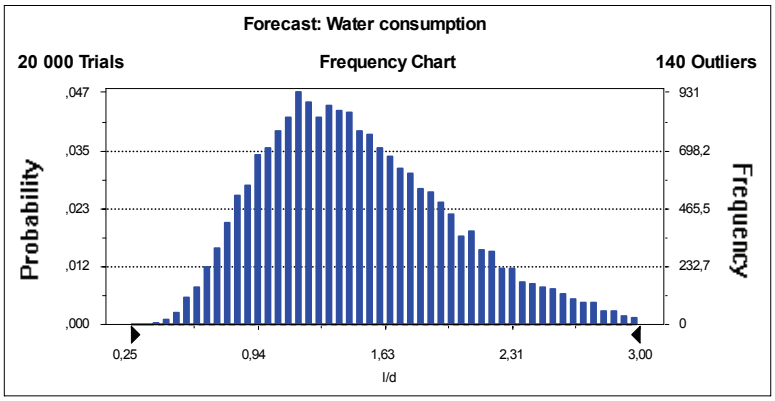

Figure 9. Frequency distribution of total water intake.

Statistical distribution of inorganic arsenic intake from drilled well water at home was calculated by aggregating the concentration data (Fig. 8) and water consumption data (Fig. 9). The average intake from drilled well water was estimated to be 0.56 (range 0.00-57 µg/kg/d, Fig. 10).

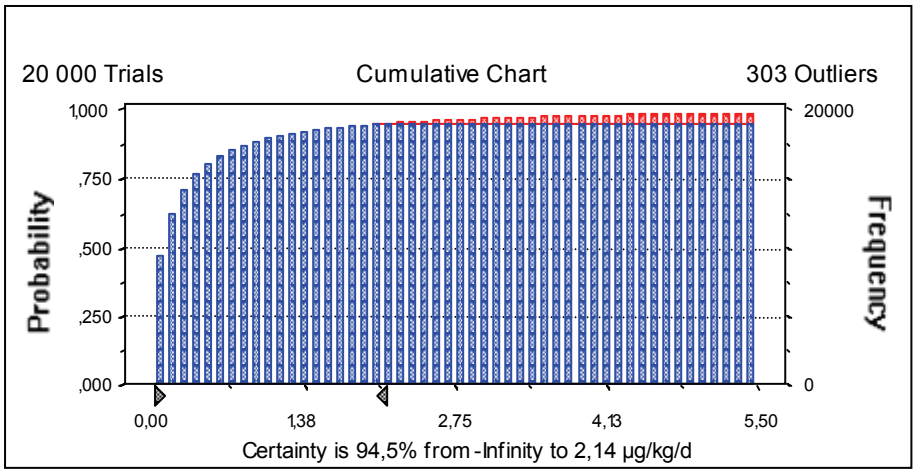

Figure 10. Cumulative frequency distribution of the estimated arsenic intake from drilled well water in Pirkanmaa.

As could be concluded from the plain concentration data, differences between the As intake estimates in geological units were considerable. In the CFGC geological unit As intake from drilled well waters was estimated to be 0.00 – 0.37 µg/kg/d. In the Tampere Belt the corresponding range was 0.00 – 72 µg/kg/d, and in the Pirkanmaa Belt 0.00 – 44 µg/kg/d.

Concentrations of arsenic in dug well waters have been relatively low. Consequently, the average As intake from dug well water was estimated to be only some 0.03 µg/kg/d. The use of the maximum concentration and the average value for water consumption resulted in an intake estimate of 1.1 µg/kg/d, respectively. The stochastic maximum was 1.4 µg/kg/d. In the different geological units, the stochastic estimates of As intake were (µg/kg/d): CFGC 0.00 – 0.94, TB 0.00 – 0.10 and PB 0.00 – 1.46. The concentration data from TB unit were limited (n = 19) and it is possible that some high As concentrations have remained undetected.

Wood treatment plant areas, old mine areas and handling of CCA treated wood are well known sources of anthropogenic arsenic intake. Assuming the average of 1.5 l/d water use, the calculated As intake varied from 17 to 86 µg/d (0.24 to 1.22 µg/kg/d). Direct exposure (soil ingestion, skin contact) to the contaminated soil (82 mg/kg) would increase the intake about 1.2 µg/d (0.02 µg/kg/d). With the inclusion of food intake total intake estimates vary between 0.55 and 1.53 µg/kg/d.

6.1.2 Risk characterization

The above-presented As intake estimates (water + background + soil) were compared to acceptable daily doses issued by different organizations. A calculated hazard quotient (HQ) below 1.0 means that the given exposure will likely not result in adverse non-cancer health effects. However, a HQ greater than 1.0 does not necessarily suggest a high probability of adverse effects. A HQ greater than 1.0 indicates that adverse health effects may exists. In fact, since HQ does not include the probability factor it is not a clear indicator of risks.

Judging from the PTWI value issued by WHO, only those people using drilled well water of high As level in Pirkanmaa might be harmfully exposed to arsenic. When we assume that the statistical distributions used in the evaluation of As intake describe the population in Pirkanmaa and that there is no dependence between water use and As concentration, we arrive at an estimate that the fraction of drilled well water users exposed to a harmful As levels is about 6 % (Fig. 11). If we use the TDI issued by RIVM, we end up to an estimate of some 17 %, respectively. In addition, some 0.1 % of the dug well water users might be exposed to intolerable As doses. The RfD presented by USEPA represents the lowest acceptable daily dose which according to our calculations is exceeded in all exposure scenarios. This was ecpected, since background exposure was estimated to be on this level.

According to the risk calculations, health risks associated with arsenic within the CFGC geological unit are expected to be insignificant. Only solitary dug well water users might experience doses exceeding the ADD value issued by RIVM. In the Tampere Belt area the health risks seem inevitable, unless rigorous risk management actions are implemented among drilled well water users. Judging from the WHO's ADD value, 12 % of the drilled well water users would be exposed to too high arsenic doses. The use of the ADD of RIVM results in an estimate of 27 %, respectively. In the Pirkanmaa Belt 3 % of the drilled well water users would be exposed to As quantities exceeding the WHO's ADD value and 9 % the respective RIVM's value. In the case of dug wells, estimated exposure was below the former value in all scenarios while only 0.03 % exceeded the latter value.

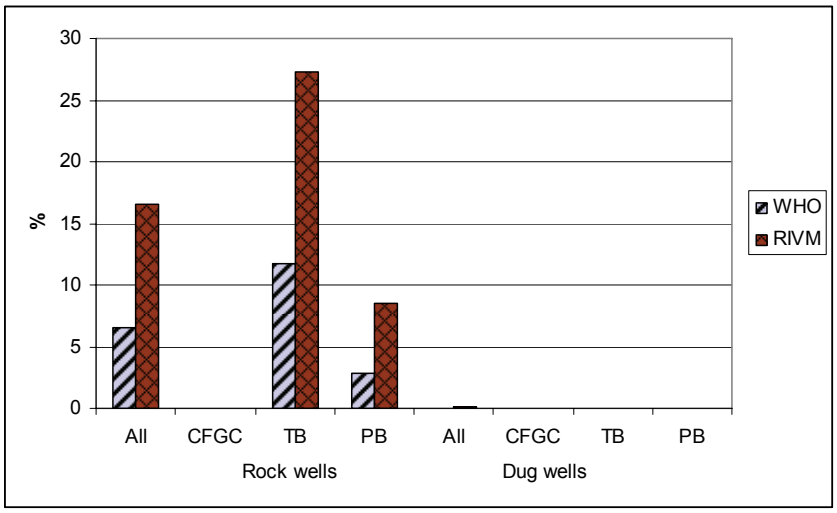

Figure 11. Estimated total intake of inorganic arsenic vs. the acceptable daily doses (ADDs) issued by WHO and RIVM: probability of exceeding the ADDs among the drilled (rock) and dug well water users.

Total intake of inorganic arsenic of people using groundwater at a CCA plant area was estimated to be 0.55 – 1.53 µg/kg/d. The maximum total intake estimate was below the ADD issued by WHO but exceeded the corresponding values issued by RIVM and USEPA. Therefore, health risks do not seem to be high. However, the safety marginal is small or lacking.

Cancer risk was estimated to be unacceptably high in every scenario. It must be taken into account that cancer risk calculation is based on the assumption of a lifetime (70 a) continuous exposure which in practice, is relatively seldom fulfilled. In addition, the cancer risk slope factor includes uncertainties and cancer risk is probably overestimated when using the linear model assumed by USEPA. In fact, USEPA is currently revising the cancer risk assessment for inorganic arsenic.

The risk calculations were made for adults. The actual doses might be higher for children meaning also higher risk. On the other hand, well water investigations have been concentrated to the locations where high arsenic concentrations have previously been detected. In any case, risk calculations evidently show that at the areas with the highest natural As concentrations, the drilled well waters are unsuitable for human consumption.

6.2 Phase 2: HRA based on aggregate data

In the phase 2, new data collected during the RAMAS project and a more comprehensive data about background exposure were included in the health risk assessment. The complemented concentration data covered both areas with naturally occurring arsenic and anthropogenic arsenic (see Section 4.1.1). The input values used in the derivation of risk estimates are descibed in Section 4.4.4.

6.2.1 Risks originating from natural arsenic

Arsenic intake from other routes but water. The intake of inorganic arsenic from food was calculated using the latest information on the As content in different food items and fraction of inorganic arsenic (Table 18, Section 4.4.4). Main part of the food mediated inorganic arsenic intake was from cereal products followed by root crops. The calculated total inorganic arsenic intake estimate was 11 µg/d, being close to the lower end of the National Public Health Institute's estimate of the total dietary intake in Finland.

In addition to arsenic intake from water and food, intake via inadvertent soil ingestion and dermal contact with soil were included in the estimate of the total intake. As concentrations in the surface soil of the farms studied in RAMAS were low and hence, potential soil intake would not contribute significantly to the estimate of the average total daily dose. With the inclusion of soil ingestion, we ended up in an estimate of 11.1 µg/d which is equivalent to 0.16 µg/kg/d when using the average body weight of 70 kg.

Total inorganic arsenic intake estimates. The new data collected in RAMAS did not have a significant effect on the distribution of arsenic concentration in well waters. Lognormal distribution was used in intake calculations as in the case of the preliminary data. Statistical distribution of the total intake of inorganic arsenic for drilled well water users is presented in Fig. 12. Intake from water was estimated using Monte Carlo simulation while point estimates were used in the case of other sources. For the whole Pirkanmaa region the calculations resulted in 0.16-55 µg/kg/d of total intake of inorganic As with an average of 0.68 µg/kg/d (median 0.27) in the case of drilled well water users. For dug well water users, the corresponding dose estimates varied between 0.16 and 1.6 µg/kg/d with an average value of 0.68 µg/kg/d (median 0.16).

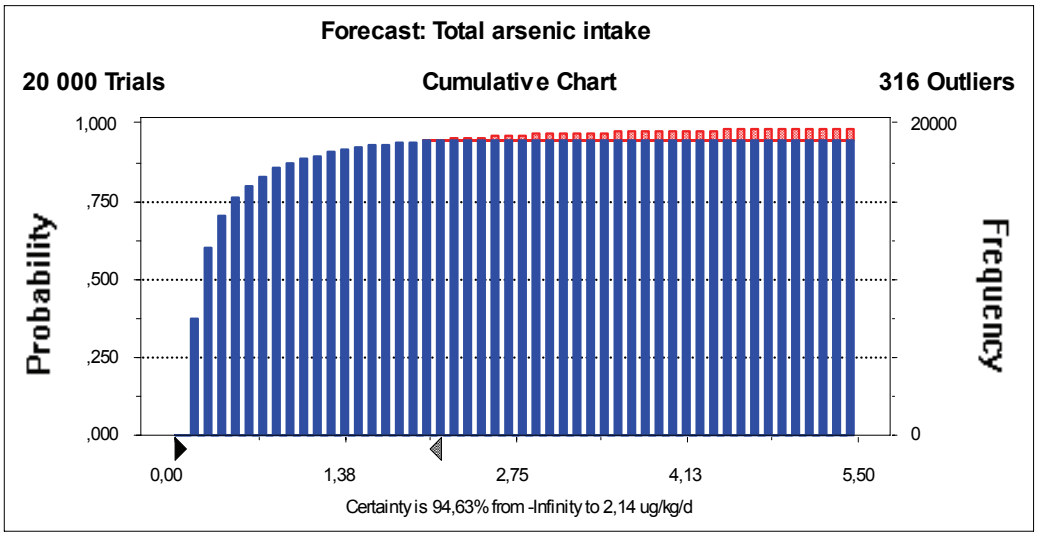

Figure 12. Cumulative frequency distribution of the estimated total intake of inorganic arsenic, drilled well water users.

It can be clearly seen that only a minor fraction of the whole population within the study area might be harmfully exposed to arsenic from natural sources. However, the situation is spatially variable. In the CFGC geological unit, arsenic intake from drilled well waters was estimated to be 0.16 – 0.53 µg/kg/d while in the Tampere Belt, the estimated range is 0.16 – 49 µg/kg/d (average 1.0, median 0.4 µg/kg/d), and in the Pirkanmaa Belt 0.24 – 47 µg/kg/d (average 0.48, median 0.2 µg/kg/d). Within the Pirkanmaa Belt the estimated exposure to arsenic was generally about half of that of the

Tampere Belt but a few high well water concentrations raised the maximum intake estimates nearly to the same level.

As was revealed in the Phase 1 risk assessment, arsenic intake among the dug well water users was generally negligible, but some high intake values exist within the Central Finland Granitoid Complex (CFGC) and Pirkanmaa Belt. Within the separate geological units, the estimated arsenic intakes were (µg/kg/d): CFGC 0.16 – 0.89 (average 0.18, median 0.17), TB 0.16 – 0.25 (average 0.17, median 0.16) and PB 0.16 – 1.5 (average 0.17, median 0.16). Data from the TB unit were limited (n = 19) and and it is possible that some high As concentrations have remained undetected.

6.2.2 Risks originating from anthropogenic arsenic

Exposure to arsenic at contaminated chromated copper arsenate (CCA) wood treatment plant sites results mainly from direct contact with contaminated soil or using groundwater contaminated by arsenic. There was no production of food plants at these sites and the dietary As intake associated with arsenic in the sites only consisted of intake from water. In the assessment of exposure through soil ingestion, we used the average As concentration in soil because the sites were relatively small and exposure was assumed to be evenly distributed over the areas.

Children are typically more liable to soil contaminants compared with adults since they often play on the ground, and put their hands in their mouth and sometimes intentionally eat soil and dirt. Children are often assumed to eat some 100 mg/d of soil. Using a body weight 15 kg makes a dose of 0.1-1.7 µg/kg/d when concentrations in soil is 82 – 1474 mg/kg. In addition, ingestion of contaminated food or juice made with As-contaminated water may represent a more significant source of exposure for children than for adults. It can be readily seen that in case of high As concentrations in surface soil, direct contact with soil may considerably add to the total inorganic arsenic intake of young children.

For adults, intake of inorganic arsenic from groundwater at Vilppula CCA plant area was estimated to be 0.24 to 1.22 µg/kg/d the total dose being about 0.55 – 1.53 µg/kg/d. Arsenic concentration in soil was low and would not considerably add to the total intake. The maximum dose estimate was below the ADD issued by WHO but exceeded the respective values of RIVM and USEPA. Therefore, health risks were not considered to be high. However, the safety marginal is small or lacking.

During the RAMAS project the soil, leachability of arsenic from soil and groundwater at a former CCA treatment plant Ruovesi I were studied. On the basis of the As concentrations in the solid and liquid phases, we can derive partition coefficients (K_d) ranging from 250 to 880 l/kg. K_d -values were smaller with the liquid-solid –ratio (L/S) of 2 than with the ratio of 10 indicating diminishing amount of the leachable arsenic with time. Therefore, the average K_d value 320 l/kg representing the test in L/S = 2 was used to estimate leaching of arsenic from soil to groundwater. The CCA plant studied (Ruovesi, closed in 1960) is located in a 1st class groundwater area[5]. The distance to the nearest waterworks with groundwater abstraction is 145 meters. Water intake has been about 250 m^3/d but the permission allows 400 m^3/d. As concentration in the water produced in this waterworks was 0.3 µg/l in march 2007. The nearest lake is situated 169 meters from the site. A small stream

[5] According to the definition an aquifer belongs to the class I if its water is used by waterworks which provides or will within 20-30 years provide water for more than 10 households or the area is needed to provide domestic water in the case of crisis

runs through the study area and the wood preservation plant is situated on the northern side of the stream. The CCA plant is situated a few meters above the water level of the stream, and to the northeast of the contaminated site is a cliff. The contaminated land area was estimated to be some 1,000 m^2. Using the maximum As concentration detected for the entire contaminated area results in a potential of 13 µg/l increase in the As concentration in waterworks. Because the actual concentration in the soil is lower than the maximum value and since not all of the leached arsenic is expected to migrate to water intake well, it is obvious that CCA plant area may have a substantial effect on the quality of the water in the waterworks but could not raise As concentration above the limit value of 10 µg/l.

Additional As intake may result from inhalation of contaminated air. Concentration of arsenic in the ambient air was measured in the Ylöjärvi mine site where wide open tailing areas are susceptible to wind erosion. Moreover, tailings area is used for ammunition explosions which often cause high dust concentrations. Estimates of a lifetime excess cancer risk associated with airborne arsenic were calculated using the unit risks issued by WHO and USEPA (Table 33). Cancer risk was estimated to be relatively small owing to the fact that the area is only in occupational use and exposure occures only during the working hours.

