BRIEF PAPER

Remediation of Sites Contaminated by Arsenic

Data to Estimate the Government Cost for Risk Reductions

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Contents

1. Introduction 3

2. What Defines a Contaminated Site and What Legislative Framework is at Hand? 3

3. Risk Assessment at Contaminated Sites 5
From contaminant to health effect 5
Risk assessment – the environmental medicine approach versus the Swedish EPA's approach 6

4. The Need for Economic Valuation of Risk Reduction at Contaminated Sites 9
Arsenic contaminated sites 10
Arsenic and associated health risks 10

5. Presentation of Data 13
Site Characteristics 13
Concentrations 14
Exposed populations 16
Data for Quantification of Health Risk 18
Remediation costs 20

6. Concluding remarks 23

Appendix A 24
Quantification of extra cancer risk posed by arsenic contaminated air on the basis of the concentration in soil 24

1. Introduction

In Sweden, 83 000 sites are potentially contaminated due to previous industrial activities. According to the Swedish Environmental Protection Agency (EPA), the administrator of the governmental funds for remediation, approximately 1 500 of these sites contain contaminant concentrations that could seriously harm human health and the environment (Swedish EPA, 2007a). Based on the sites' average remediation cost of SEK 40 million (Swedish EPA, 2007b), the approximated cost to mitigate the potential risks at the most harmful sites is estimated at SEK 60 billions.[1] Currently, about 10 percent (SEK 455 023 000) of the national funds for environmental protection is spent on site remediation (Gov. Bill 2007/08:1). In addition, an annual amount of SEK 200 million is borne by others than the Swedish EPA.[2]

Reducing risk to human health and the environment is the principal rationale behind site clean-ups. Yet, the high costs associated with risk reductions on contaminated sites raise questions about the actual risks at hand, how they are assessed and about for whom they are reduced. This memo addresses these questions by presenting data from governmentally funded arsenic remediation projects. The information will be used in a forthcoming analysis of governmental preferences regarding health risk reductions at contaminated sites.[3]

2. What Defines a Contaminated Site and What Legislative Framework is at Hand?

A contaminated site refers to a polluted land or water area that may cause harm or detriment to human health or the environment (Gov. Bill 1998:808, Chapter 10, 1 §). The Swedish Environmental Code is the legal framework behind site remediation activities in Sweden. Chapter 10 contains all legal definitions, liabilities, and exceptions involved in a remediation process.

[1] 60 billions = 60 000 millions.

[2] These include an association of oil companies, SPIMFAB (about SEK 100 million/year), the Swedish Armed Forces (about SEK 50 million/year), and the Swedish Rail Administration (about SEK 50 million/year).

[3] At sites with arsenic as the primary pollutant, the remediation objective (i.e. the guideline value) is based on risks for human health (Swedish EPA, 2008a).

In addition to the legal framework, one of the 16 environmental objectives enacted by the Swedish parliament addresses contaminated sites. The environmental objectives are not legally binding, yet they work as benchmarks for the national environmental policy, with the ultimate aim to solve the major environmental problems within one generation (i.e. to 2020). One of the most challenging objectives, 'A Non-Toxic Environment', holds that man-made or extracted compounds and metals threatening human health or biological diversity should be eliminated (Environmental Objectives Portal, 2006). To concretise and set the time frame for objective fulfilment, several interim targets have been announced. Two of the interim targets linked to 'A Non-Toxic Environment' regard remediation of contaminated sites. In sum, they state that highest priority should be given to the sites that pose the highest risks to human health and the environment.[4] A contaminated site is regarded as risky if humans and the environment are exposed to it or if it is a source of contaminant migration subsequently leading to human and environmental exposure (Swedish EPA, 2007c).

As the targets of government spending on remediation are human and environmental risk reductions, it is crucial to understand how these risks are assessed. Hence, a short overview of the prevailing methods for risk assessment of contaminated sites in Sweden is provided in the next section.

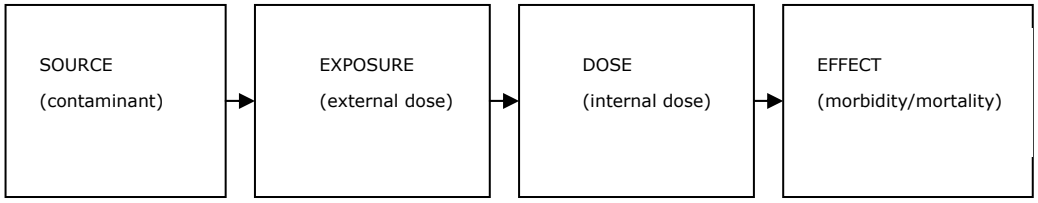
[4] Interim Target 6: Studies will have been carried out and, where necessary, appropriate action will have been taken by the end of 2010 at all contaminated sites that pose an acute risk on direct exposure, and at contaminated sites that threaten important water sources or valuable natural environments, today or in the near future. Interim Target 7: From 2005 to 2010, measures will be implemented at a sufficiently large portion of the prioritised contaminated sites to ensure that the environmental problem as a whole can be solved by 2050 at the latest.