Table 33. Calculation of cancer risk from airborne arsenic at the Ylojärvi mine area using the measured average arsenic concentration in the air and two different unit risk estimates (WHO and USEPA).

Parameter	**Method**	
	WHO 2000	*USEPA 1988*
As concentration ng/m^3	26.90	26.90
Exposure frequency d/a	182	182
Exposure duration h/d	8	8
Absorption fraction	0.50	0.50
Unit risk estimate 1/(ng/m^3)	$1.5*10^{-6}$	$4.3*10^{-6}$
Risk	$3.4*10^{-6}$	$9.6*10^{-6}$

Arsenic intake from breathing the contaminated air was estimated to be around 0.04 µg/kg/d, which is insignificant compared to the safe intake values. The army institute is served by a public water supply, and concentration of arsenic in drinking water is low. Therefore, risk from soil arsenic to people working in the area is insignificant.

6.2.3 Risk characterization

The estimates of As intake were compared to the acceptable daily doses issued by different organizations (Table 34). Because new data about natural arsenic concentrations in crops and soils within the study area did not differ from the nationwide data, estimates of dietary intake in Phase 2 HRA did not differ considerably from the estimates produced in Phase 1. The well water data, too, remained roughly the same, and the total intake estimates based on the aggregate data (Phase 2 HRA) differed only slightly from the preliminary estimates (Phase 1 HRA). Using the PTWI value issued by WHO and assuming that the distributions used in the stochastic assessment represent the population in Pirkanmaa, we can estimate that less than 6 % of drilled well water users can be harmfully exposed to arsenic (Fig. 13). The TDI issued by RIVM results in an estimate of some 14 % in the case of drilled well users, respectively. In addition, some dug well water users might be exposed to intolerably high arsenic doses. The RfD issued by USEPA represents the lowest acceptable daily dose and consequently, on the basis of this value, almost 50 % of drilled well water

users would be exposed to too high levels of arsenic. In the case of dug wells, the unacceptably exposed people were estimated to cover 1.8 % of all dug well water users.

Table 34. Calculated intake estimates ([s] stochastic estimates, [p] point estimate) compared to the acceptable daily doses (µg/kg/d). HQ= Hazard Quotient. PTWI = Provisional Tolerable Weekly Intake, TDI = Tolerable Daily Intake, RfD = Reference Dose.

Exposure scenario	Calculated dose µg/kg/d	WHO (1993)		RIVM (Baars *et al.* 2001)		EPA (2006)	
		PTWI/ 7	HQ	TDI	HQ	RfD	HQ
People using drilled well water+ background, maximum	55[s]	2.14	26	1.0	55	0.3	180
People using drilled well water+ background, 95 % fractile	2.28[s]	2.14	1.1	1.0	2.3	0.3	7.6
People using dug well water+ background, maximum	1.6[s]	2.14	0.75	1.0	1.6	0.3	5.3
People using dug well water+ background, 95 % fractile	0.22[s]	2.14	0.10	1.0	0.22	0.3	0.73
People using groundwater at CCA plant area+ background	0.53–1.5[p]	2.14	0.25 -0.7	1.0	0.53 – .5	0.3	1.8-5.0

According to the risk calculations, there should not be a general health risk problem from arsenic in the CFGC geological unit. Only solitary dug well water users might be exposed to doses exceeding the acceptable daily doses issued by RIVM and USEPA. In the Tampere Belt area the health risks seem inevitable, unless rigorous risk management procedures were implemented among drilled well water users. In reference to the WHO's ADD value, some 10 % of drilled well water users are expected to be exposed to too high arsenic doses (Fig. 13). The use of the ADD value issued by RIVM results in the fraction of some 23 %. In the case of dug well users, exposure to arsenic was negligible. In the Pirkanmaa Belt 3 % of drilled well water users would be exposed to arsenic quantities exceeding the ADD issued by WHO and 8 % quantities exceeding the corresponding RIVM value. In the case of dug wells located within PB, estimated exposure was below the former ADD in all cases and only 0.03 % exceeded the latter value.

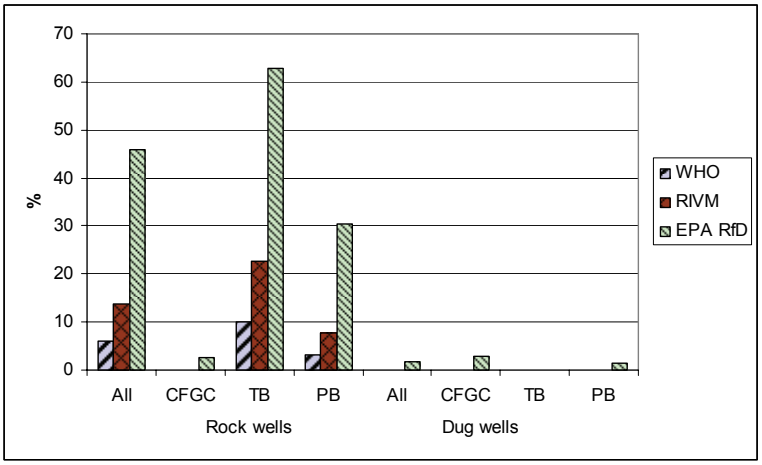

Figure 13. Estimated total intake of inorganic arsenic vs. the acceptable daily doses (ADDs) issued by WHO, RIVM and USEPA: probability of exceeding the values among the drilled (rock) and dug well water users.

Cancer risk estimates based on the oral cancer slope factor issued by USEPA are presented in Table 35. The criteria for 'acceptable risk' or 'significant risk' has not been established by the European Union. The concept of significant or acceptable risk has often been defined to mean a lifetime probability between 10^{-4} -10^{-6} of getting cancer due to the environmental pollutants (e.g., Provoost *et*

al., 2006). The upper range is suitable for a single pollutant while the lower range takes into account the cumulative effect of several pollutants. In the decree of the Council of State for the assessment of contamination level and remediation need of soil in Finland (Decree 214/2007), new guideline values were calculated using an acceptable excess cancer risk of 10^{-5} for lifetime exposure. The TDI values issued by RIVM (Baars *et al*. 2001) are based on the probability of cancer incidence of 10^{-4}. The drinking water limit value issued by WHO (0.01 mg/L) is equivalent to a theoretical cancer risk of 6×10^{-4} which is relatively high. In reference to the definitions presented above, estimated cancer risk can be considered as unacceptable in every exposure scenario studied.

Table 35. Cancer risk calculated from intake estimates (^{s}stochastic estimate, ^{p}point estimate) using the oral cancer slope factor issued by USEPA.

Exposure scenario	Calculated dose µg/kg/d	USEPA (1988)	
		Unit risk $(\mu g/kg/d)^{-1}$	Risk
People using drilled well water + background, maximum	55^{s}	1.5×10^{-3}	8.3×10^{-2}
People using drilled well water+ background, 95 % fractile	2.28^{s}	1.5×10^{-3}	3.4×10^{-3}
People using dug well water+ background, maximum	1.6^{s}	1.5×10^{-3}	2.4×10^{-3}
People using dug well water+ background, 95 % fractile	0.22^{s}	1.5×10^{-3}	3.3×10^{-4}
People using groundwater at CCA plant area+ background	$0.53–1.5^{p}$	1.5×10^{-3}	7.9×10^{-4} -2.3×10^{-3}

The biomonitoring verified exposure from drinking water, i.e., the concentrations of arsenic excreted in the urine were the highest among the users of water containing elevated concentrations of As (from drilled wells). Hence, the results supported the results from the exposure modelling. Overall, in 11 samples (out of 40), the the biomonitoring action limit (5.2 µg/l), was exceeded. Both the concentration of total arsenic and inorganic arsenic in urine correlated well with the concentration in well water ($R^2 = 0.95$, $R^2 = 0.83$). However, in few cases high urinary concentrations were detected in the case of people not exposed through drinking water. This might be associated with occupational exposure or exposure in hobbies, however, the actual reason for these deviations remained unresolved. The detailed description of the study and the results can be found in Appendix 3.

The preliminary analyses of cancer incidence within Pirkanmaa also suggest an increased number of several cancer types that have generally been associated with As exposure. The highest estimates for relative risk were observed in the case of liver cancer. In addition, the cancers of bladder, kidney and skin (excluding melanoma) might warrant more attention. Although many of the observed risks were statistically non-significant, they were observed for cancers that have previously been associated with exposure to arsenic and the risk estimates tended to be higher with higher exposure cut-points. Nevertheless, all the results need to be interpreted with caution due to several sources of uncertainty that may bias the results. The whole study on cancer register is presented in a separate report in Appendix 4.

6.3 Uncertainties of the risk estimates

The risk calculations were made for adults. For children the calculated doses might be higher meaning also higher risk. On the other hand, well water investigations have been concentrated to the locations where high arsenic concentrations of arsenic have been detected.

WHO has used an 20 % allocation to drinking water. In the study area as in the whole country, the intake of inorganic arsenic from other sources than drinking water is of minor importance and therefore, higher concentrations might in principle be tolerated for drinking water. On the other hand, cancer risk with the tolerable exposure level of 15 µg/kg/d (derived from the PTWI) is relatively high and exceeds the general acceptable cancer risk levels of 10^{-6}…10^{-4}. Nutritional status among the people living in the study area is good indicating higher tolerance to arsenic compared with many developing countries. Moreover, in Finland selenium has been added to fertilizers and selenium intake has been judged to be at a good level. Proper selenium nutrition strengthens the tolerance to elevated arsenic intake.

It must be taken into account that cancer risk calculations assume a lifetime (70 a) continuous exposure which in practice is relatively seldom fulfilled. Also, the cancer slope factor used in the calculations is quite uncertain and EPA is currently revising the cancer risk assessment for inorganic arsenic. In fact, cancer risk is probably overestimated when using the EPA linear model. For example, Lamm *et al.* (2007) observed skin cancer cases only with the highest As concentration, i.e., when the concentration in drinking water was higher than 150 µg/l. Using different models, a threshold of 122 -150 µg/l was derived. Considerations of duration, age, latency, and misclassification did not appear to markedly affect the analysis. On the other hand, Ahsan *et al.* (2006) found consistent increase of premalignant skin lesions in study populations using drinking water containing 8.1–40.0 µgAs/l or more compared to those with <8.1 µgAs/l in drinking water. The effect seemed to be influenced by gender, age, and body mass index.

Intake estimates were calculated for exposure at stable situation and no difference between home and work environments were taken into account. The scenario ‘occupational exposure’ can be used in rural environments, where people often live and work at the same location. The scenario was considered relevant because groundwater is mainly used in the rural areas while the cities are served by public water supply systems. These are based on the use of shallow groundwater and surface water reserves. The water quality in the waterworks is controlled on a regular basis and arsenic is one of the elements monitored.

According to the investigation of As speciation in drilled well waters, in most wells arsenate is the dominating species. However, the proportion of arsenite varied from 0.66 % to 73.8 % (Backman *et al.* 2006). In this study, no difference was made between the different inorganic As species since there is no scientifically sound basis to distinguish toxicologically between arsenite and arsenate. While the literature generally implies that arsenites are somewhat more toxic than arsenates, in most cases the differences are within the ranges of uncertainty. Additionally, these species are interconvertable in the human body and in the environment.

The limited number of people participating in the biomonitoring study decreases the applicability of theresults in population level. Moreover, the few exceptionally high urinary As concentrations not associated with exposure from well water reveled other, unknown intake routes. The main uncertainties included in the study on cancer incidences are related to the possible confounding factors which were not studied.

7 SUMMARY AND CONCLUSIONS

7.1 Risks at regional scale

7.1.1 Risks owing to natural arsenic

The result from the health risk assessment (HRA) based on exposure models showed that in Pirkanmaa, the overwhelming majority of the human intake of inorganic arsenic arises from drinking water. The exposure from other sources e.g., food items, was on the same level as in the whole country. The risks associated with naturally occurring arsenic depend on the source of water. In Pirkanmaa, the unacceptably high exposure to arsenic is focused on people using water from drilled wells while only a few people were considered to be liable for adverse health effects from arsenic in dug well waters. We estimated that the probability of exceeding the safe arsenic exposure level is 5.9 – 46 % within drilled well water users depending on the acceptable daily doses used in the characterization of risks. The risks are highest within the geological region Tampere belt. However, in the regional scale also the risk associated with the use of dug well water need to be considered as significant. The water supplied by the public water works is virtually arsenic-free. The results from the biomonitoring study verified the exposure to arsenic in drilled well waters and showed concentrations exceeding the reference values used in the assessment of occupational risks associated with exposure to arsenic. The epidemiological study also gave some evidence on the increased cancer incidences related to past exposure to arsenic in drinking water.

On the basis of the results from the ecological risk assessment (ERA) even naturally occurring As may pose adverse effects to the most sensitive species. Hence, we can expect that some selection of species has occured at areas with high concentrations of naturally occurring arsenic in soil. On the other hand, the highest natural concentrations in soil are found in the deeper layers which limits the exposure of biota. However, in the case of excavations, such material can be brought in to surface layers where it can pose significant risks to biota. Moreover, when arsenic is in the deeper soil layers the risks to groundwater quality may be high. Due to the toxicity and steep dose-response effects of arsenic, safety margins need special attention in areas with elevated background levels

7.1.2 Risks owing to anthropogenic arsenic

The risk assessment indicated that health risks from antropogenic arsenic are low at regional scale. However, locally health risks may be significant e.g., if old CCA wood treatment plant areas are built for housing. Elevated exposure to arsenic can cause adverse health effects particularly in young children, because children are more liable to direct exposure from soil. Health risk arising from the occupational exposure to arsenic in air dust at the old Ylöjärvi mine area appeared to be relatively small due to the limited exposure time. Health risks arising from the contamination of water system are expected to be insignificant owing to the lack of significant exposure routes.

The ecological risk assessment proved that the risks are high at old mine sites and CCA wood treatment plants. However, due to small surface area, the risks at CCA plants are spatially restricted whereas at the mine sites, the environmental effects extend to a large area reaching both terrestrial and aquatic ecosystems. At least in the mine site of Ylöjärvi, the risks to aquatic ecosystem are not expected to decrease with time considering the overwhelming amount of arsenic stored in the environment. The study on the behaviour of arsenic in the water system also showed that roughly half of the total arsenic in water is in soluble i.e., easily available and migrating form (Bilatetdin *et al*., 2007). In the vicinity of the Ylöjärvi mine site, the risks to terrestrial biota also remain if present

activities are continued and no risk management actions are taken. In the terrestrial ecosystem, dusting is obviously the main mechanism which may spread arsenic outwards from the tailings area. However, the factual spatial dimensions of the land contamination at mine sites in Pirkanmaa were not defined in RAMAS project. Hence, the question on the spatial range of ecological risks remains. The ecological risks at former CCA plants are normally quite easy to manage due to the limited size of the sites.

Both human health risk assessment and ecological risk assessment was focused on specific sites with known sources of anthropogenic arsenic i.e., on former CCA plants sites and mine sites. These site types were identified in RAMAS project as the most important ones. Hence, these were prioritized in the risk assessment and further sites studies were also concentrated on these site types. It is difficult to draw conclusions on the regional-scale risks related to environmental arsenic only on the basis of site-specific studies. The assessment of regional-scale ecological risks would have assumed data on the actual abundance and biodiversity of biota in regional level. Various site-specific conditions such as contamination level, groundwater conditions, vicinity of surface waters, land cover, existence and characteristics of potential receptors etc., also affect significantly the magnitude and probability of both ecological and health risks. Hence application of the results to other equivalent As-contaminated areas is not straightforward. There are also various other activities which might release arsenic in the environment. Such sources at least include landfills and disposal sites for wood ashes. Shooting ranges also act as sources of arsenic since the ammunition include small amounts of arsenic. The contribution of these areas as potential arsenic sources is expected to be minor compared with that of mine sites and CCA-plant sites. However, they might add to the As-related risks on regional level.

7.2 Critical data and suitability of assessment methods

Concerning the health risk assessment, new data produced in RAMAS did not have significant effect on the total risk estimates. This is due to the fact that concentration of arsenic in groundwater was well known in advance and that exposure from water composed the major part ofthe average daily intake of inorganic arsenic. New information of the concentrations of arsenic in surface soil (in farms and forest areas without anthropogenic As sources) and crops verified that these exposure routes do not contribute significantly to the total intake of inorganic arsenic. Hence, knowledge about the geological features and environmental fate of arsenic is the most important factor in the assessment of human exposure to naturally occurring arsenic. The fact that sampling of well waters has mainly been focused on areas with known high levels of arsenic is expected to cause some bias in the results on regional level.

In the derivation of health risk estimates we used several acceptable daily doses (ADDs) indicating safe levels of exposure. In fact, the main problem in risk assessment is related to the estimation of health effects at low doses. Since there is no definite ‘right value’ for ADD, the estimates of safe doses issued by different organizations vary considerably pointing out the uncertainty of the dose-response estimates.

We used several methods to derive estimates of risks to human health i.e, exposure modelling, biomonitoring and epidemiological study. The strength of such methodology comes from the possibility to verify the results from modelling which often includes various uncertainties. However, the combination of the results from different HRA methods is challenging particularly since the different methods are not temporally compatible. The assessment endpoints may also vary. While modelling can be used to assess present and future exposure and potential effects arising from it,

biomonitoring only produces information on the past exposure. In our case, biomonitoring being based on urine analyses, the results revealed the exposure within the past few days. Epidemiological studies, on the other hand, indicate the true effects caused by exposure in the remote past, in our case that is earlier than some 10 years ago. Moreover, the actual exposure to arsenic in drinking water and hence, the true dose-response connection remained unknown in the epidemiological study.

In the case of ecological risks, assessment based on modelling is still much more inaccurate compared with the equivalent assessment of health risks. This is due to the large number and variety of species and and interactions between them. For example, different uptake models used to assess risks to birds gave very different results with the maximum of almost a 30-fold difference between the exposure estimates. Hence, it is obvious that models in general can only provide rough estimates of possible risks to biota. Moreover, phenomena such as adaptation, avoidance, compensation and recovery potential are factor which have a considerable effect on the realization of adverse effects in the environment. The data needed for the use of different uptake and exposure models is also very limited and its applicability to Finnish conditions has not been studied. Due to all these limitations associated with modelling, bioassays such as ecotoxicity tests are in many cases necessary in order to minimize the uncertainties and to verify the toxic effects. In ecotoxicity tests, the joint toxic actions of concomitant contaminants can also be detected while in the case of model-based assessment, there are hardly any methods to assess these reliably. However, ecotoxicity tests, too, may have some disadvantages. Often the number of tests needed to produce adequate and accurate data for the assessment of risks is high increasing the costs of risk assessment. So-called TRIAD-methodology is a good option for ERA. TRIAD is based on the use of different methods i.e., chemical studies, ecotoxicity tests and ecological studies. The results of these different 'lines of evidence' are combined to give an overall risk estimate. Although TRIAD can be considered a good method the results are not always unambiguous. Hence, it is important to know the limitations and applicability of different methods used in order to define the reliability of the results produced in according to the TRIAD. In RAMAS no ecological studies were carried out due to lack of resources. Hence, we were not able to apply TRIAD methodology in the ERA.

At present, it is generally recommended to follow a tiered approach in risk assessment. This means proceeding from the use of simple assessment methods such as comparison of environmental concentrations with some toxicity-based benchmarks (i.e. concentration limits) towards to a more detailed assessment based e.g., on modelling, toxicity tests, ecological studies, biomonitoring. The idea of a tiered approach is to opimize the use of resources that is, to focus the collection of data to those issues which are the most crucial in the attainment of reliable results in risk assessment. In RAMAS it was not possible to follow the tiered approach since the work had to be carried out according to the predetermined work plan. In practice, this meant that the ecotoxicity tests before the start of the project and ran simultaneously with other site studies.

In RAMAS, the assessment of both human healt risks and ecological risks was based on the studies on few site types (mine site, CCA-plant, areas with high levels of natural arsenic). In practice, regional level ecological risks are not managed nor assessed form the viewpoing of a single contaminant (generally all stressors are included).

Both in the HRA and ERA, statistical analysis based on Monte Carlo simulation was carried out in order to study the uncertainty of the deterministic risk estimates. Judging from the literature, this is perhaps the most common method used in the uncertainty analysis of risk assessment related to contaminated environment. In our analyses, we ignored the possible correlations between the parameters. Such data on correlations is hardly available and producing it requires significant amount of information. The difficulties associated with the determination of correlations can be seen as one

of the major disadvantages of Monte Carlo based techniques since ignoring of important correlations may cause significant bias in the results.

7.3 Future study needs

The generic future study needs include the need for additional investigations on toxicity of arsenic, particularly in low doses, and the 'reliable safe values' for human intake. These should be clarified considering the varying conditions among the EU member states.

In the case of ecological risks, the generic data gaps include particularly the information on the bioavailability of arsenic originating from different sources i.e., from anthropogenic vs. natural sources. Unfortunately, it was not possible to point out these differences in our studies on the ecotoxicity of soil samples taken from different locations. Such information on the variation of bioavailability could be produced for example by testsing soils with identical properties other than arsenic concentrations. However, in practice such studies are very difficult to accomplish since soil properties vary considerably giving rise to differences in toxicity responses of test organisms and making it impossible to draw any conclusions in the differences of bioavailability of contaminants. Use of statistical methods could provide some answers but this would require a very large number of tests.

Concerning our study area, Pirkanmaan, information on the actual use of groundwater would be necessary in order to estimate real health risk at population level. In this study the intake estimates were based on reasonably accurate data on As concentration data but only rough estimates of the groundwater use. Exact information on use of drinking water would have been helpful also in the interpretation of the biomonitoring study and hence, a personal follow-up during the week preceding sampling might have been useful. Moreover, it would be useful to repeat the biomonitoring study using a larger study group and organizing personal interviews (for the tracking of exposure from other sources).

During the finalization of the health risk assessment, by accident we received information on an unknown potential exposure route at the Ylöjärvi mine site. This exposure route is not included in our calculations. It appeared that in summer time, a swimming school for children is organized in the adjacent lake Ylöjärvi in which very high concentrations of arsenic have been detected both in water and sediment. Since the exposure time is short the risks are expected to remain low. However, this might require some further studies. In particular, the possibility to restrict the exposure by moving such activities to other locations should be investigated.

Other study need related to site-specific risks include at least the following:

- studies on the surface soil in the farm yards to track possible additional site-related human exposure;
- additional investigations related to risks to terrestrial ecosystems and nearby residents at Ylöjärvi mine site, including both chemical and ecological studies in order to track the spatial scope of contamination by As;
- ecological studies on the watersystem affected by the effluents from the Ylöjärvi mine site in order to track the risks to aquatic biota;
- study on the possible confounding factors involved in the epidemiological study (such as socio-economic factors, actual exposure through drinking water);

- additional studies on other possible sources of As-associated risks, particularly: rock engineering (quarries), landfills (leachates and their capability to mobilize naturally occurring As from soil).

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APPENDIX 1

Summary of the chemical data available for risk assessment.

Table 1. Arsenic concentrations at former CCA wood treatment sites (Parviainen *et al*., 2006). n = number of analyses.

Site	Contaminated area, m^2	Concentration in soil, mg/kg		Concentration in water, µg/l	
		Average	Median	Groundwater	Surface water
Ruovesi I	1,000	1,152 (n=7)	440	assessed	49
Ruovesi 2	-	1,474 (n=15)	320	-	-
Vilppula	850	82 (n=20)	18	11 – 57	250
Virrat	11,600	220 (n=39)	140	-	-

Table 2. Concentration of arsenic (µg/l) at the various monitoring points of the Vahantajoki river basin connected with the Ylöjärvi mine site. Data compiled by Bilaletdin *et al*. (2007).

Sampling point	Year	Mean	Min	Max	Med	N
Ditch from tailings to the Lake Parosjärvi	1982-1999	258.8	43	580	250	25
Lake Parosjärvi surface	1975-2005	66.6	0.5	160	68	58
Lake Parosjärvi bottom	1975-2005	155.3	1.2	910	130	56
Stream Parosjärven oja 1	1975-2005	60.2	1	380	60	68
Stream Parosjärven oja 2	1975-2005	57.9	0.5	850	31	73
Stream Vähä-Vahantajärven oja	1975-2005	16	0.8	65	14	73
Stream Vahantajoki alav mts	2005	7.1	4	14	6.3	9
Lake Näsijärvi surface	2005	2.9	1.5	6	3	8
Lake Näsijärvi bottom	2005	23.4	6	66	14	8

Table 3. Concentrations of arsenic in the river surface sediments (A) and in the lake sediment cores (B) of the Vahantajoki river basin connected with the Ylöjärvi mine site. The distances downstream are
average distances from the Lake Parosjärvi (Parviainen *et al.*, 2006).
A.

Stream sediment sampling point	Concentration, mg/kg
Stream Parosjärven oja, ~ 300 m downstream	110
Stream Parosjärven oja, ~ 900 m downstream	128-228
Stream Parosjärven oja, ~ 1,300 m downstream	134
Stream Vähä-Vahantajärven oja, ~ 6,000 m downstream	25

Table 3 continues…

B.

Sediment core sampling point	Depth, cm	Concentration, mg/kg
Wetland below the dam in Lake Parosjärvi	0-50	2,600-4,480
	51-171	1,130-1,590
	107-178	1,760-3,690
	180-183	179
Lake Vähä-Vahantajärvi ~ 3000 m	top	200-500
	8	583
	10-15	54-246
	30	54
Lake Näsijärvi ~ 7000 m	4-5	37
	20-21	235
	29-30	17

Table 4. Concentration of arsenic in surface water (µg/l) at sites in which quarrying has been practiced.

Sample 1 pond	Sample 2 stream	Sample 3 pond	Sample 4 pond	Sample 5 pond	Sample 6 pond
5.43	2.19	6.79	3.82	2.98	27.3

Table 5. Concentrations of arsenic in some food items (documented in Mäkelä-Kurtto *et al*., 2006).

Food item	Concentration mg/kg-fw	Concentration range
Milk	0.00012	0.0009 - 0.0016 mg/kg fw
Wheat	0.006	<0.002 - 0.016 mg/kg dw
Oats	0.024	<0.004 - 0.063 mg/kg dw
Barley	0.01	<0.005 - 0.022 mg/kg dw

Table 6. Concentration (mg/kg-dw) of arsenic and metals in the samples studied in the ecotoxicity tests.

aqua regia digestion								
sample	**As**	**Cd**	**Co**	**Cr**	**Cu**	**Ni**	**Pb**	**Zn**
Natural 1	3.47	0.5	15.9	40.9	14.0	19.6	14.1	97.4
Natural 2	13	0.5	24.6	56.5	23.2	27.6	15.7	111
Natural 3	5.71	0.5	27.2	59.3	26.7	30.7	17.1	176
Natural 4	29.9	0.5	7.39	44.7	23.6	12.8	12.7	47.4
Natural 5	111	0.5	12.7	55.6	38.9	21.3	11.7	54.1
Natural 6	10	0.5	7.70	17.8	11.9	8.37	7.29	37.1
Natural7	10	0.5	11.3	32.3	21.0	12.8	18.2	65.5
CCA plant 1	421	1.23	3.99	228	183	6.45	12.5	21.5
CCA plant 2	351	1.08	3.64	126	153	6.22	13.4	22.5
CCA plant 3	261	0.77	3.82	128	144	5.94	7.27	21.0
CCA plant 4	724	2.09	3.65	291	269	6.47	11.9	24.8
CCA plant 5	1960	5.40	3.44	875	910	7.43	23.5	32.1
CCA plant 6	4080	9.02	2.54	1990	1050	6.72	23.7	18.5
CCA plant 7	49.6	0.5	4.10	57.8	27.6	5.69	6.70	18.9
Mine 1	2380	4.67	20.4	29.6	125	14.8	25.6	224
Mine 2	2070	3.87	12.1	31.7	70.4	12.9	25.2	219
Mine 3	1060	2.07	7.20	33.2	31.6	11.9	25.5	188
Mine 4	2340	4.46	15.5	33.1	120	14.4	30.2	226
Mine 5	2280	4.20	8.77	30.7	39.3	11.6	25.8	180
ammonium acetate soluble								
	As	**Cd**	**Co**	**Cr**	**Cu**	**Ni**	**Pb**	**Zn**
Natural 1	3	0.1	0.94	0.35	3	0.46	2	0.8
Natural 2	3	0.1	2.02	0.36	3	1.16	2.05	1.07
Natural 3	3	0.1	0.9	0.3	3	0.63	2	0.8
Natural 4	4.82	0.1	0.55	0.3	3	0.21	2	0.8
Natural 5	4.84	0.1	0.38	0.3	3	0.14	2	0.8
Natural 6	3	0.1	0.3	0.3	3	0.1	2	0.8
Natural7	3	0.1	0.3	0.3	3	0.29	2.43	0.8
CCA plant 1	29.6	0.1	0.3	4.83	55.4	0.1	3.13	1.33
CCA plant 2	26.9	0.1	0.3	4.26	42.5	0.1	2	1.29
CCA plant 3	20.4	0.1	0.3	4.02	43.0	0.1	2.19	1.23
CCA plant 4	46.3	0.12	0.3	8.21	94.9	0.1	3.03	2.75
CCA plant 5	137	0.34	0.3	20.2	511	0.20	6.32	7.05
CCA plant 6	151	0.40	0.3	17.4	546	0.15	4.46	3.12
CCA plant 7	3	0.1	0.3	3.65	12.0	0.1	2	0.8
Mine 1	703	2.01	2.21	0.3	28.9	0.48	4.93	4.78
Mine 2	602	1.76	0.45	0.3	18.3	0.12	2.36	1.15
Mine 3	193	0.49	0.3	0.3	4.47	0.17	2	0.8
Mine 4	627	1.82	0.78	0.3	22.6	0.30	4.67	2.88
Mine 5	454	1.25	0.3	0.3	11.4	0.13	3.44	0.8

APPENDIX 2

Laboratory methods used in the ecological risk assessment

Leaching tests

Standardized batch leaching test EN 12457-3 was used to study the leaching properties from soil samples. This test is a two-stage test intended for compliance test of waste destined for a landfill. First, the samples were agitated at a liquid to solid ratio of 2 L/kg (L/S 2). Then the liquid was separated by filtration (membrane filter poresize 0.45 μm). At the second stage the solid fraction was agitated again with a fresh water at a L/S-ratio of 8. The eluates of the two stages were used for chemical and ecotoxicological analyses (luminescent bacteria test and RET test). Results of the chemical analyses were calculated according to standard EN-12457-3 as the leached amount (mg/kg) relative to the total mass at the first stage (L/S 2) and cumulative L/S 10 ratio.

Ecotoxicological methods

Seed germination

Seed germination tests were performed with two different plant species: ryegrass (*Lolium multiflorum*), lettuce (L*actuca sativa*). The lettuce test followed the method of ISO 17126 and the ryegrass method was a modification of the ISO standard 11269-2. Test conditions in test chambers were the same for both species: temperature 20 °C, light 4300 ± 430 lux , light cycle16 h light/8 h dark, humidity 80 %. At the beginning of the test, the test vessels were kept in dark for the first 48 hours followed by light cycle until the end of test period. Clean natural sand was used as the control soil. Dry seeds were put on top of the test soil, 90 g of the cover sand was spread out evenly on the top of the seeds. The material was wetted with de-ionized water. At the end of the test the number of the germinated seeds were recorded. Results were calculated as percentage inhibition and the statistical significance was calculated with Student's t-test (SPSS software, version 11.0).

Duckweed growth inhibition test

Phytoavailability of the soil contaminants via water and effects on duckweed plants were demonstrated by a modification of the standard growth inhibition test (ISO/FDIS 20079). Effects of RAMAS samples on duckweed growth were measured in the presence of 50 g of solid samples in 100 ml of growth medium. The medium contained all nutrients necessary for normal growth. Duckweed (*Lemna minor*) plants are small free floating plants, which take all nutrient directly from water. According to the standard's scope the test is suitable for testing of wastewaters and water soluble chemicals. The present modification of standard test measures the effects of easily leachable contaminants, because no shaking of the soil and solution was performed. Plants were grown at 20 ºC for 7 days and the number of the fronds and the frond area were measured as test parameters using image (Scanalyzer, LemnaTec GmbH, Germany). Plants were grown in glass vessels (diameter 80 mm), which were covered by a glass lid. In the control vessels, soil was replaced by clean natural sand. Growth in control and sample vessels were compared and the effects were calculated as percentage inhibition of growth.

Enchytraeid survival and reproduction test

Pot worm. *Enchytraeus albidus,* test was performed according to the ISO standard 16387, Annex B. Briefly, ten adult worms per vessel containing 30 g test material were incubated for three weeks to determine the acute toxicity. After the acute phase, adult worms were removed and incubation was continued for 3 additional weeks to assess the effects on reproduction. At the end of the test, juveniles were isolated from the test material, and the animals were stained and the numbers were counted. Samples were diluted with the artificial soil (OECD artificial soil, containing Sphagnum peat, kaolinite and crushed quartz, pH adjusted to 6,0 ± 0,5) to different test concentrations. Chemical concentrations o f the contaminants served as the basis for selection of samples to be tested and the dilution series. The aim was to calculate the EC50 –values for both mortality and reproduction using probit -analysis (SPSS software, version 11).

Earthworm survival and reproduction test

Earthworms (*Eisenia fetida*) survival and reproduction tests were performed according to standards ISO 11268-1 and ISO 11268-2 with some modifications. The standard procedure was followed in other respects, but the number of animals was reduced from 10 to 6 and the number of replicates was three instead of four, and the test material per vessel was 200 g instead of the 500 g recommended in the standard. The vessels were incubated 4 weeks for determination of survival. Adult worms were removed, and incubation was continued for 4 additional weeks. At the end of test, juvenile worms were isolated from the medium for counting. Nine samples out of the 19 samples in total were investigated either at a single concentration or multiple concentrations. The aim was to determine the EC50 –values as in the pot worm test, although for reproduction this was possible only in two cases.