3. Risk Assessment at Contaminated Sites[5]

From contaminant to health effect

Exposure is the scientific term for human contact with chemical substances or the environment. Chemical substances can enter the human body by inhalation, ingestion of food or water, or through the skin or mucous membranes. As illustrated in Figure 1, *exposure* is the external dose (concentration in the environment), whereas the *dose* refers to the concentration absorbed by the human body.

Figure 1 From contaminant to effect.



Source: Liljelind & Barregård (2008).

Assessment of human exposure to chemical substances from contaminated sites involves looking at four sources: soil, groundwater, surface water and sediments. Contamination can appear as a 'hot spot', i.e. high concentrations within a confined area, or as lower concentrations with a wide spread.

The level of exposure (i.e. external dose) depends on contaminant characteristics. Generally, solid substances are less accessible to the human body than volatile ones, i.e. fluids or gases. The type and level of human exposure is also dependent on the amount and type of human activities at the contaminated site. The degree of exposure differs, for instance, between people who live on a contaminated site and people who occasionally visit the same site. Moreover, individual physiological variables like sex, body mass, fitness and age matter as well. Due to their natural behaviour, children are more exposed to chemical substances than adults and some adults are, for unknown reasons, more sensitive than others. In the literature, *exposure factors* provide information about how much air a person inhales, how much water a person consumes, how much soil a child ingests etc.[6] By using these sources, 'typical' and 'worst-case' exposure scenarios can be outlined and evaluated.

Based on assumptions about the exposure, a reference value like for instance the Reference Daily Intake (RDI) can be used to approximate the internal dose.[7] If the RDI is exceeded due to additional exposure from a contaminated site, the 'new' internal dose could be high enough to cause health effects, and thus, motivate

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[5] This section is based on Liljelind & Barregård (2008).

[6] See for instance US-EPA, the U.S. Environmental Protection Agency (2007).

[7] For further information about RDI and estimated intake through food, see Swedish NFA, the Swedish National Food Administration (2007a ; 2007b).

remediation actions. The internal dose, usually expressed in mass per kilo body weight, depends on the absorption. The health effect for a given dose is usually estimated on the basis of toxicological or, if available, epidemiological data. Naturally, doses differ and hence, the degree and characteristics of the expected health effects vary. To determine the relation between the dose and the severity of its effect on exposed individuals, a *dose-effect relationship* can be applied (see Figure 2). The *dose-response relationship* (see Figure 2) illustrates the share of the population affected at a given dose.

Figure 2 Dose-effect and dose-response relationships.

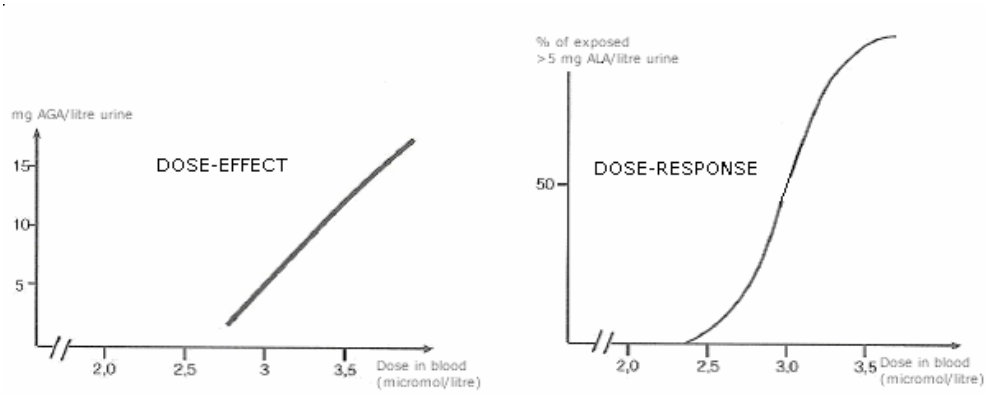

Illustration of dose-effect and dose-response relationship for increased concentration of aminolevulinic acid (ALA) in urine as a cause of occupational lead exposure.

Source: Nordberg (2004)

In a narrow sense the dose-response relationship refers to the dose actually absorbed by a receptor. If data on the actual absorbed dose is unavailable, the concentration (external dose) of a pollutant for various exposure pathways is used to implicitly account for the absorption of the pollutant from the environment into the body. If so, the dose-response function is replaced by an exposure-response function or a concentration-response function.

In sum, exposures to specific contaminants are essential for verifying the potential risks. Yet, the underlying assumptions behind exposure estimation can differ and hence affect the final risk estimation. The next section contrasts the Swedish EPA's approach for risk assessment of contaminated sites with the one applied within the area of environmental medicine.

Risk assessment – the environmental medicine approach versus the Swedish EPA's approach

In general, risk at contaminated sites is estimated by independent consultants guided by the Swedish EPA's guidelines for risk assessments. The implications of this procedure are that risks, both in regard to the environment and human health, are evaluated based on general, or site-specific, guideline values compiled by the Swedish EPA. The guideline values are in turn based on conservative assumptions about toxicological data and human exposure that often overestimate the risk posed by a site. Occasionally, health risk assessments are supplemented with formal opinions

from environmental medicine experts.[8] In contrast to the conventional procedure for health risk assessment, environmental medicine personnel make use of their qualifications in exposure assessment, toxicology and medicine to answer questions like: (*i*) what is the actual exposure at the specific site and (*ii*) what human health risks arise at this specific level of exposure?