Luminescent bacteria test

Luminescent bacteria test was performed as the "flash" version of the standard test (ISO SFS-EN 11348-3) using the freeze-dried bacteria (*Vibrio fischeri* NRRL B-11177) and BioTox™ -reagents (Biotox, Finland). One gram of solid sample was mixed with 10 ml of 2 % NaCl solution in a vortex shaker. Then, soil suspensions were diluted with 2% NaCl-solution to attend a series of five dilutions of soil suspensions. After that, 300 µl of bacteria suspension was injected into the dilutions and kinetic measurement of luminescence was started. The luminescence was measured every 0.2 second during 30 s and also after 30 min incubation in a Sirius Luminometer (Berthold Detection Systems, Germany). The percentage inhibition of light production compared to the control (2 % NaCl-solution) were used to calculate the EC50 –values. Acute toxicity of the leaching test eluates was measured according to the standard ISO SFS-EN 11348-3, but the luminescence was measured with the kinetic Flash method in a Sirius Luminometer. The dilutions of 0, 3.13, 6.25, 12.5, 25, 50 vol/vol % were prepared with NaCl-solution to obtain a 2 % NaCl concentration. The percentage inhibition of bioluminesence after 30 minutes was used to calculate the EC50-values.

Reverse electron transport, RET

Samples were extracted with de-ionized water prior to RET test using 10 g sample and 10 ml water. The slurry was mixed in a rotary shaker for one hour, filtered and centrifuged to get a clear supernatant for the assay. Reverse electron transport is an enzymatic chain of reactions taking place in mitochondria for energy production. These reactions are ubiquitous among eukaryotic cells and hence, effects in RET reactions should represent possible effects on a

wide range of organisms. As it is an *in vitro* test, it measures effects in direct contact with chemical compounds and only serves to detect the potential toxicity to whole organisms. RET test was also performed for the leaching test eluates of the selected set of samples. Leaching tests were carried out according to the standard EN-12457-3 Iin a two-stage batch leaching tests. Before RET assay the pH of the samples was adjusted with 0.1 mol/L NaOH to 7.5 ± 0.2. The assay is based on the use of sub-mitochondrial particles prepared from isolated beef heart mitochondria (Knobeloch *et al*., 1994; Read *et al*., 1998). The reaction mixture consisted of succinate, antimycin A, NAD+, ATP, sub-mitochondrial particles and sample dilution or water as control, in HEPES buffer, pH 7.5. The reduction of NAD+ to NADH was measured kinetically at 340 nm in a microplate reader (iEMS, Ascent, Labsystems, Finland). Sample extracts were diluted with water in twofold serial dilutions to achieve assay concentrations from 78, 3 to 0.038 % of the extracts. The enzyme activities from the sample dilutions and controls were compared to calculate the inhibition percentages. EC50 -values were estimated from the regression curves of inhibition versus sample concentration.

APPENDIX 3

Biomonitoring study for the health risk assessment in the Tampere region

Report for the Risk Assessment and Risk Management Procedure for Arsenic in the Tampere Region (RAMAS) –project

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1. Introduction

As part of the RAMAS project, a human biomonitoring study was conducted in the study area, Pirkanmaa. The idea was to verify the results from the modelling of human exposure and to test the suitability of biomonitoring as a tool for regional-scale risk assessment procedure. The study was carried out by the Finnish Institute of Occupational Health (subcontracting work paid by the Geological Survey of Finland) and Finnish Environment Institute (RAMAS –partner).

The level of human exposure to arsenic can be estimated with a range of biomarkers (Hughes 2006). We chose the most common biomarker, the analysis of arsenic in urine and designed a set of questionnaires to support the interpretation of the analysis results. In order to have a more definitive indication of the exposure to inorganic arsenic, the chemical species of urinary arsenic were determined using the method developed for the assessment of occupational As exposure in Finland (Hakala, 1995a). The same method was also used in an earlier study on exposure to arsenic, also carried out in Pirkanmaa (Kurttio *et al.* 1998). This study, however, was spatially restricted and covered only one small town.

Absorbed arsenic is excreted primarily in urine, with a half-time of approximately four days (NRC, 1999; WHO, 2001). Inorganic arsenic is excreted as inorganic arsenic (arsenite, As^{3+} and arsenate, As^{5+}) and as biotransformed arsenic species (MMA = monomethylarsonate and DMA = dimethylarsinate). The concentration of arsenite and arsenate or that of the metabolites, expressed either as inorganic arsenic (As-i) or as the sum of metabolites (As-i + MMA + DMA = As-tot) provide the best quantitative estimate of recently absorbed dose of inorganic arsenic (WHO, 2001). The indicator As-i is not disturbed by methylated As (mainly DMA). This is an advantage when the actual exposure is not known in detail. For example, one As-containing marine fish meal prior to sampling may increase the urinary DMA concentration above 600 µg/l (Finnish Institute of Occupational Health, unpublished data).

2. Materials and methods

2.1. The study population

The participants of the human monitoring were primarily selected from the 13 farms that had been involved in the RAMAS project and given permission to environmental sampling in their property. We received urine samples from 40 persons, including 24 persons from nine study farms and 16 persons from other households. The study population represented inhabitants within the two southern geological belts (TB, PB) of As anomaly area and seven municipalities.

2.2. Sampling procedure

All the households involved in the biomonitoring were asked to fill a short questionnaire concerning the use of their well water. In addition, everybody received a personal questionnaire recommended to be filled during the week of sampling. This personal questionnaire included, among others, a personal estimate of the intake of fluids from different sources and information on daily diet and possible exposure to arsenic at work or during spare time. Special attention was paid to the consumption of food items which might contain elevated amounts of arsenic, e.g., crustaceans, rice, seaweed, and the crop cultivated at the own or neighbouring property.

Detailed written instructions on sampling, plastic sampling vessels and material with a pre-paid parcel for posting were sent to each household participating in the study. Each sample vessel was marked with a unique code to ensure anonymity.If samples were not mailed at the very day of sampling, the study participants were instructed to keep their samples in a refrigerator. In the laboratory, the samples were refrigerated until analysis without any pretreatment. The samples arrived between November 7th and 30th 2006 and the laboratory analyses were run in February 2007.

2.3. Urine analysis and treatment of results

Arsenic species were determined by a hyphenated high performance liquid chromatography - hydride generation - atomic fluorescence spectrometry (HPLC- HG-AFS) technique (Hakala, 1992). Because of the higher sensitivity and linearity, atomic fluorescence detection was used instead of atomic absorption described in the original procedure (Hakala, 1995a). The limit of quantification for As species was 2.5 nmol/l (0.2 µg/l) and the overall uncertainty of the analyses was 15 - 20 % at the concentration level of 70 nmol/l (5.24 µg/l). The urinary concentrations of arsenic were standardized to a relative density of 1.024 to gain comparative results.

To study the concentrations of arsenic species in the urine samples the study population was divided into two groups: A) persons who were known to use household water with elevated As concentration and B) persons who used water from a public water supply network or other As-free water as their household water. The concentrations of inorganic arsenic (As-i), and total arsenic (As-tot) were calculated before statistical evaluation. We also determined the ratio of inorganic arsenic to the total arsenic, and the ratio of trivalent arsenic (As^{3+}) to the inorganic arsenic. Moreover, we studied the correlation of the As in urine with the As in well water using regression analysis.

2.4. Concentrations in well water

Well water samples for arsenic analyses were taken in 2002 and 2005 by the Finnish Geological Survey and analyzed in the Geolaboratory of the same institute using inductively coupled plasma (ICP) mass spectrometric (MS) analysis (Backman *et al.* 2006).

Some of the households involved in the biomonitoring study were served by registered waterworks. The water delivered by these waterworks is analyzed regularly based on a monitoring program and controlled by local health authorities. Therefore, we asked the local authorities to provide us with an estimate of arsenic concentration in the water supplied for our study households. Unfortunately, no information on the analysis methods associated with these estimates was readily available. According to the monitoring records, the concentrations had rarely exceeded 1 µg As/l, and only once a concentration of 3 µg As/l had been reported.

3. Results

3.1. Characteristics of the study population

According to the replies to our questionnaire, most of these people spent their weekdays mainly at home and only less than one third worked or studied outside their home during weekdays (Table 1). From those 9 households (19 persons) that reported having a well drilled into bedrock six households reported that they also have an old dug well. Water used for cooking or in housework came almost without exception from the same source as drinking water. The irrigation water was also usually taken from the same source as drinking water.

Table 1. Some characteristics of the study population, information collected using questionnaires.

Characteristics	Variation	Number of persons (out of 40)	Comments
Age (years)	15 - 83 (median 49)		
Mainly at home during weekdays		29	
Farm animals		3	
Household well as main source of drinking water		20	9 wells drilled into bedrock, 1 dugwell
Well drilled (year)	1960-2004		Mainly between 1984 -1998
Beverages from food shops as main source of drinking water		2	
Connected to public water supply network		17	5 households or farms
Self-reported daily fluid intake (l) **< 2** **> 2**	1-5	 9 31	
Surface water for irrigation		2	

3.2. Arsenic exposure from other sources than water

According to the responses given to the questionnaires, in our study population the most frequently used local food items included tuberous plants, potatoes being the most prevalent (Fig. 1). Very few people reported the use of potentially arsenic-rich food items once a week or more frequently (Fig. 2). Some respondents reported having handled material which may have contained arsenic (e.g., wood treated with copper-chromium-arsenate, i.e., CCA chemical) at work or in hobbies (Fig. 3).

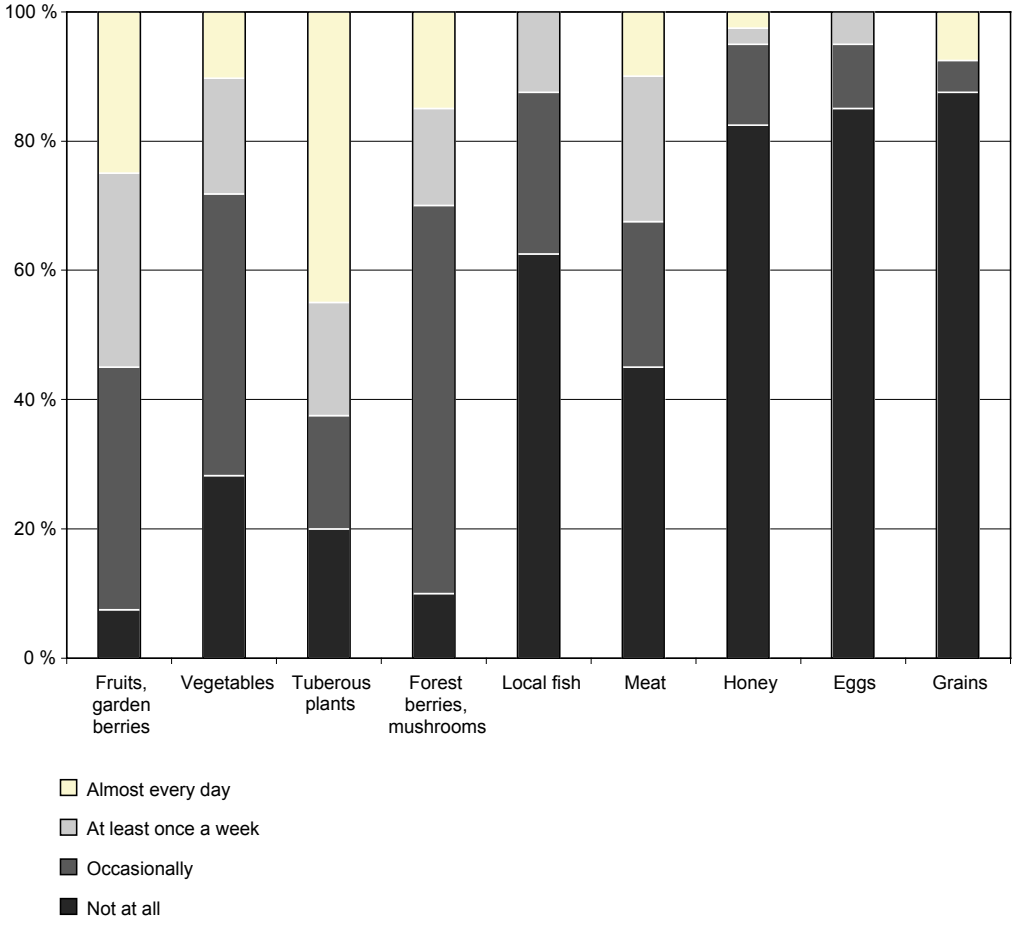


Figure 1. Characteristics of the study population: the use of food items grown at the own property or at the neighbouring property. Results based on the questionnaire.

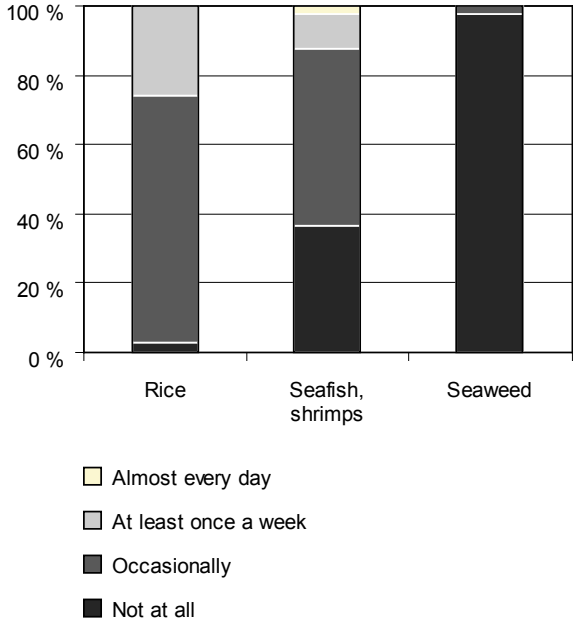


Figure 2. Characteristics of the study population: the use of food items which may contain elevated amounts of arsenic. Results based on a questionnaire.

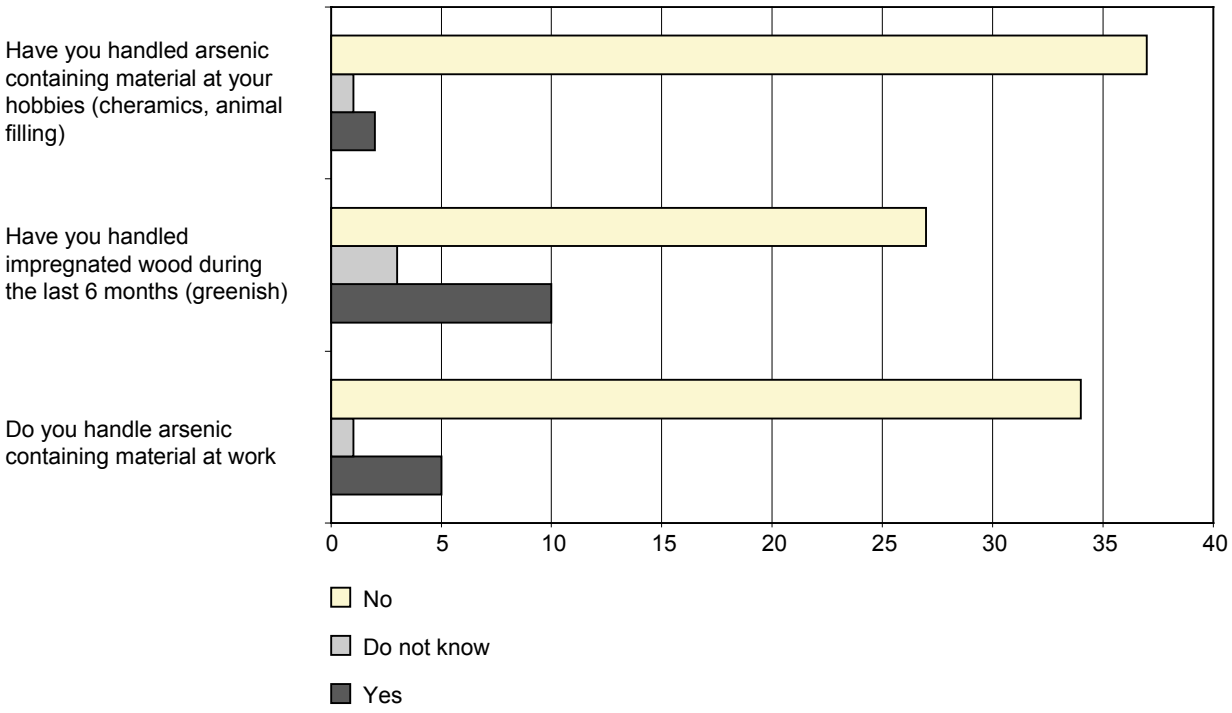


Figure 3. Results from the questionnaire targeted to the study population: handling of material which may contain arsenic.

3.3. Arsenic in urine

On the average, the concentrations of all arsenic species excreted in the urine were the highest among the users of water containing elevated concentrations of As (Table 2). The medians of the total concentration were 14.4 µgAs/l for the whole study group and 20.3 µgAs/l for group A (users of arsenic-containing household water), and 9.6 µgAs/l for the group B (users of public or other

water supply). Within the two groups the concentration of arsenic in urine varied between 8.3 and 346 µg/l and 3.3 and 24 µg/l, respectively.

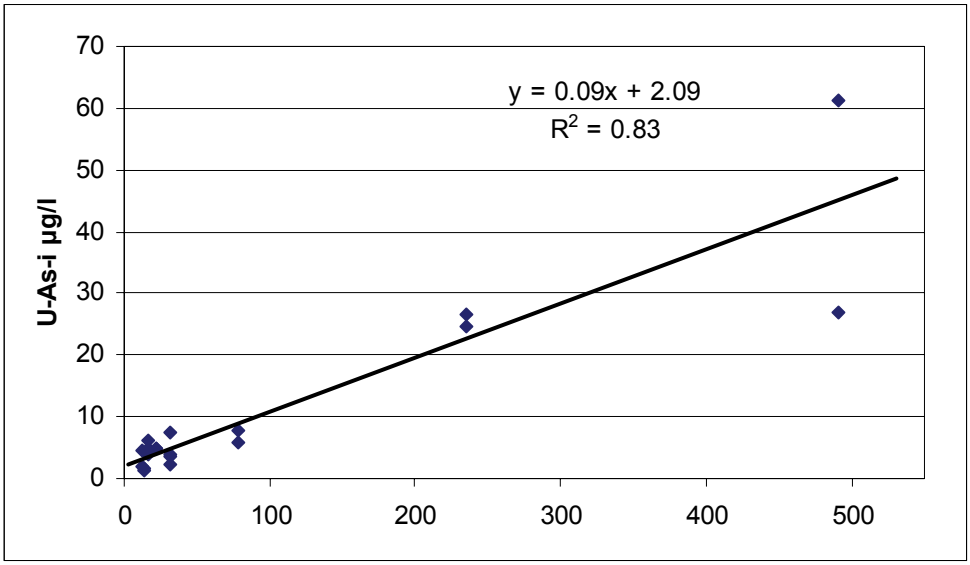
The ratio of inorganic As to the total As was about 20 % in the group A, and almost twice as high in the group B (37 %) whereas the ratio of trivalent As to the inorganic As was lower, i.e., 24 %, in group B compared to that of group A (41 %).

Table 2. Results from the biomonitoring: the concentrations of arsenic species in urine (as arsenic), calculated values for inorganic and total arsenic, the proportions of inorganic arsenic to total arsenic, and trivalent arsenic to inorganic arsenic, and the respective concentration of arsenic in household water. The results from another study in Pirkanmaa (Kurttio *et al.*, 1998) are presented for comparison. U-As = As in urine, U-As-i = the sum of As^{3+} and As^{5+} in urine.

	As^{3+} µg/l	**As^{5+} µg/l**	**MMA µg/l**	**DMA µg/l**	**U-As-i µg/l**	**U-As(tot) µg/l**	**U-As-i/ As(tot) %**	**As^{3+}/ U-As-i %**	**As in water µg/l**
Whole study population n = 40									
Median	0.9	2.6	1.3	8.6	4.1	14.4	26	35	
Arithmetic mean	3.0	3.9	7.1	25.3	6.9	39.3	29	34	
Range	<0.2 - 35	0.3 - 26	<0.2 - 73	0.9 – 212	0.7 - 61	3.3 - 346	9 - 71	1 - 75	<1 – 491
Group A: users of water with elevated As concentration n = 20									
Median	1.7	3.2	3.6	12.2	4.8	20.3	20	41	27
Arithmetic mean	5.1	5.3	13.6	44.2	10.4	68.2	21	42	95
Range	<0.2 - 35	0.5 - 26	0.2 - 73	4.8 – 212	1.2 -61	8.3 - 346	9 - 38	1 - 75	12 – 491
Kurttio *et.al* 1998, n= 35									
Geometric mean			5	39	11	58	19		170
Range			0.6 - 117	0.8 – 351	2 - 106	3 - 500	6-53		17-510
Group B: users of public or other water supply n = 20									
Median	0.7	2.3	0.6	4.6	3.1	9.6	37	24	
Average	0.8	2.6	0.7	6.3	3.4	10.4	37	27	
Range	0.3 - 2.9	0.3 – 5.2	<0.2 - 1.4	0.9 – 18	0.7 - 6.1	3.3 - 24	14 - 71	9 - 51	<1 (max 3)
Kurttio *et.al* 1998, n= 9									
Geometric mean			0.8	5	2	5	22		<1
Range			0.6-3	2-37	1-5	4 - 44	4-42		

For the regression analysis, the correlation coefficients were calculated solely on the basis of the arsenic concentrations in well waters and the respective urinary As of their users. The results are presented in Fig. 4. Unfortunately, it was not possible to determine reliable estimates for the volume of water consumed by each person on the basis of the responses given in questionnaires. Hence, it was impossible to calculate the intakes of arsenic at a reasonable exactness and reliability.

a.



b.

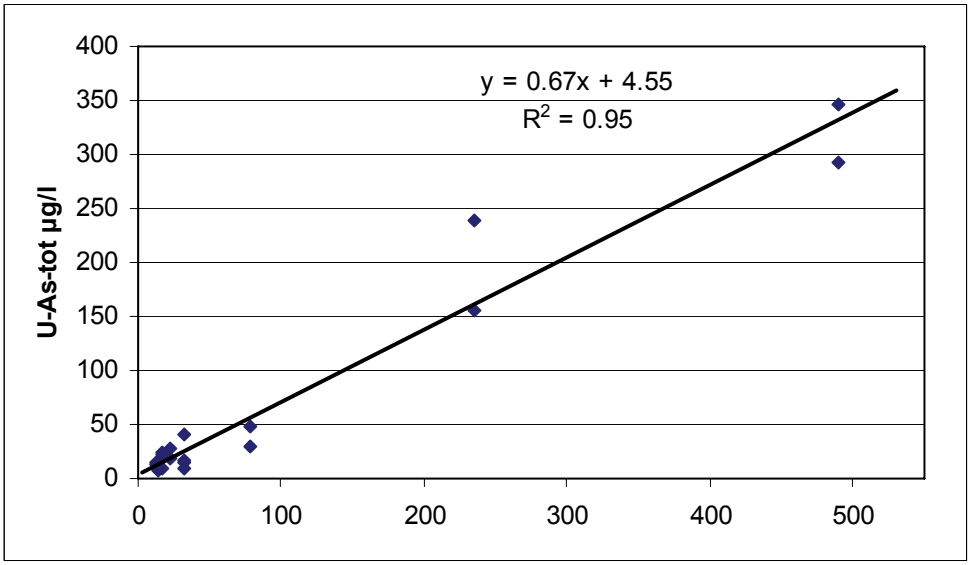


Figure 4. Results from the biomonitoring: correlation of a) urinary inorganic arsenic and b) total arsenic to the concentration of arsenic in well water.

The correlation coefficients (R^2) were quite high, i.e., 0.83 and 0.95 for inorganic arsenic and total arsenic, respectively. The result also show rather low variability within a single household, i.e., a relative standard deviation of 3 - 57 % for inorganic As and 12 - 48 % for total As with geometric means of 27 % and 30 %, respectively. However, in some households the deviations were high, and even so that when the deviation of inorganic or total As was high the other was low. The high deviations can be related to an exceptional high water consumption before the urine sampling, or very different water consumption, or exposure to arsenic from other sources (see the discussion, section 4).

4. Discussion

4.1. Connection between urinary arsenic and health risks

The concentration of 'metabolites' of inorganic arsenic (As^{3+} and As^{5+}) and methylated species MMA and DMA in urine reflects the absorbed dose of inorganic arsenic through all possible exposure routes on an individual level. Hence, the measurement of total arsenic or arsenic species in the urine does not give any information on the contribution of different absorption routes. This is due to the fact that the metabolic patterns of inorganic arsenic are the same for all As exposure routes.

Currently, there are no reference values for urinary As concentrations which could be used in the assessment of health risks owing to exposure from drinking water. However, for occupational exposure limit values of biomonitoring exist in many countries. Various units, e.g. µg As/l, nmol As/l, µg As/g creatinine or µmol As/mol creatinine, are used in the connection of limit values. Hence, one must be careful in the interpretation of different values. Standardization of analysis results is usually done with respect to the specific gravity of urine or to the excretion of creatinine. If the concentration of creatinine is not given (this is the normal case) the As concentrations per the volume of urine and per creatinine are not comparable.

The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended the use of the value of 35 µg As/l in urine as a reference value for exposure (ACGIH, 2007). This level refers to a guideline for potential occupational health hazards and covers both excreted inorganic As and its methylated metabolites. Most reference values of arsenic are actually based on the concentration the metabolites of inorganic As. However, consumption of certain seafood may confound the estimation of the exposure to inorganic As owing to the metabolism of arsenosugars to DMA in the body or the presence of DMA in seafood. Therefore, the evaluation of occupational exposure to inorganic arsenic should rather be based on the measurement of inorganic As species in urine than on the measurement of all As metabolites (WHO 1996).

In Finland, the limit values for occupational As exposure are based on the concentration of inorganic As. The reference value for non-exposed workers is 30 nmol/l (2.25 µg/l, U-As-i) and the biomonitoring action limit is 70 nmol/l (= 5.24 µg/l) (Hakala 1995b, FIOH 2007). The reference value is equivalent to the 95th percentile of the concentration of arsenic detected in non-exposed Finnish workers (Finnish Institute of Occupational Health, unpublished). The biomonitoring action limit is a guideline issued by the Finnish Institute of Occupational Health. The value was derived on the grounds of good working habits considering the occupational exposure limit of 10 µg As/m³ in work place air, the exposure limit being based on the risk of lung cancer in long term exposure (STM, 2005). The resulting Finnish biomonitoring action level is quite well in accordance with the value issued by ACGIH (35 µg As-tot/l) since usually about 10 - 20 % of the intake of inorganic arsenic is considered to be excreted in inorganic form (WHO, 1981; WHO, 2001). However, there is significant inter-individual variability in methylation patterns and hence, in the proportions of As species in urine.

Globally, the total arsenic concentration in urine ranges from 5 to 20 µg As/l, but may even exceed 1000 µg/l (WHO, 2001). In our study group, the maximum concentration detected was considerably higher, i.e., 346 µg/l. The concentration of inorganic As in urine (U-As-i) varied between 0.7 and 61 µg/l. Elevated concentrations are expected to originate from the drinking water containing high concentrations of arsenic, or to the occupational exposure or to ingestion of arsenic in the diet.

Compared to the Finnish reference value for occupational exposure (2.25µg/l), the median (and average) concentration of inorganic As in our whole study group was higher, indicating higher exposure to arsenic than on the average in Finland. The median concentration of inorganic As was slightly smaller in the study group of Kurttio *et. al* (1998), but still higher than the occupational limit value for exposure. Only in four samples in our subgroups A and B the U-As-i was below the reference value. The increased As concentrations in the group A is expected to be associated with the elevated concentration of As in drinking water. In this group, the biomonitoring limit value for action (5.2 µg/l), was exceeded in eight samples (max 61 µg/l), but also in three samples (max 6.1 µg/l) belonging to the group B (no drinking water related As exposure). Kurttio *et. al* (1998) also detected quite similar concentrations (max 5 µg As/l) of on their small (n=9) reference group (people who did not use drilled well water).

At the U-As levels above the biomonitoring action level, the risk of As-induced lesions are possible. Increased risk of lung cancer has been observed at cumulative exposure levels ≥ 0.75 mg/m³ · year (WHO 2001), e.g. when workers have been exposed to 17 µg As/m³ (ca. 2-fold risk) during 45 years . This would correspond with the U-As-i of approximately 9 µg/l, which was exceeded in the group A of our study population in the case the concentration of 100 µg/l in well water was exceeded. The arsenic concentrations of 50 µg/l and over, in water, has been associated to increase the risk of arsenic induced cancers.

The register of the incidence of occupational diseases during the years 1996 - 2002 includes two cases related to As exposure: one irritation-contact dermatitis and one case classified belonging to 'other diseases' (Finnish Institute of Occupational Health, unpublished). Both cases are associated with exposure to wood preservatives (CCA chemical). The register contains no cancers induced by arsenic.

Proportions of inorganic arsenic species may give some reference to the valence state of absorbed arsenic. For example, in the areas with elevated concentrations of As^{5+} owing to emissions from a copper smelter (e.g. anode casting), also the workers' urine have contained elevated concentrations of pentavalent arsenic (Hakala, 1995b). However, valence states can change *in vivo* in the body and thus, the relevance on the toxic effects might be minimal even though trivalent As is considered to be more toxic than pentavalent As.

4.2. Uncertainties involved in the study

Contrary to our expectations, we detected several elevated As concentrations also in the study group B, in which there was no exposure associated with drinking water. We can not give any definite explanation for the elevated U-As levels in our study group. The highest U-As values and additional four values ranging from 2.3 to 4.4 µg As/l might be explained by exposure associated with handling of impregnated wood In addition, other explanatory factors can be identified, e.g.
- handling of other As-containing materials
- ingestion of As-rich food (e.g., rice) before sampling
- inhalation of soil dust.

Due to the fact that arsenic has historically been involved in numerous materials, it is also possible that some hobbies may give rise to elevated As exposure. Inorganic arsenic compounds have been traditionally used in Finland, e.g. in conservation of animals for various collections (taxidermy). Arsenic has also been used in glass and ceramics industry for certain colour shades (white, blue) and controlling of the firing temperature.The responses given in the questionnaire forms

considering possible exposure at hobbies or work were not detailed enough to verify any specific explanation.

It is known, that some food items contain arsenic also in an inorganic form. However, the data is very limited. According to the replies given to our questionnaire there were some differences in the diet, e.g., in the use of rice which has been identified as one potential source of arsenic (see Fig. 1 and Fig. 2). Unfortunately, there was no detailed data available on the As content of food items consumed by our study population.

Since the sampling of urine samples was carried out in winter time with soil covered by snow, it is improbable that inhalation of soil dust would cause any significant exposure to arsenic although a significant proportion of indoor dust may originate from the outdoor air. In the study of Trowbridge and Burmaster (1997) the 50^{th} percentile for the ratio of indoor dust to outdoor dust was 42 %. The amount of dust originating from outdoors is naturally highly dependent on the frequency of cleaning activities. Moreover, the particle size affects the amount of particles deposited on surfaces versus those that remain in air. It is noteworthy that the concentration of As in the soil or in air in the courtyards of the study farms were not investigated within RAMAS and consequently, we have no data to confirm whether soil dust can be a significant source of As exposure.

The correlations observed between urinary arsenic and As concentrations in household water were quite high, and might be even higher if we had the exact information on the actual volume of ingested well water to use in the regression analyses. This would have called for detailed individual interviews along with the sampling of urine. Also, in addition to the confounding factors, described above, the As concentrations in wells may have been changed by the time of urine sampling since the wells were analysed 1 – 4 years earlier. In fact, the monitoring data associated with a couple of wells included in our study show the maximum, almost fivefold difference between the As concentration analysed in 2002 (109 µg As/l) and 2005 (513 µg As/l). The correlation between U-As-i and As concentration in drinking water is also affected by inter-individual variations of the proportion of U-As-i to the total arsenic (9 – 38 % in the group A). The lower average proportion of inorganic As among the group A compared with group B may indicate more efficient biotransformation from inorganic to methylated species (metabolic induction).

5. Conclusions

Monitoring of urinary inorganic arsenic and it's metabolites describes well the exposure to inorganic arsenic. The temporal variation of inorganic As is faster than that of the total As and it is not disturbed by methylated As (DMA). This is an advantage when the actual exposure is not known in detail, for example one As-containing meal prior to sampling may increase the urinary DMA concentration to over 600 µg/l (Finnish Insitute of Occupational Health, unpublished data).

Concentrations of As species or total-As in urine are distinct indicators of exposure to arsenic and they provide the best estimates of recently absorbed arsenic. However, there are some restrictions in the assessment of risks induced by As containing water on an individual level owing to the confounding factors, e.g.,

- possible exposure from other sources than ingestion of water
- large inter-individual variation of proportions of urinary arsenic species
- intra-individual temporal variations in concentrations.

The significant intra-individual temporal variations are related to the short half-lives of inorganic species, i.e., approximately 8 - 12 hours (Hakala, 1995a). Consequently, a significant lag between exposure and sampling may cause bias in the results. Regardless of the confounding factors, at high U-As levels the evaluation of risk is possible.

For the purpose of risk management, it is important to know the potential source of urinary As since this cannot be determined on the basis of urine analysis. In our case in particular, additional studies are needed in order to explain the high U-As levels of some people who had not been exposed to arsenic from their drinking water. Hence, the investigation of the soil particularly in the courtyards is a clear future study need. Moreover, more data is needed on the As levels in Finnish food items. Above all, a written questionnaire seems to be too inaccurate method for the determination of actual individual As exposure prior to sampling of urine. Therefore, we recommend that more detailed personal interviews are carried out along with the sampling and an exact follow-up (approximately one week) of the consumption of drinking water and possibly also the diet prior to it. Since the concentration of As in well water (used as drinking water) may also vary in time, it is advisable to determine actual concentration simultaneously with the sampling of urine.

We also recommend to use larger study populations covering people exposed to arsenic and a reference group from an area without a local (or site-related) source of arsenic in order to minimize the effect of confounding factors. This of course, raises the costs of biomonitoring. In our case, the cost of sampling and analysis covering 40 persons was approximately 4000 €. In addition, the resources needed for the planning and preparation of the questionnaire, contacting people, mailing, etc. are to be added to the total costs.

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APPENDIX 4

Report for the Risk Assessment and Risk Management Procedure for Arsenic in the Tampere Region (RAMAS) –project

Spatial epidemiological analyses of cancer risk by arsenic level in drilled wells in the Tampere region, 1981-2000

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1 Introduction

One of the major goals of the Risk Assessment and Risk Management Procedure for Arsenic in the Tampere Region (RAMAS) – project has been to evaluate health risks due to environmental arsenic. In addition to the traditional risk analysis based on exposure modelling alone, we had the opportunity to carry out a spatial epidemiological study using high resolution cancer and population register data. Our purpose was to find out whether there is in the Tampere region any epidemiological evidence for an association between elevated cancer risks and high levels of arsenic in drilled wells. A system applying new geographic information system (GIS)-based tools suitable for such an epidemiological study has been implemented at the National Public Health Institute (KTL) Department of Environmental Health in Kuopio. This report summarizes the risk estimates for selected cancers diagnosed between 1981 and 2000 in the population cohorts living in the Tampere region in 1980 and in 1990, comparing the risks of incident cancers in areas with high and intermediate arsenic levels with the corresponding risks in reference areas during the same time period. Arsenic data from Geological Survey of Finland (GTK) was used for assessing the risk for exposure to arsenic from drilled wells (Backman *et al* 2006). The subcontracted work done by National Public Health Institute (KTL) was paid by by Geological Survey of Finland (GTK).