All risk assessments aim at providing a scientific description of the risks at hand. The major divergences between the Swedish EPA's approach and the environmental medicine approach when it comes to assessing risk to human health can be summarised as follows:

- The Swedish EPA assesses risk based on divergence from guideline values for acceptable concentrations given a standardised (i.e. worst case) exposure situation on an individual level. The environmental medicine approach, however, targets prevailing health risks by accounting for the actual number of exposed individuals and the relevant exposure pathways. In other word, while the Swedish EPA approach implicates that areas with concentrations exceeding 'natural levels' by default are considered harmful, the environmental medicine determines harmfulness based on actual exposure for an actual group of people. The Swedish EPA approach can, hence, be regarded as more conservative than the environmental medicine approach.[9]

- The Swedish EPA approach involves rigid assumptions about the evaluated time frame. Whereas the environmental medicine approach focuses on the present risks and potential future risks within a time frame of a few decades, the Swedish EPA looks at (*i*) the present risk, (*ii*) the expected risk 50-100 years ahead, and (*iii*) the expected risks 100 to 1 000 years ahead (Swedish EPA, 2007c).[10]

- As the environmental medicine approach focuses on human exposure and actual health effects, the acceptable concentrations could differ with soil depth. That is, as the human exposure is higher on the ground surface than deeper down, higher concentrations could be accepted further down than on the surface. The Swedish EPA, however, commonly resists such a procedure with the motivation that land-use can change over time.

- The environmental medicine approach uses the most recent information in the scientific literature to establish human health risks, while the Swedish EPA approach relies on risk estimates from various international bodies which may be less updated.

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[8] Environmental medicine is a multidisciplinary field that deals with the interaction between risk factors in the environment and human health (Möller, 2000). The aim is to prevent disease by increasing the knowledge about exposure to risk factors, the mechanisms of the adverse effects, and the potential health consequences. Such knowledge can in turn provide the basis for safety limits, control of potentially dangerous substances or removal of unnecessary risks posed by for example contaminated sites.

[9] A potential explanation for this could be the *precautionary principle,* holding that in absence of a scientific consensus that harm will not prevail, measures should be taken (World Commission On Environment and Development, 1987). That is, in lack of a scientfically established cause-effect relationship that guarantees no harm for humans and the environment, a site should be remediated.

[10] A reason for the rather stringent strategy for risk assessment could be that the Swedish EPA is guided by the environmental objectives, and hence, aims at long-term sustainability. Additionally, the Swedish EPA might want to set an example in order to justify more ambitious remediations from the liable parties.

Prevailing differences in risk assessment are likely to have consequences for the subsequent risk valuation. Too much reliance on for instance the precautionary principle could exaggerate the risk at hand and, thus, stimulate preventions to be undertaken that largely reduce potential rather than actual risks. This is of course the right way to go if the individuals' risk valuation from contaminated sites is higher than the risk valuation from other environmental threats (e.g. radon indoor exposure, particle inhalation from air etc.). However, if not, it is important to transparently discuss the assumptions behind risk assessment and their effect on risk valuation.

4. The Need for Economic Valuation of Risk Reduction at Contaminated Sites

A Swedish EPA proposal for revised guideline instructions for prioritisation among contaminated sites was recently up for consideration. Several of the pronouncements opposed the rather conservative assumptions behind the Swedish EPA's recommendations for generating guideline values and risk assessment (see SCDA, Stockholm City Development Administration, 2007; EHA, Swedish Environment and Health Administration, 2007). An often raised argument against the stringent risk assessment is the high costs involved in remediation activities. In urban areas, where background concentrations are commonly exceeded even in absence of contamination, the current criteria for site clean-ups could have rather severe consequences. For example, about SEK 22 400 000, or SEK 25 000/m^2, was recently spent on remediation of 900 m^2 (0.22239 acre) in the city centre of Stockholm.[11] To fulfil the quantitative remediation objectives of 15 mg/kg arsenic in the ground surface, 4 550 m^3 of soil was excavated and brought to an external landfill. 'New' material, with arsenic concentrations below 15 mg/kg, was then transported to the site. Even if the site is located in the city centre where humans could be exposed, the high arsenic concentrations were found 2 to 4 metres under the ground surface. This raises questions: What was the actual exposure at the site before the clean-up? How much did it cost to reduce the particular risks at hand? Arguably, the health risks associated with living or working at, or adjacent to, a contaminated site might be low compared to other human risk exposure in urban environments. One argument for rather expensive site clean-ups in urban areas could be the demand for residential housing. However, if the demand is high enough, private operators would be enticed and, thus, governmental resources could be better spent somewhere else.

Additionally, opponents of the Swedish EPA's risk assessments criticise the current procedure for approximating bioavailability (i.e. assumptions about the level of uptake in the human body from various exposure pathways) (see SCDA, 2007). The uptake in the human body generally differs between different exposure pathways. The Swedish EPA's present application assumes the same bioavailability for all considered pathways.

In sum, our primary motive for economic valuation of remediation activities is to (*i*) provide insights about the value of current governmental preferences in this regard and (*ii*) to compare the value of risk reduction achieved by remediation to the value of risk preventive measures in other regards (e.g. measures to decrease indoor radon, ambient air pollution, noise, or prevent traffic accidents etc.). After a short explanation of why arsenic site remediation is chosen before other contaminant remediation, the following section provides data on formerly arsenic contaminated sites.

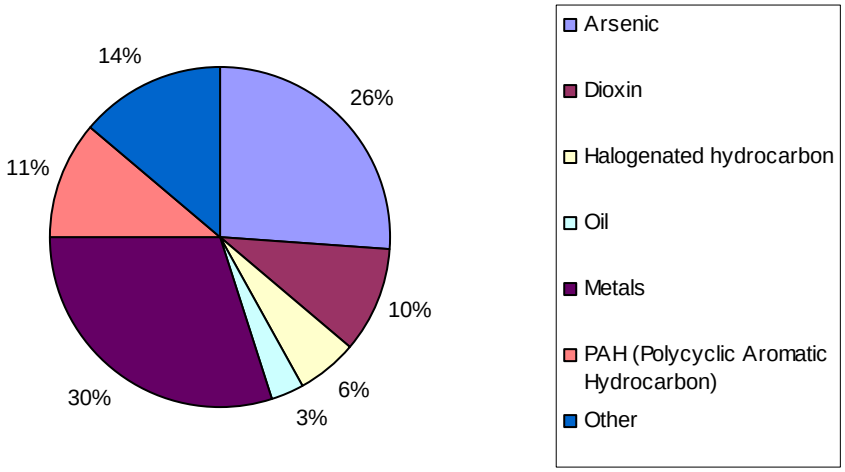
[11] Based on remediated arsenic contmainated sites in Sweden, the cost/m^2 of SEK 25 000 at Akterspegeln is about five times higher than the cost/m^2 at Tröingeberg, the site with the second highest cost/ m^2 in our sample. The average cost/m^2 amounts to SEK 2 991.

Arsenic contaminated sites

The main reason why we base our analysis of revealed governmental procedures for health risk reduction on sites contaminated by arsenic is twofold: Firstly, arsenic is the single most common contaminant among the prioritized and acute remediation projects in Sweden (Swedish EPA, 2008b). Secondly, the principal reason for arsenic mitigation is to protect human health rather than to reduce environmental damage (Swedish EPA, 2008a).

As illustrated in Figure 3 about 26 percent of the prioritized sites in Sweden are contaminated by arsenic. As shown other common pollutants are dioxin, polycyclic aromatic hydrocarbon (PAH), and metals.

Figure 3 Most common contaminants at prioritised sites in Sweden.



Source: Swedish EPA (2008b).

Arsenic and associated health risks[12]

Arsenic is a metalloid, i.e. having properties of both a metal and a non-metal, and occurs naturally in the environment. It is usually found as inorganic species (together with, e.g. oxygen, chlorine and sulphur), although organic compounds (combined with carbon) also may occur.

No natural destruction of arsenic occurs in the environment. The kind of arsenic species can, however, change by reacting with oxygen or other molecules in air, water, soil etc. Generally, arsenic from a contaminated site may occur in particles contained in windborne soil. The particles subsequently settle to the ground or get washed away by rain. As arsenic compounds dissolve in water, the contaminant reaches rivers and underground water by means of snow and rain. It then either sticks to particles in water, sediments or bottoms of rivers and lakes, or it continues to migrate. The final

[12] This section is primarily based on the ATSDR, Agency for Toxic Substances and Disease Registry, 2007.

destination is generally soil or sediment. It may also end up in fish. So-called 'fish-arsenic' is an organic form and is regarded as less harmful than the inorganic forms of arsenic.

The arsenic concentration in soil varies extensively. Non-contaminated soils in Sweden generally show concentrations of 0.2 – 40 mg/kg (Liljelind & Barregård, 2008). A common source of natural arsenic is the bedrock, ultimately affecting the groundwater. Arsenic may dissolve from the primary minerals into the groundwater and subsequently migrate to the surroundings. A well drilled in such a rock may – or may not – suffer from very high concentrations of arsenic. The Swedish limit for arsenic concentrations in drinking water is 10 µg/litre (WHO, World Health Organization, 1993; Swedish NFA, 2005). A national study on well water quality carried out in 14 of Sweden's 21 counties showed that 5 percent of the investigated wells contained arsenic concentrations above 10 µg/litre (SSI, Swedish Radiation Protection Authority, 2008). About 2 percent showed levels above 100 µg/litre. The latter can be contrasted to the tolerable weekly intake of 15 µg/litre arsenic. Notably, most households have public water supply, and only about 1 million households get their drinking water from private wells (SGU, Geological Survey of Sweden, 2008). Moreover, the SGU survey oversampled areas with geological features indicating possible high arsenic levels.

Since arsenic occurs naturally in the environment, humans are exposed just by eating food, by drinking water and by breathing. The scientific task is to determine the levels of arsenic exposure and their effect on human health and the environment when additional contaminant sources are involved. In terms of contaminated sites, the chemical form and size of arsenic particles are often hard to determine. Some forms are for instance closely attached to particles or embedded in minerals and, hence, not taken up by humans and the environment. To assess the health risks associated with an arsenic contaminated site, information about the substance's various geochemical forms is, hence, essential. In fact, recent research on arsenic in soils estimates that the bioavailability can vary from 5 percent to 80 percent (Lowney, 2008).