2 Material and methods

2.1 Rapid Inquiry Facility with the Finnish datasets

Spatial epidemiological analyses of cancer were carried out using the Rapid Inquiry Facility (RIF) – software which is an extension module for the widely applied ESRI ArcGIS software. RIF has been developed for register-based epidemiological studies by an international team lead by the Small Area Health Statistics Unit (SAHSU) at the Imperial College London, UK. KTL Environmental Epidemiology Unit has participated in this software development process during several years. The latest version of the software can now be used with high resolution cancer data (collected by the Finnish Cancer Registry) and mortality data (collected by the Statistics Finland). Two different

population cohorts are available in the Finnish RIF databases. Age- and sex classified numbers of population counts and cancer cases in the first cohort (the "Cohort of 1980") have been aggregated to small grids (250 m x 250 m) according to individual-level address information in 1980. Similarily, the location of residences in 1990 has been utilized when aggregating the second cohort (the "Cohort of 1990"). In this study, the cancer types likely most relevant to arsenic exposure were selected for analyses. The temporal end point of the analysis was set in the year 2000.

In short, RIF enables both risk mapping and risk analysis at several levels of geographic resolution (including whole Finland, hospital districts, municipalities, 2 km x 2 km grid squares, as well as 250 m x 250 m grid squares). Spatial analysis tools available in ArcGIS can be used to make relevant selection of exposed population (study area) and reference population (comparison area). GIS tools are also useful for visualization of the study design and results ("disease mapping"). There are also tools and data that would enable a refined control for confounding by e.g. by life-style or areal level factors but these have not been used in the present study due to funding limitations. However, use of such tools would increase reliability of risk estimates and thus risk assessment in relation to the environmental exposure of interest.

2.2 Calculation of the relative risk for cancer

The risk analyses were carried out with the RIF software, based on the age- and sex- adjusted comparison of new cancer cases in the selected study areas for the exposed population and the reference population. Relative risks (RR) were calculated dividing the observed number of cases by the expected number separately for men and women and for selected time periods. Expected numbers for each age group were estimated using the age-sex classified population and cancer data from the reference area. The RIF calculates also 95 % confidence intervals, but even statistically significant results need to be interpreted with caution due to possible multiple comparison problems. On the other hand, statistically non-significant risk estimates are often based on a small number of cases and may indicate real risk increases especially when systematically found for biologically plausible cancer sites. Based on the available epidemiological knowledge (IARC 2004, Silvera & Rohan 2007,Cantor & Lubin 2007, Kurttio et al. 1999), total cancer and cancers of the bladder, kidney, skin, lung, liver and prostate were selected as outcomes of the present study. Personal life-styles often induce confounding to risk estimates, but could in principle be at least partly controlled for using the socioeconomic status of each individual as a proxy measure. In this work, adjustment for socioeconomic status has not been utilized, but we aimed at reducing possible bias by a careful selection of a reference area. In the future, the RIF databases enabling socioeconomic adjustment of the risk analysis would need to be implemented on a regular basis. Such adjustment would have enhanced the reliability of the results.

2.3. Geological background

In Finland, the contact between bedrock and overburden is sharp. A geological discontinuity prevails between old crystalline bedrock and the overlying Quaternary glaciogenic sediments. Both formations support significant ground water reserves.

The bedrock in the Tampere region can be divided in three geologically distinct units based on the dominant rock types encountered in the area. The main geological subdivisions in the study area are the Central Finland Granitoid Complex (CFGC) in the north, the Tampere Belt (TB) in the centre, and the Tampere Belt (PB) in the south (Fig. 1). The division bases mainly on bedrock type but air-

borne geophysical magnetic measurement data support this division. (Backman *et al.* 2006). The CFGC mainly consists of tonalites, granites and granodiorites with minor proportions of supracrustal rocks and mafic plutonic rocks (Korsman *et al*. 1997). The TB is mainly composed of turbiditic metasedimentary rocks and felsic-intermediate arc-type metavolcanic rocks and plutonic intrusions that cut the supracrustal sequence (Ojakangas 1986, Kähkönen 1989, Kähkönen & Leveinen 1994). In the PB area, mafic and ultramafic plutons and granitoids cut the migmatitic metasedimentary rocks, sporadically containing graphite-bearing gneiss interlayers (Nironen *et al*. 2002). All the rock types encountered in the area are crystalline hard rocks.

The volcanic-sedimentary belts - (TB & PB) are enriched in gold, arsenic, silver, cobalt, copper, lithium, molybdenum, phosphorus, antimony, uranium, and zinc (Koljonen *et al*. 1992). The abundance of the sulphide-forming elements Ag, As, Cu, Mo, Sb, and Zn are above average in comparison with other sites in Finland. The median value of arsenic content in the area of granitoids (CFGC) was 1 mg/kg (n=218), in area of Tampere mica schist (TB) 2.22 mg/kg (n=128) and in Tampere migmatic area 1.9 mg/kg (n=257) (Lahtinen *et al.* 2005). The median value of arsenic content in bedrock in whole country was 0.9 mg/kg (n= 6544).

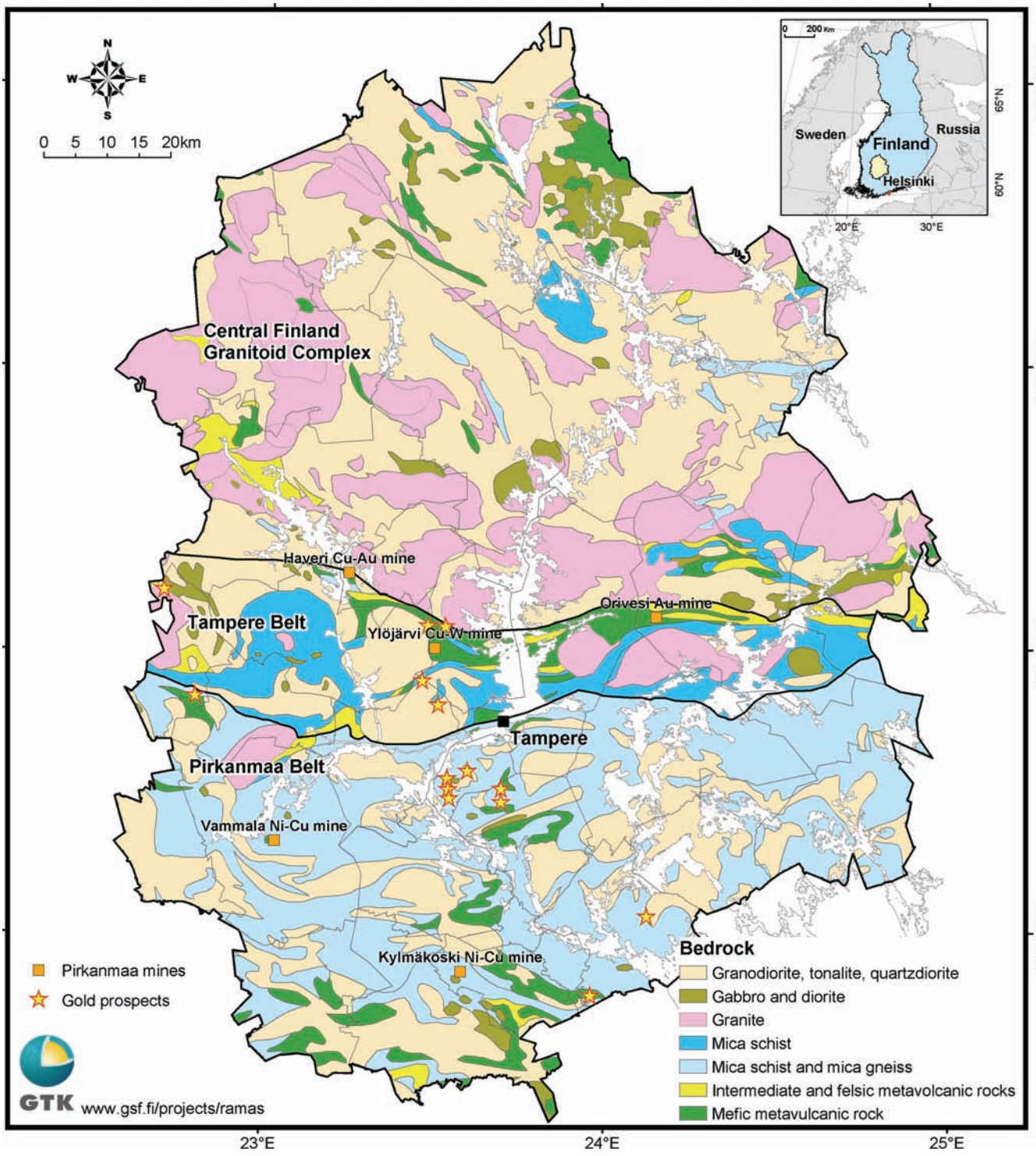

Figure 1. Bedrock in the Tampere region processed from the GTK data (Geological mapping data © Geological Survey of Finland, Base map data © National Land Survey of Finland).

2.4 Arsenic levels in the spatial epidemiological analyses

The groundwater data collected by GTK has been used in this study (Backman *et al.* 2006). Groundwater sampling has been carried out in the Tampere region several times. The first large-scale groundwater study on arsenic levels in the bedrock drilled wells was conducted in the Tampere area in 1994 (Backman *et al*. 1994). Since then, a sampling campaign has been conducted in the area almost every year. Supplementary sampling especially for the RAMAS-project was carried out in 2005. The total number of drilled bedrock wells with arsenic measurements was 1272, with arsenic levels varying from <0.05 µg/L to 2230 µg/L. The limit value of 10 µg/L recommended by the Finnish Ministry for Social Affairs and Health (2001) was exceeded in 22.5 % of the wells. The median value of arsenic concentration in bedrock groundwater in the area of granitoids (CFGC) was 0.61 µg/L (n=133), in area of Tampere mica schist (TB) 5.50 µg/L (n=588) and in Tampere migmatic area 1.6 µg/L (n=551) (Backman *et al.* 2006).

The arsenic contents in till deposits varied from <0.05 to 9 280 mg/kg (Backman *et al.* 2006). The highest arsenic contents were in deeper part of the till in areas where the bedrock arsenic was high, also.

The regional geological arsenic level map was based on the three data sources from arsenic contents in bedrock, in till and in groundwater. For this study, the arsenic level data was aggregated into 250 m x 250 m grid cells. Figure 2 represents the basic idea of the calculations of the arsenic level in each grid cell. Groundwater data was classified into four classes according to the limit values used for safe drinking water (WHO 1993, STM 2001) (Fig. 2, A). Till and bedrock data were also classified into four classes and the used limit values based on the legislation of the decree on the assessment of soil contamination and remediation needs (VNa 2007/214) (Fig. 2, A). Each grid cell contains various arsenic values from one to three different geological data, therefore, one grid cell has three numbers between –1 and 3 (Fig. 2, B). These grid data were then classified into three different classes: 0 = low level exposure, 1 = intermediate level exposure, 2 = high level exposure and -1 = grid that has no available data. (Fig. 2, C) The arsenic level map is shown in Figure 3.

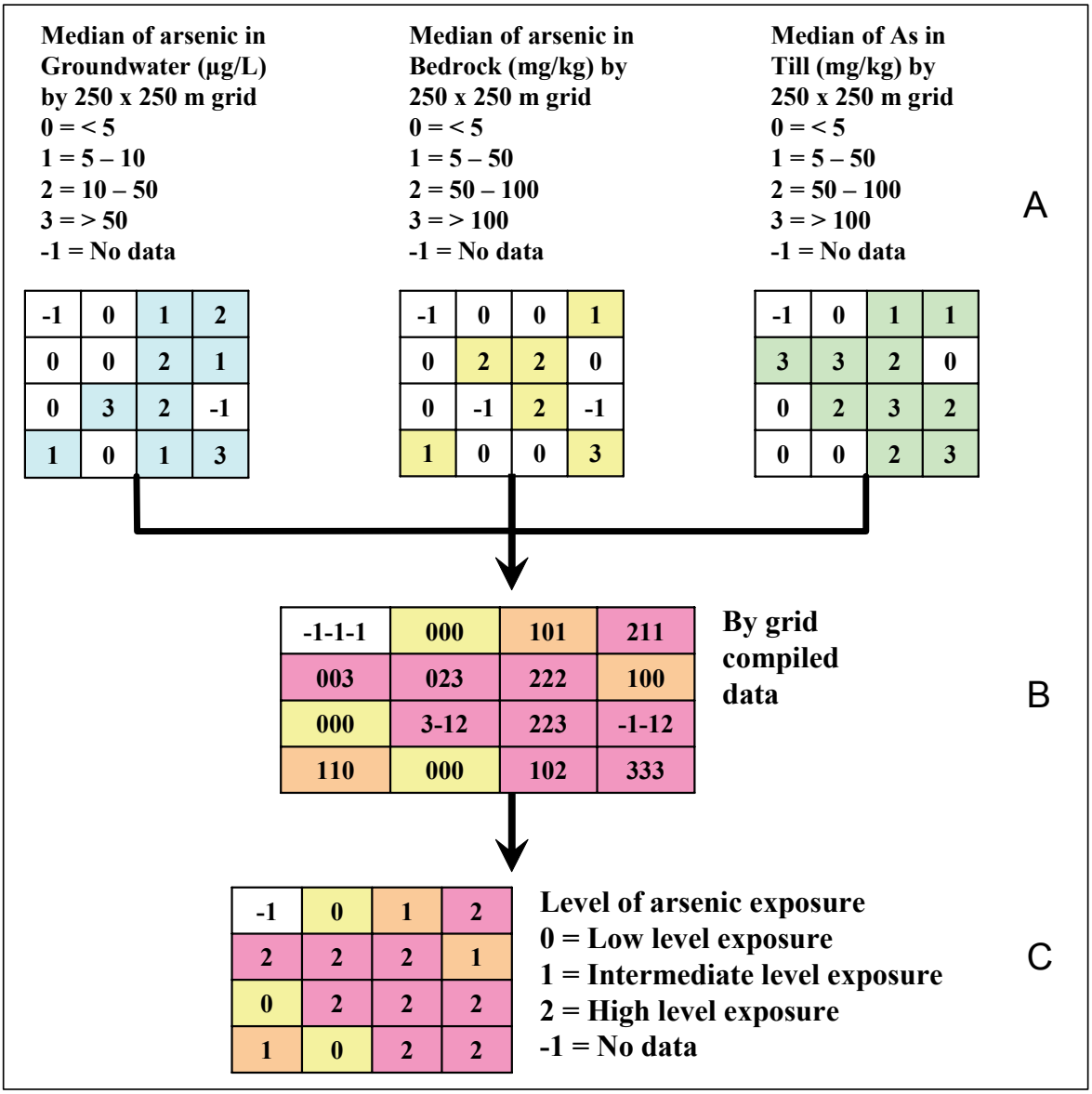

Figure 2 Method of data processing and classification for arsenic level map.