One potential health effect from more severe inorganic arsenic exposure is *acute poisoning*, i.e. the body is exposed to the toxic substance at a high dose, on one occasion or during a short period of time.

Long-lasting, or *chronic exposure* to moderate-high inorganic arsenic levels could affect the skin, the blood vessels and the nervous system. The most common health effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes, such as patches of discoloured skin and thickened skin in the palms. In addition, arsenic exposure may increase the risk of diabetes and general cardiovascular diseases. The health effects in children are similar to those in adults. Yet, the already mentioned differences in behaviour, body mass etc. affect the exposure and, subsequently, the internal dose.

In addition, arsenic is classified as carcinogenic to humans (IARC, International Agency for Research on Cancer, 2004; 2008).[13] That is, arsenic exposure is

[13] The IARC is part of the World Health Organization. IARC is the publisher of the Monograph series (1972-2002), which contains evaluations and classifications of environmental agents and exposures linked to development of human cancer. The categories are: Group 1: Carcinogenic to humans ; Group 2A: Probably carcinogenic to humans ; Group 2B: Possibly carcinogenic to humans ; Group 3: Not classifiable as to carcinogenicity to humans ; and Group 4: Probably not carcinogenic to humans.

scientifically proven to increase the risk of developing cancer, primarily in the lungs, the urinary bladder and the skin, but probably also in the liver and the kidneys (U.S. Department of Health and Human Services, US-HHS, 2007). At long term low-level exposure to inorganic arsenic, cancer is the most important health risk, since skin changes (other than cancer) and blood vessel disease does not occur below a certain exposure level. In contrast, the excess cancer risk is assumed to be proportional to dose, with no threshold. The cancer risk at low-level exposure is theoretically calculated by extrapolating to zero from dose-response relationships in epidemiological studies with high-level exposure (i.e. studies in Taiwan, Chile, and other areas with high arsenic in drinking water). For inhalation, similar extrapolations have been performed from studies of occupationally exposed workers inhaling dust containing arsenic. Therefore, limit values for arsenic and recommendations on preventive measures concerning arsenic in contaminated sites are based on cancer risks. The quantitative risk estimates have been based on skin cancer (WHO 1993, Swedish EPA 2008), and more recently lung and bladder cancer (US NAS 2001, Liljelind and Barregård 2008).

5. Presentation of Data[14]

Site Characteristics

As illustrated in Table 1, sites relevant for this project have arsenic as their *primary pollutant*, implying that despite the presence of other chemical compounds, arsenic was considered the most hazardous.[15] Table 1 further shows that the *arsenic quantity* on the sites varies from a few kilograms to several tonnes, where the bulk is residuals from previous industrial activities. The high representation of wood industry activities is to a large extent due to the historically incautious treatment of the preservative chromate copper arsenate (CCA), a compound that contains arsenic. Although the chemical is still used to prevent rot, mold and insect infestations in wood products, the current use is strictly regulated (KIFS, Kemikalieinspektionens författningssamling, 1998:8). Hazardous residues also originate from former glasswork and sulphate industries. Chemicals containing arsenic were, for example, used in the glasswork industry to purify crystal, whereas arsenic residues from the sulphate industry originate from the by-product pyrites.

Table 1 also provides information about the *remediation status*. As shown, 10 out of 23 arsenic remediation projects are considered completed whereas the rest are considered to be still in progress. Technically, a project is not completed until a final report, containing a description of the site clean-up and involved costs, is produced (Söderström, 2008).

[14] For detailed information about assumptions, calculations, and sources, see Forslund & Barregård (2008).

[15] At the time of data collection a total number of 50 sites were remediated. Of these 15 referred to arsenic mitigation. Two of the completed projects were eliminated from the data set due to lack of information, whereas 10, by then, on-going projects were added to improve the data basis.

Table 1 General site specific characteristics

Object	Industry	Primary pollutant (Secondary pollutant)	As Quantity (kg)	Remediation status
Akterspegeln	Sulfuric acid	As (Pb, Cu, Zn)	1.000-10.000	Completed
Robertsfors	Wood impregnation	As (Cr)	10.000-100.000	In progress
Burträskbygden	Wood Processing	As (-)	10-100	In progress
Tvärån	Wood impregnation	As (-)	1.000-10.000	Completed
Svartbyn	Wood impregnation	As (-)	10-100	Completed
Sjösa	Wood impregnation	As (Dioxin)	100-1.000	In progress
Lyshälla	Wood impregnation	As (-)	100-1.000	Completed
Mjölby	Wood impregnation	As (-)	100-1.000	In progress
Rimforsa	Wood impregnation	As (Dioxin)	100-1.000	In progress
Hjulsbro	Electroplating	As (Pb)	100-1.000	In progress
Glasbrukstomten	Glassworks	As (Pb)	>10.000	In progress
Grimstorp	Wood impregnation	As (PAH)	>10.000	In progress
Elnaryd	Wood impregnation	As (PAH)	>10.000	In progress
Högsby - Ruda	Wood impregnation, Glassworks	As (Cr, Cu)	>10.000	Completed
Tröingeberg	Electroplating	As, Ni (solvents)	100-1.000	In progess
Oxhult	Saw mill	As, Cr (Cu)	100-1.000	Completed
Gudarp	Wood impregnation	As (Cu, Cr)	>10.000	In progress
Konsterud	Saw mill, Wood impregnation	As (Cu)	-	Completed
Kramfors	Sulphate industry	As (Pb)	1.000-10.000	Completed
Svanö	Sulphate industry	As (Zn)	1.000-10.000	Completed
Svartvik	Saw mill, Sulphate industry	As (Dioxin)	100-1.000	In progress
Forsmo	Wood impregnation	As (PAH)	>10.000	Completed
Fagervik	Saw mill	As (Petrol)	>10.000	In progress

Note. The data in Table 1 is based on the quarterly report (quarter 4, 2007) provided by the county administrative boards by order of the Swedish EPA.