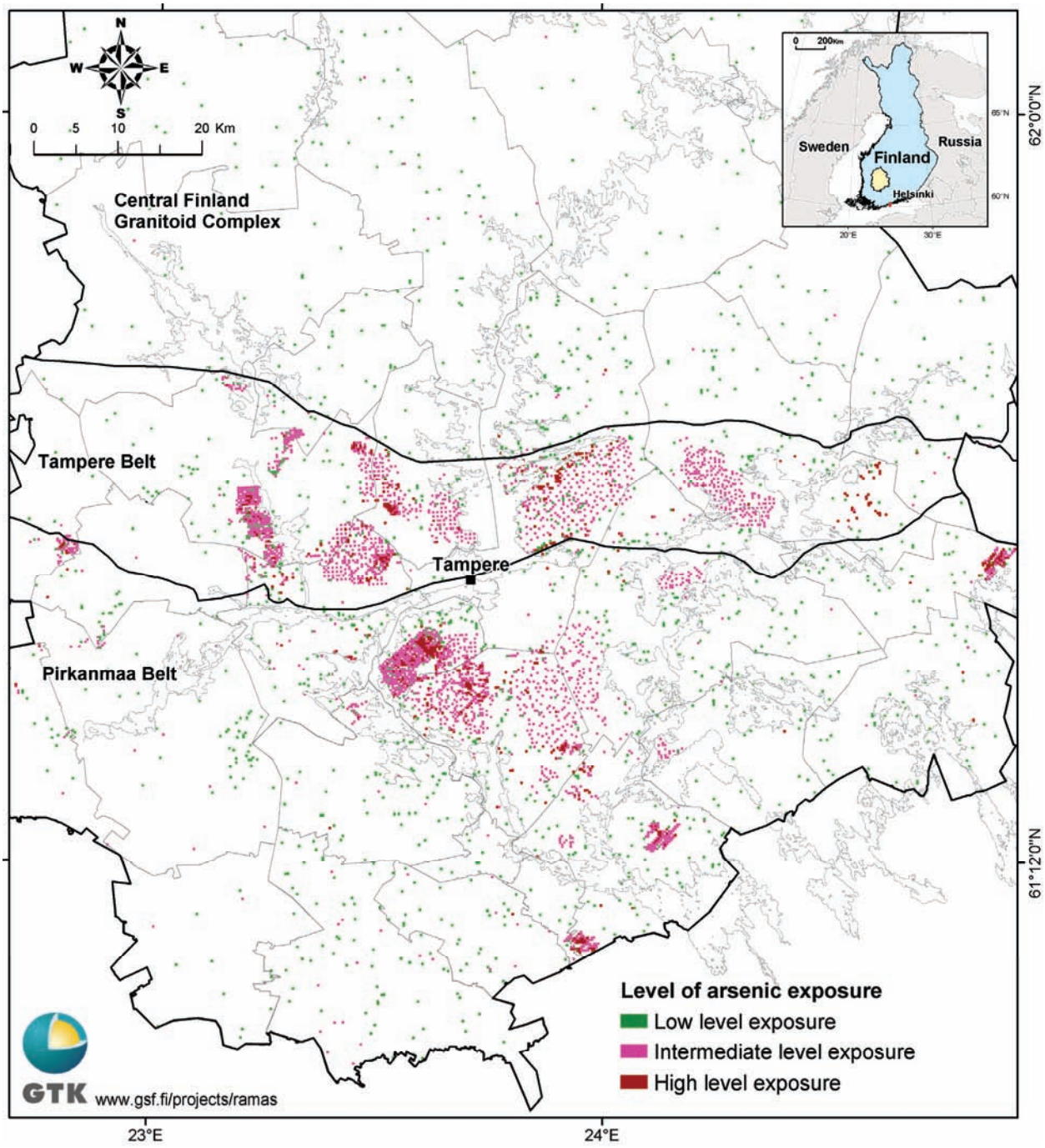

Figure 3. Map of arsenic exposure in groundwater, bedrock and till in the Tampere region

The grid level analyses were run for the populated 250 m x 250 m grids outside the densely populated areas (defined according to the situation in 1980). The spatial data indicating the densely populated areas in 1980 was taken from the Monitoring System of Spatial Structure (MOST), which has been developed by the Finnish Environment Institute. MOST is used to define urban areas and, in the present study, to exclude the urban areas (where people don't use private wells) from statistical analyses of cancer risk. The remaining rural population was classified by their possible exposure to arsenic (Fig. 4) according to the method described above. The number of people in the cohort of 1980 in the 250 m x 250 m level data was rather small for each exposure class (Table 1).

The reference population (N=17,830) covering the whole Tampere rural area was selected by the 2 km x 2 km resolution grids where only low arsenic levels were measured.

Table 1. Population in rural 250 m x 250 m grids where arsenic level data was available.

	Population 1980				Population 2005			
As level	**Population**	**Cum. pop**	**%**	**cum %**	**Population**	**Cum. pop**	**%**	**cum %**
low	2424	2424	55.65	55.65	3802	3802	46.26	46.26
intermediate	1219	3643	27.98	83.63	3185	6987	38.75	85.01
high	713	4356	16.37	100.00	1232	8219	14.99	100.00
Total	4356				8219			

For municipality level analyses, the selection of risk and reference areas were defined on the basis of the same arsenic data that we used in the grid-based analysis and additional information available on the bedrock geology in the Tampere area. The municipality of Tampere was excluded to avoid the dominance of urban environment and the effect of life style on the occurrence of cancer. The risk area and reference area in Northern Tampere are visualized in figure 5. In 1980, more than 120,000 people lived in the municipalities with high levels of arsenic. However, in practice only a part of these were exposed to arsenic through well water whereas a part of residents in the reference area may also have been exposed. This kind of exposure misclassification will likely bias the risk estimates towards unity, in other words increase the possibility of false negative results.

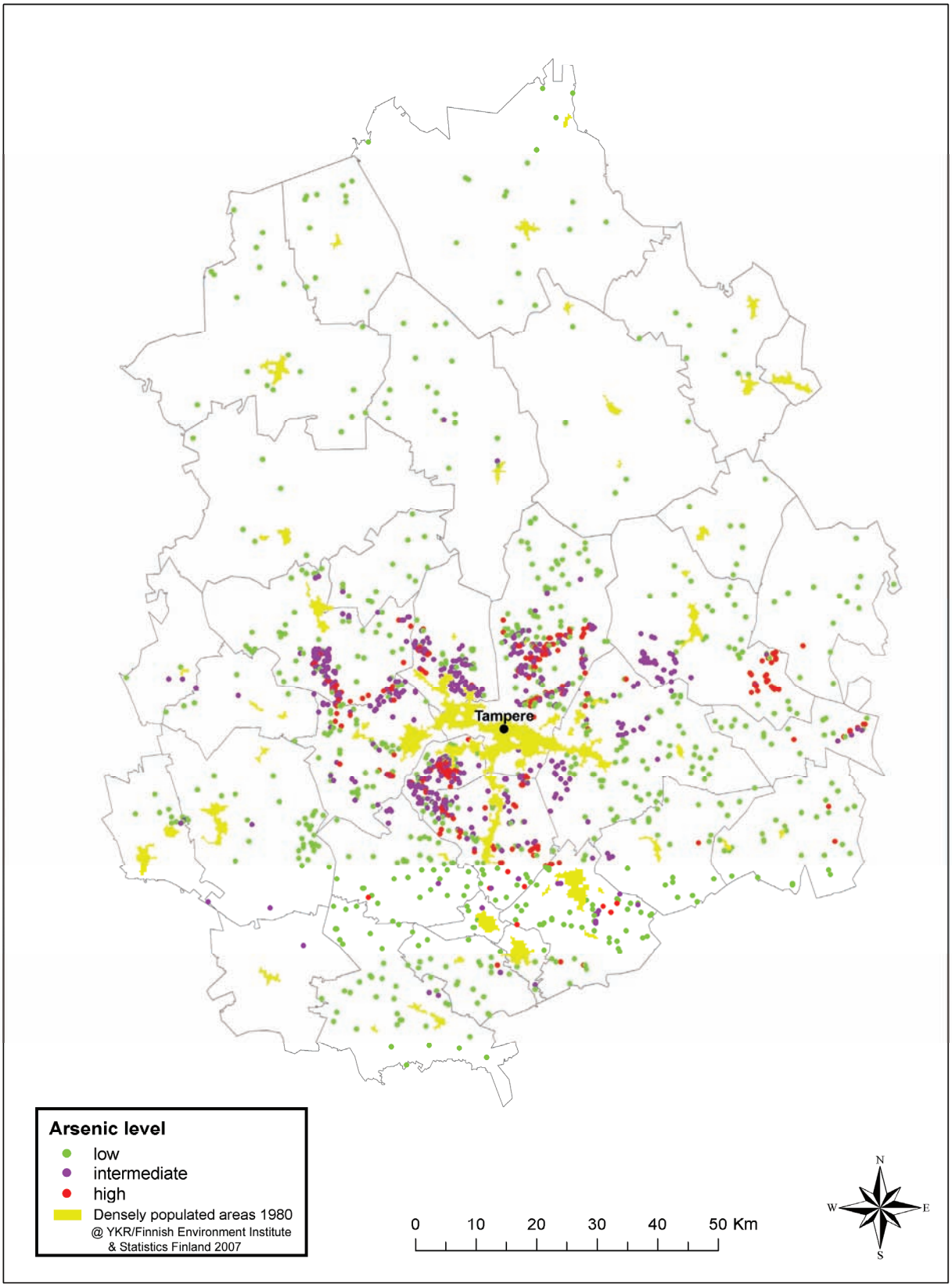

Figure 4. Spatial variation of the of arsenic exposure level assessed on the basis of arsenic concentration in ground water, bedrock or soil. Data inside the densely populated areas were excluded.

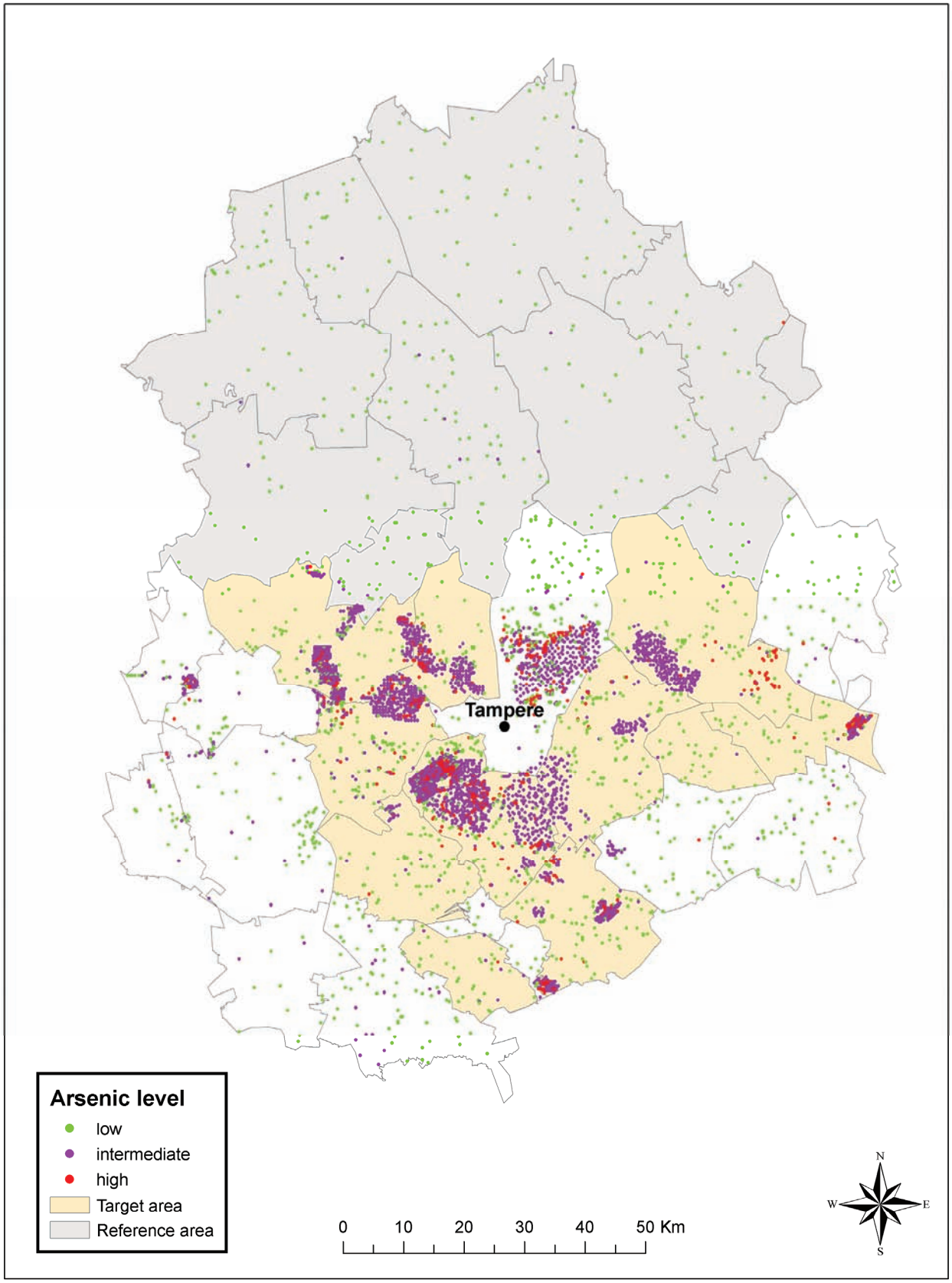

Figure 5. The municipalities with higher arsenic levels ("target area"), the municipalities with regular arsenic levels ("reference area"), and measurement point data on arsenic risk in the Tampere region. Identification of risk areas was based on expert elicitation, taking into account the information on arsenic concentrations and knowledge of bedrock geology.

3 Results and discussion

3.1 Municipality level analyses

The risk of total cancer in the Tampere region was at the level expected based on the national rates (data not shown). The risk of non-melanoma of the skin was elevated by at least 15 % for both time periods (1981-1990 and 1991-2000) and both cohorts. On the contrary, the risk estimates for cancers of the bladder, lung and liver cancers were clearly lower compared to the whole country. As there are well-known geographical differences in cancer incidence rates due to (geographical) differences in life-styles and - to a much lesser extent - in diagnostic procedures, the observed differences in cancer risk between the Tampere region and the whole country can be taken to confirm the importance of the selection of control area. This was addressed by using local reference areas in the cancer risk analyses.

The municipality level analyses within the Tampere region (Table 2) showed systematically elevated and statistically significant risks for almost all of the a priori selected cancer types. The risk of total cancer was also slightly elevated (RR 1.06, 95% CI 1.04-1.09). The highest relative risks were found for liver cancer (RR 1.52, CI 1.19-1.92). The risks for the cancers of the bladder (1.19, 1.06-1.33), kidney (1.20, 1.08-1.34), lung (1.12, 1.05-1.19), and prostate (1.13, 1.07-1.20) were all statistically significantly elevated. The risks for melanoma (1.06, 0.94-1.20) and non-melanoma (1.02, 0.92-1.12) of the skin were close to the risk in the reference population.

Some of the increases observed in the municipality level analyses may be due to confounding for instance by smoking. Also aggregation bias (here: use of grid-cell level exposure assessment) may in theory either dilute or artificially create risks. However, there is little pragmatic evidence to support or disvalidate the importance of the aggregation bias. In any case, the fact that the higher risk ratios were observed at municipality level represented the cancer types selected *a priori* on the basis of their known or suspected association with exposure to arsenic, increases the possibility of real carcinogenic effects.

3.2 Small area level analyses

To investigate the relationship between cancer risk and possible exposure to arsenic from drilled wells, we conducted a series of analyses based on the geological, measured arsenic concentrations assigned to 250 m x 250 m grids and classified into low, intermediate and high arsenic levels.

Firstly, the effect of arsenic level was analysed using the whole Finland as a reference area (data not shown). Statistically significant risks for the cancers of kidney and prostate in men and non-melanoma in women were found among the most exposed group. The risks for the cancers of kidney, prostate and skin among the reference population (in 2 km x 2 km grids) in Tampere compared to whole Finland were also found to be slightly elevated (significantly only for prostate cancer). The advantage of this analysis is that expected numbers are based on a large population, which makes the relative risks more stable and the confidence intervals relatively narrow. The disadvantage is that the exposure level for the reference population is unknown.

Secondly, analysis for the 1980 cohort was conducted (Table 3) using perhaps the most relevant reference population – namely rural population aggregated to 2 km x 2 km grids where only very low arsenic levels were measured. When looking at cancer risks combining data for both genders, the most exposed (high level) group showed no statistically significantly increased risks, while statistically non-significant excesses were observed for cancers of the kidney (RR 1.97, 95% CI

0.72-4.29), liver (1.94, 0.05-10.8) and non-melanoma (2.02, 0.74-4.4) of the skin. In men, the risk for cancers of the kidney (3.26, 1.06-7.6) and prostate (2.00, 1.20-3.12) were statistically significantly elevated whereas a non-significant increase was observed also liver cancer (8.81, 0.22-49.1). In women, we found a statistically significantly increased risk for non-melanoma of the skin (3.72, 1.36-8.09). Finally, the analysis was conducted according to the residence in year 1990 (Table 4). Similarly to the results for the 1980 cohort, the RRs for the 1990 cohort tended to be highest (but not significant) with the highest exposure for the cancers of kidney, prostate and non-melanoma of the skin.

Table 2. Cancer risk from 1980 to 2000 among people living in 1980 in municipalities with high arsenic levels. Pirkanmaa region municipalities with low arsenic levels were used as reference.