Concentrations

To estimate the risk reduction associated with arsenic mitigation of contaminated sites the *average concentrations pre remediation* have been collected. A reason for estimating risk based on an average concentration is that, over time, an individual will be exposed to an average concentration rather than to exceptionally high or low concentrations (US-EPA, 1992; Swedish EPA 2008).[16] However, as the average site concentration depends on depth and range of the investigated area, number of samples, purpose of sampling (i.e. to define the contaminated area or to define the average concentration), and the distribution of concentrations (e.g. many samples with low concentrations and few with very high concentrations), a conservative average concentration (i.e. UCL_{95}) is preferred (US-EPA, 2002 ; Swedish EPA, 2008c). To validate the concentrations

[16] Commonly applied concentration values in risk assessments of contaminated sites are: average concentration; Upper Confidence Limit (UCL) of the mean (based on t-statistics); a specific percentile (e.g. the 50th percentile or the 95th percentile), and maximum measured concentration (Swedish EPA, 2008c).

applied in this memo, the figures have been derived with help from agent officials involved in remediation. Additionally, to avoid underestimation of the objective risk, conservative assumptions primarily in regard to number of people exposed and exposure times have been made; that is, the numbers of exposed individuals are based on the upper bound of the applied intervals (see Table 3), and the exposure times in regard to for instance residential activities are assumed to be as high as 24 h a day.

As illustrated in Table 2, the average arsenic concentrations pre remediation in the sample show a range from 23 to 1128 mg/kg TS.[17]

The *acceptable arsenic concentrations* refer to the quantitative remediation objective. As illustrated in Table 2, the majority of the sample sites have remediation objectives that correspond to the Swedish EPA's guideline values for either *sensitive*, i.e. 15 mg/kg TS, or *less sensitive*, i.e. 40 mg/kg TS, land use. *Sensitive* land use refers to for example residential areas, daycares and cultivation involving extensive exposure times and sensitive exposure groups, i.e. children and old individuals. *Less sensitive* land use refers to for example industrial areas and areas for infrastructural activities, i.e. roads, involving limited exposure times and less sensitive exposure groups, i.e. employees. As shown, some of the quantified remediation objectives have, however, been adjusted in regard to the site-specific background concentrations of arsenic.

The migration potential for sites in the sample is considered to be *moderate* to *very high*. This implies that the arsenic contamination in the soil and groundwater may migrate at rates from about 0.1 m/year to 10 m/year (Swedish EPA, 2002). Thus, an elevated migration potential could motivate a site clean-up even if the average concentrations are modest.

[17] The natural background concentrations of arsenic vary. Depending on geographical location, concentrations from 3 to 15 mg/kg are found. Notably, for almost half of the sample sites the investigation reports do not provide information on background concentrations. Thus, these are not included in the subsequent quantifications.

Table 2 Site specific concentration data

Object	Arsenic concentration (mg/kg)[a, b]		Migration[c]
	Ex Ante	Ex Post	
Akterspegeln	163	15	Very High
Robertsfors	250	15	High
Burträskbygden	260	40	High
Tvärån	608	17	High
Svartbyn	80	15	High
Sjösa	30	6	High
Lyshälla	170	15	Very high
Mjölby	46	40	High
Rimforsa	49	15	High
Hjulsbro	87	15	Very high
Glasbrukstomten	102	20 ;10 ;10	High
Grimstorp	424	10	Very high
Elnaryd	130	40	Very high
Högsby - Ruda[d]	55 ; 41	5	Very high
Tröingeberg	23	15	Moderate
Oxhult	94	15	Very high
Gudarp	119	80	Moderate
Konsterud	119	15	Moderate
Kramfors	500	15	Very high
Svanö	418	100	Very high
Svartvik	150	40	Very high
Forsmo	1128	10	Very high
Fagervik	65	40	Very high

Note. [a] The ex ante arsenic concentration is based on the average site concentration based on information in site investigation reports or from involved consultants.[b] The ex post arsenic concentration is based on acceptable concentrations presented in the site investigation reports or collected from involved consultants. The acceptable concentration refer to the quantitative remediation objective as directed by the Swedish EPA's guideline values for the designed land use.[18][c] The migration status is based on the quarterly report (quarter 4, 2007) provided by the county administrative boards by order of the Swedish EPA. [c] As the average concentration for this site is missing the median concentration have been applied.

Exposed populations

Table 3 shows the numbers of exposed individuals. The data was collected from agent officials (i.e. municipality or county administrative board personnel) who where asked to approximate the number of individuals on and adjacent to (i.e. within 500 m of) a particular site. To simplify the approximation, the following intervals were given: 1-10, 10-100 and 100-1000. In addition, the respondents were asked to address the current and designated land use as well as the occurrence of children on or adjacent to the site.