Total cancer		Obs	RR	95 % CI		**Liver cancer**		Obs	RR	95 % CI	
Male	1981-1990	1921	**1.04**	0.99	1.09	Male	1981-1990	17	**1.79**	1.04	2.86
	1991-2000	2656	**1.15**	1.11	1.19		1991-2000	15	**1.00**	0.56	1.65
	1981-2000	4577	**1.10**	1.07	1.13		1981-2000	32	**1.31**	0.89	1.85
Female	1981-1990	2034	**1.00**	0.96	1.04	Female	1981-1990	20	**2.88**	1.76	4.44
	1991-2000	2611	**1.06**	1.02	1.10		1991-2000	18	**1.24**	0.73	1.95
	1981-2000	4645	**1.03**	1.00	1.06		1981-2000	38	**1.77**	1.25	2.43
Both	1981-1990	3955	**1.02**	0.99	1.05	Both	1981-1990	37	**2.25**	1.58	3.10
	1991-2000	5267	**1.10**	1.07	1.13		1991-2000	33	**1.12**	0.77	1.57
	1981-2000	9222	**1.06**	1.04	1.09		1981-2000	70	**1.52**	1.19	1.92
Bladder cancer						**Melanoma**					
Male	1981-1990	101	0.90	0.74	1.10	Male	1981-1990	60	**1.18**	0.90	1.51
	1991-2000	121	**1.28**	1.07	1.53		1991-2000	76	**1.12**	0.89	1.41
	1981-2000	222	**1.08**	0.94	1.23		1981-2000	136	**1.15**	0.97	1.36
Female	1981-1990	40	**1.73**	1.23	2.35	Female	1981-1990	64	**1.03**	0.79	1.31
	1991-2000	42	**1.60**	1.16	2.17		1991-2000	66	0.94	0.73	1.20
	1981-2000	82	**1.66**	1.32	2.06		1981-2000	130	0.98	0.83	1.17
Both	1981-1990	141	**1.05**	0.89	1.23	Both	1981-1990	124	**1.09**	0.92	1.30
	1991-2000	163	**1.35**	1.16	1.57		1991-2000	142	**1.03**	0.88	1.22
	1981-2000	304	**1.19**	1.06	1.33		1981-2000	266	**1.06**	0.94	1.20
Kidney cancer						**Nonmelanoma**					
Male	1981-1990	71	0.95	0.74	1.20	Male	1981-1990	60	0.81	0.62	1.04
	1991-2000	128	**1.68**	1.41	2.00		1991-2000	121	**1.30**	1.09	1.56
	1981-2000	199	**1.32**	1.15	1.52		1981-2000	181	**1.08**	0.94	1.25
Female	1981-1990	64	**1.13**	0.87	1.44	Female	1981-1990	77	0.86	0.68	1.08
	1991-2000	65	0.98	0.76	1.25		1991-2000	129	**1.03**	0.87	1.23
	1981-2000	129	**1.05**	0.88	1.25		1981-2000	206	0.96	0.84	1.10
Both	1981-1990	135	**1.03**	0.87	1.22	Both	1981-1990	137	0.84	0.71	0.99
	1991-2000	193	**1.35**	1.18	1.56		1991-2000	250	**1.15**	1.02	1.30
	1981-2000	328	**1.20**	1.08	1.34		1981-2000	387	**1.02**	0.92	1.12
Lung cancer						**Prostate cancer**					
Male	1981-1990	466	**1.15**	1.05	1.26	Male	1981-1990	294	**1.04**	0.92	1.16
	1991-2000	399	**1.15**	1.04	1.27		1991-2000	808	**1.17**	1.09	1.26
	1981-2000	865	**1.15**	1.07	1.23		1981-2000	1102	**1.13**	1.07	1.20
Female	1981-1990	47	0.68	0.50	0.91						
	1991-2000	94	**1.17**	0.95	1.44						
	1981-2000	141	0.95	0.80	1.12						
Both	1981-1990	513	**1.08**	0.99	1.18						
	1991-2000	493	**1.16**	1.06	1.26						
	1981-2000	1006	**1.12**	1.05	1.19						

Obs: observed number of cases, RR: relative risk (denotes here age- and sex standardized incidence ratios), CI: confidence intervals

Table 3. Cancer risk from 1981 to 2000 among people living in Pirkanmaa region in 1980, by arsenic level defined for 250 m * 250 m grid cells. The 2 km * 2 km cells with not elevated arsenic levels were used as reference.

		Exposure categories by Arsenic level											
		intermediate				high				intermediate or high			
Total cancer		Obs	RR	95 % CI		Obs	RR	95 % CI		Obs	RR	95 % CI	
Male	1981-1990	36	**1.08**	0.76	1.5	22	**1.30**	0.82	1.97	58	**1.16**	0.88	1.5
	1991-2000	34	0.91	0.63	1.28	27	**1.28**	0.84	1.87	61	**1.05**	0.8	1.34
	1981-2000	70	0.99	0.77	1.26	49	**1.29**	0.95	1.71	119	**1.10**	0.92	1.31
Female	1981-1990	35	**1.07**	0.74	1.49	12	0.66	0.34	1.16	47	0.92	0.68	1.23
	1991-2000	34	0.96	0.67	1.34	23	**1.18**	0.75	1.77	57	**1.04**	0.79	1.35
	1981-2000	69	**1.01**	0.79	1.28	35	0.93	0.65	1.29	104	0.98	0.81	1.19
Both	1981-1990	71	**1.08**	0.84	1.36	34	0.97	0.67	1.36	105	**1.04**	0.86	1.26
	1991-2000	68	0.94	0.73	1.19	50	**1.23**	0.91	1.63	118	**1.04**	0.87	1.25
	1981-2000	139	**1.00**	0.85	1.18	84	**1.11**	0.89	1.38	223	**1.04**	0.91	1.19
Bladder cancer													
Male	1981-1990	1	0.53	0.01	2.96	0	0.00	0	3.97	1	0.36	0.01	1.98
	1991-2000	0	0.00	0	2.22	1	**1.02**	0.03	5.68	1	0.38	0.01	2.11
	1981-2000	1	0.28	0.01	1.57	1	0.52	0.01	2.92	2	0.37	0.04	1.33
Female	1981-1990	1	**2.06**	0.05	11.5	0	0.00	0	11.2	1	**1.22**	0.03	6.82
	1991-2000	0	0.00	0	4.11	0	0.00	0	7.97	0	0.00	0	2.71
	1981-2000	1	0.72	0.02	4.03	0	0.00	0	4.65	1	0.46	0.01	2.56
Both	1981-1990	2	0.84	0.1	3.05	0	0.00	0	2.93	2	0.55	0.07	1.99
	1991-2000	0	0.00	0	1.44	1	0.69	0.02	3.86	1	0.25	0.01	1.39
	1981-2000	2	0.41	0.05	1.47	1	0.37	0.01	2.06	3	0.39	0.08	1.15
Kidney cancer													
Male	1981-1990	2	**1.29**	0.16	4.66	1	**1.10**	0.03	6.13	3	**1.22**	0.25	3.56
	1991-2000	3	**2.53**	0.52	7.39	4	**6.38**	1.74	16.3	7	**3.86**	1.55	7.95
	1981-2000	5	**1.83**	0.59	4.26	5	**3.26**	1.06	7.6	10	**2.34**	1.12	4.3
Female	1981-1990	0	0.00	0	3.42	1	**1.55**	0.04	8.63	1	0.58	0.01	3.23
	1991-2000	0	0.00	0	2.4	0	0.00	0	4.29	0	0.00	0	1.54
	1981-2000	0	0.00	0	1.41	1	0.66	0.02	3.7	1	0.24	0.01	1.35
Both	1981-1990	2	0.76	0.09	2.75	2	**1.29**	0.16	4.65	4	0.96	0.26	2.45
	1991-2000	3	**1.10**	0.23	3.22	4	**2.69**	0.73	6.89	7	**1.66**	0.67	3.42
	1981-2000	5	0.93	0.3	2.18	6	**1.97**	0.72	4.29	11	**1.31**	0.65	2.34
Lung cancer													
Male	1981-1990	9	**1.14**	0.52	2.17	4	0.93	0.25	2.39	13	**1.07**	0.57	1.83
	1991-2000	5	0.86	0.28	2.02	3	0.88	0.18	2.58	8	0.87	0.38	1.72
	1981-2000	14	**1.02**	0.56	1.72	7	0.91	0.37	1.88	21	0.98	0.61	1.5
Female	1981-1990	0	0.00	0	3.84	1	**1.90**	0.05	10.6	1	0.67	0.02	3.75
	1991-2000	1	**1.19**	0.03	6.61	0	0.00	0	8.58	1	0.79	0.02	4.37
	1981-2000	1	0.55	0.01	3.09	1	**1.05**	0.03	5.83	2	0.72	0.09	2.62
Both	1981-1990	9	**1.02**	0.47	1.93	5	**1.04**	0.34	2.42	14	**1.03**	0.56	1.72
	1991-2000	6	0.91	0.33	1.97	3	0.78	0.16	2.29	9	0.86	0.39	1.63
	1981-2000	15	0.97	0.54	1.6	8	0.93	0.4	1.83	23	0.95	0.6	1.43

Obs: observed number of cases, RR: relative risk (denotes here age- and sex standardized incidence ratios), CI: confidence intervals

Table 3 (continued). Cancer risk from 1981 to 2000 among people living in Pirkanmaa region in 1980, by arsenic level defined for 250 m * 250 m grid cells. The 2 km * 2 km cells with not elevated arsenic levels were used as reference.

		Exposure categories by Arsenic level											
		intermediate				high				intermediate or high			
Liver cancer		Obs	RR	95 % CI		Obs	RR	95 % CI		Obs	RR	95 % CI	
Male	1981-1990	0	0.00	0	20.5	1	**14.04**	0.35	78.2	1	**3.99**	0.1	22.2
	1991-2000	0	0.00	0	55.7	0	0.00	0	87.1	0	0.00	0	34
	1981-2000	0	0.00	0	15	1	**8.81**	0.22	49.1	1	**2.78**	0.07	15.5
Female	1981-1990	0	0.00	0	8.27	0	0.00	0	14.1	0	0.00	0	5.22
	1991-2000	0	0.00	0	13.3	0	0.00	0	26.4	0	0.00	0	8.84
	1981-2000	0	0.00	0	5.1	0	0.00	0	9.21	0	0.00	0	3.28
Both	1981-1990	0	0.00	0	5.9	1	**3.01**	0.08	16.8	1	**1.04**	0.03	5.82
	1991-2000	0	0.00	0	10.7	0	0.00	0	20.2	0	0.00	0	7.02
	1981-2000	0	0.00	0	3.81	1	**1.94**	0.05	10.8	1	0.67	0.02	3.76
Melanoma													
Male	1981-1990	0	0.00	0	5.08	1	**2.70**	0.07	15	1	0.91	0.02	5.08
	1991-2000	1	0.72	0.02	4.01	0	0.00	0	4.65	1	0.46	0.01	2.55
	1981-2000	1	0.47	0.01	2.63	1	0.86	0.02	4.79	2	0.61	0.07	2.2
Female	1981-1990	2	**2.22**	0.27	8.02	0	0.00	0	6.68	2	**1.38**	0.17	4.97
	1991-2000	2	**1.93**	0.23	6.98	0	0.00	0	6.08	2	**1.22**	0.15	4.4
	1981-2000	4	**2.07**	0.56	5.29	0	0.00	0	3.18	4	**1.29**	0.35	3.31
Both	1981-1990	2	**1.23**	0.15	4.44	1	**1.08**	0.03	6.04	3	**1.18**	0.24	3.44
	1991-2000	3	**1.24**	0.26	3.61	0	0.00	0	2.63	3	0.78	0.16	2.29
	1981-2000	5	**1.23**	0.4	2.88	1	0.43	0.01	2.4	6	0.94	0.35	2.05
Nonmelanoma													
Male	1981-1990	1	0.82	0.02	4.59	0	0.00	0	7.11	1	0.58	0.01	3.21
	1991-2000	1	0.69	0.02	3.85	0	0.00	0	4.42	1	0.44	0.01	2.44
	1981-2000	2	0.75	0.09	2.71	0	0.00	0	2.73	2	0.50	0.06	1.8
Female	1981-1990	3	**2.26**	0.47	6.6	1	**1.37**	0.03	7.65	4	**1.94**	0.53	4.98
	1991-2000	3	**1.73**	0.36	5.07	5	**5.65**	1.83	13.2	8	**3.06**	1.32	6.03
	1981-2000	6	**1.96**	0.72	4.27	6	**3.72**	1.36	8.09	12	**2.57**	1.33	4.49
Both	1981-1990	4	**1.57**	0.43	4.03	1	0.80	0.02	4.47	5	**1.32**	0.43	3.08
	1991-2000	4	**1.26**	0.34	3.22	5	**2.91**	0.94	6.79	9	**1.84**	0.84	3.49
	1981-2000	8	**1.40**	0.6	2.75	6	**2.02**	0.74	4.4	14	**1.61**	0.88	2.7
Prostate cancer													
Male	1981-1990	8	**1.47**	0.63	2.89	7	**2.62**	1.05	5.41	15	**1.85**	1.03	3.05
	1991-2000	10	0.83	0.4	1.52	12	**1.76**	0.91	3.07	22	**1.16**	0.73	1.76
	1981-2000	18	**1.03**	0.61	1.62	19	**2.00**	1.2	3.12	37	**1.37**	0.96	1.89
Female	1981-1990	0	0.00	0	0	0	0.00	0	0	0	0.00	0	0
	1991-2000	0	0.00	0	0	0	0.00	0	0	0	0.00	0	0
	1981-2000	0	0.00	0	0	0	0.00	0	0	0	0.00	0	0
Both	1981-1990	8	**1.47**	0.63	2.89	7	**2.62**	1.05	5.41	15	**1.85**	1.03	3.05
	1991-2000	10	0.83	0.4	1.52	12	**1.76**	0.91	3.07	22	**1.16**	0.73	1.76
	1981-2000	18	**1.03**	0.61	1.62	19	**2.00**	1.2	3.12	37	**1.37**	0.96	1.89

Obs: observed number of cases, RR: relative risk (denotes here age- and sex standardized incidence ratios), CI: confidence intervals

Table 4. Cancer risk from 1991 to 2000 among people living in rural Tampere Region in 1990, by arsenic level defined for 250 m * 250 m grid cells. The 2 km * 2 km cells with not elevated arsenic levels were used as reference.

	Exposure categories by Arsenic level											
	intermediate				high				intermediate or high			
Total cancer	Obs	**RR**	lo95	up95	Obs	**RR**	lo95	up95	Obs	**RR**	lo95	up95
Male	37	0.92	0.65	1.27	30	**1.15**	0.77	1.64	67	**1.01**	0.78	1.29
Female	33	0.91	0.63	1.28	25	**1.13**	0.73	1.67	58	1.00	0.76	1.29
Both	70	0.92	0.72	1.16	55	**1.14**	0.86	1.48	125	**1.00**	0.84	1.2
Bladder cancer												
Male	1	0.66	0.02	3.67	1	0.89	0.02	4.95	2	0.76	0.09	2.73
Female	0	0.00	0	4.96	0	0.00	0	7.07	0	0.00	0	2.91
Both	1	0.44	0.01	2.46	1	0.61	0.02	3.38	2	0.51	0.06	1.85
Kidney cancer												
Male	3	**2.67**	0.55	7.79	3	**3.86**	0.8	11.3	6	**3.15**	1.16	6.87
Female	0	0.00	0	2.88	0	0.00	0	4.14	0	0.00	0	1.7
Both	3	**1.25**	0.26	3.64	3	**1.80**	0.37	5.26	6	**1.47**	0.54	3.21
Lung cancer												
Male	4	0.56	0.15	1.43	4	0.82	0.22	2.1	8	0.67	0.29	1.31
Female	1	**1.11**	0.03	6.16	0	0.00	0	7.3	1	0.71	0.02	3.95
Both	5	0.62	0.2	1.45	4	0.74	0.2	1.91	9	0.67	0.31	1.27
Liver cancer												
Male	0	0.00	0	20.9	1	**11.78**	0.29	65.6	1	**3.83**	0.1	21.3
Female	0	0.00	0	14.2	0	0.00	0	20.6	0	0.00	0	8.41
Both	0	0.00	0	8.47	1	**3.78**	0.09	21.1	1	**1.43**	0.04	7.96
Melanoma												
Male	2	**1.50**	0.18	5.41	0	0.00	0	4.25	2	0.91	0.11	3.28
Female	0	0.00	0	3.38	0	0.00	0	5.69	0	0.00	0	2.12
Both	2	0.82	0.1	2.98	0	0.00	0	2.43	2	0.51	0.06	1.83
Nonmelanoma												
Male	1	0.66	0.02	3.66	0	0.00	0	3.72	1	0.40	0.01	2.22
Female	0	0.00	0	1.92	3	**2.52**	0.52	7.36	3	0.96	0.2	2.82
Both	1	0.29	0.01	1.62	3	**1.38**	0.28	4.02	4	0.71	0.19	1.82
Prostate												
Male	13	**1.10**	0.59	1.88	10	**1.29**	0.62	2.38	23	**1.18**	0.75	1.76
Female	0	0.00	0	**0**	0	0.00	0	**0**	0	0.00	0	**0**
Both	13	**1.10**	0.59	1.88	10	**1.29**	0.62	2.38	23	**1.18**	0.75	1.76

Obs: observed number of cases, RR: relative risk (denotes here age- and sex standardized incidence ratios), CI: confidence intervals

4 Conclusions

We conducted these analyses of cancer risk by arsenic level in drinking water in the Tampere region, 1981-2000, using a Rapid Inquiry System that commands nationwide data on population, cancer and causes of death in Finland. The results suggest an increased risk for cancers of the kidney and prostate and possibly also for cancers of the liver and for non-melanoma of the skin. All these cancers have at least to some extent been previously associated with arsenic exposure. Nevertheless, all the results need to be interpreted with caution due to several sources of uncertainty that may bias the results. In future, to confirm these results, one should seek to use even more accurate interpolated or measured data about the arsenic levels in wells. Equally importantly, socioeconomic and other background data will be needed to control for the effects of cancer causing life-style factors. Finally, a retrospective cohort study with information about residential histories and person and life-style characteristics could be expected to give more definitive answers.

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The project will produce a number of Technical Reports. The following reports have been published:

1. Natural Occurrence of Arsenic in the Pirkanmaa Region in Finland
2. Anthropogenic Arsenic Sources in the Pirkanmaa Region in Finland
3. Arseenista aiheutuvien riskien hallinta Pirkanmaalla – Esiselvitys ohjaus keinoista ja teknisistä menetelmistä riskien vähentämiseksi (Management of arsenic risks in the Pirkanmaa region – Survey of available risk management instruments and tools)
4. Arsenic and other elements in agro-ecosystems in Finland and particularly in the Pirkanmaa region
5. A transport model of arsenic for surface waters - an application in Finland
6. Arsenic Ecotoxicity in Soils
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