As Table 3 shows, the number of exposed individuals pre remediation (i.e. in the last decade before remediation) is for the most part limited to less than 10, whereas only

[18] The Swedish EPA's guideline values are under revision. For arsenic this means that the guideline values for sensitive and less sensitive land use will be lowered from 15 mg/kg TS to 12 mg/kg TS (sensitive land use) and from 40 mg/kg TS to 30 mg/kg TS (Swedish EPA, 2008d). However, the former guideline values are the ones relevant for the arsenic contaminated sites considered here.

two sites show exposure numbers between 100 and 1000. The approximation of exposed individuals post remediation (i.e. in the decade following remediation) does not radically differ from the pre clean-up one. Only four sites show changed exposure situations.

The data on *land use* in Table 3 is relevant for approximating the time of exposure. An individual's exposure time if residing at, or close to, a contaminated site is, for example, about four times longer than the occupational exposure time. The daily exposure times applied in subsequent quantifications (see the next section) is 24 hours for individuals residing on or adjacent to a site, 1 hour for recreational activities, and 5.7 hours for occupational activities. If pertinent, 5.7 hours also applies to daycare/school.[19]

Table 3 Site specific exposure data

Object	Exposed individuals[a, b]		Land use[c]	
	Ex Ante	Ex Post	Ex Ante	Ex Post
Akterspegeln	100 – 1.000*	100 – 1.000*	Recreational	Recreational
Robertsfors*	10 – 100	10 - 100	Recreational	Recreational
Burträskbygden	1 – 10	1 - 10	Industrial	Industrial
Tvärån	10 – 100	10 - 100	Industrial	Industrial
Svartbyn	1 – 10	1 - 10*	Residential	Residential
Sjösa	10 - 100*	10 - 100*	Industrial	Industrial
Lyshälla	1 – 10	1 - 10*	Residential	Residential
Mjölby	1 – 10	1 - 10	Industrial	Industrial
Rimforsa	1 – 10	10 - 100	Industrial	Residential
Hjulsbro	10 - 100*	100 – 1.000*	Recreational	Recreational
Glasbrukstomten	100 – 1.000	100 – 1.000	Industrial	Industrial
	10 – 100	10 – 100	Industrial	Industrial
	10 – 100*	10-100*	Recreational	Recreational
Grimstorp	1 – 10	10 - 100	Industrial	Industrial
Elnaryd	1 – 10	1 - 10	Industrial	Industrial
Högsby – Ruda	10 – 100	10 - 100	Industrial	Industrial
	1 – 10*	1 – 10*	Residential	Residential
	10 – 100	10 – 100	Industrial	Industrial
	1 - 10	1 - 10*	Recreational	Recreational
Tröingeberg	10 – 100*	10 - 100*	Residential	Residential
Oxhult	1 - 10	1 - 10	Residential	Residential
Gudarp	10 - 100	10 - 100	Recreational	Recreational
Konsterud	10 – 100*	10 - 100*	Residential	Residential
Kramfors	1 – 10	1 - 10*	Industrial	Recreational
Svanö	10 - 100*	10 - 100*	Recreational	Recreational
Svartvik	1 – 10	100 - 1000*	Recreational	Recreational
Forsmo	1 – 10	1 - 10	Recreational	Recreational
Fagervik	10 – 100*	100 – 1.000	Recreational	Recreational

Note. [a] Number of exposed individuals are collected from agent officials. To control for exposure of children, individuals in the age of 0 – 3 years as share of the municipalities' total population (i.e. 4 – 100 years) is applied (Statistic Sweden, 2008).[b] The asterisk indicates sites where children could be exposed to arsenic. [c] The land use data are collected from agent officials.

Data for Quantification of Health Risk

Valuation of risk reduction at contaminated sites requires that the risks at hand are defined and quantified. As already emphasised, the risk targeted in this memo is human health risk associated with arsenic exposure. When deriving the guideline values for contaminated sites, the Swedish EPA recognises three types of risks to human health: (i) long-term risks from carcinogenic substances, (ii) long-term risks from non-carcinogenic substances and (iii) acute risks. As arsenic is a carcinogenic substance the risk to be quantified in this exercise is defined in terms of extra risk of developing cancer over the course of a lifetime. 'Extra' refers to the additional cancer risk due to exposure to contaminated sites.

Acute risk of arsenic exposure is, however, not taken into account. Acute risks are primarily associated with children's pica-behaviour, e.g. the risk that a child ingests a small volume of soil with an extremely high arsenic concentration. Arguably, the low level of child exposure in our data sample makes acute effects less relevant. In addition, the application of average arsenic concentrations would underestimate the acute risks, and hence, not accurately consider the acute effects.

Pertinent exposure pathways when analysing risks related to contaminant exposure are: (*i)* inhalation of air, (*ii)* ingestion of soil, (*iii)* skin contact and, if relevant, (*iv*) ingestion of groundwater and intake of vegetables. Table 4 presents and describes the exposure-response functions applied for each considered exposure pathway. As illustrated, three different functions are considered. In contrast to the Swedish EPA, the risks associated with soil ingestion, skin contact and ingestion of groundwater are quantified in regard to both lifetime risk of skin cancer based on WHO (1993) and risk for lung and bladder cancer based on US-NAS (2001). The reason for this is to make full use of the present knowledge regarding arsenic exposure and cancer risk. The applied exposure-response functions are mainly based on epidemiological studies in Taiwan and Chile. These data are then applied on Swedish background rates of lung and bladder cancer in Sweden (Liljelind and Barregård 2008) as was previously done in the USA (US NAS 2001). In other words, the risk estimates are originally determined by observing large, well-defined populations in order to discover the excess probability of developing cancer over a lifetime at a certain arsenic level.

Table 4 Quantified cancer risks, descriptions and sources.

Exposure pathway	Cancer risk (Concentration)	Description[a]	Source
Inhalation of Air	1.5×10^{-3} (1 µg/m^3)	At an air concentration of 1 µg/m^3 an estimate of excess lifetime risk is 1.5×10^{-3}. This means an excess lifetime cancer risk level of 1:100.000 at an air concentration of about 0.0067 µg/m^3.	WHO (2000)
Ingestion of Soil ; Skin Contact ; Ingestion of groundwater	a) 6×10^{-4} (0.01 mg/litre or 0.00033 mg/kg/day)	At a concentration of 0.01 mg/litre an estimate of excess lifetime skin cancer risk is 6×10^{-4}. This means an excess lifetime cancer risk level of 1:100.000 at a concentration of 0.017 mg/litre or 6×10^{-6} mg/kg/day.[b]	WHO (1993)
	b) 2.5×10^{-3} (0.01 mg/litre or 0.00029 mg/kg/day)	At a concentration of 0.01 mg/litre an estimate of excess lifetime risk is 2.5×10^{-3}. This means an excess lifetime risk of 1:100.000 at a concentration of 0.004 mg/litre or 1×10^{-6} mg/kg/day.[c]	US-NAS (2001) ; Barregård (2008b)

[a] The excess cancer risk at a risk level of 1:100.000 is derived by dividing the risk level (here 1 x 10-5) by the announced cancer risk.[b] In accordance with WHO (1993) the daily intake of water is 2 litre/day and the body weight is 60 kg, thus the conversion from 0.01 mg/litre to 0,00033 mg/kg/day is done by multiplying 0.01 mg/litre by 2 litre/60 kg, which gives 0.00033 mg/kg/day. The cancer risk of 6 x 10-4 is thereafter divided by 0.00033 mg/kg/day, which, after gives the cancer risk of 1.8 per mg/kg/day (or more realistically 1.8 x 10-3 per µg/kg/day.)[c] The estimation is based on NAS (2001) and applied to Swedish background concentrations and daily intake of water. Here the daily intake of water is 2 litre/day and the body weight is 70 kg, thus the conversion from 0.01 mg/litre to 0.00029 mg/kg/day is done by multiplying 0.01 mg/litre to 2 litre/70 kg, which gives 0.00029 mg/kg/day. The cancer risk of 2.5 x 10-3 is thereafter divided by 0.00029 mg/kg/day, which gives the cancer risk of 8.8 per mg/kg/day (8.8 x 10-3 per µg/kg/day).

To derive the health risk reduction at the 23 sites, the site specific data presented in Tables 1-3 is applied to the exposure-response functions presented in Table 4 (for a complete example, see Appendix A). The exposure-response functions were selected by environmental medicine experts, who also contributed with necessary assumptions about, for instance, dust fraction, concentration factors and bioavailability of arsenic. As addressed earlier (see page 4), the Swedish EPA approach for risk assessment involves rigid assumptions about the evaluated time frame (i.e. 10-1.000 years), whereas the environmental medicine approach focuses on the present risks and potential future risks within a time frame of about 10-20 years. In accordance with the US-EPA we will apply a time frame of 30 years (Hamilton,Viscusi and Dockins, 1997).

Remediation costs

To approximate the cost per cancer case averted by arsenic remediation, economic data was collected from the administrative authority. By order of the Swedish EPA, the county administrative boards provide quarterly reports useful for our purpose. In addition to the general site information (e.g. location, primary pollutant, migration potential etc.) the reports comprise cost data (i.e. allocated resources for remediation purposes) categorised as follows: *allocated funds*, *pre remediation costs*, *estimated final remediation cost*, *issued funds for remediation contract work*[20] and *approximated final costs*. The cost categories are subsequently subdivided into *governmental funding*, *municipal funding* and *external funding*.[21]

Governmental funding for remediation purposes has primarily taken two forms in Sweden: *sakanslag* (directed grants) and *Lokala investeringsprogram* (Local Investment Programmes, or LIP).[22] The funding principle is that up to 90 percent of a total project cost can be obtained from the administrative authority, whereas the remaining 10 percent is borne by the responsible authority. In Sweden the responsible authority is commonly the municipality in which the remediation object is located. Hence, the municipal funding in the quarterly reports refers to the amount borne by the municipality, whereas external funding refers to costs borne by other authorities, e.g. the European Union.

In Table 6, the *total pre remediation cost* is the sum of governmental funding, municipal funding and external funding allocated for costs incurred before the actual contract work at the site began, e.g. costs for site investigations and risk assessment. All cost types are here considered as direct or indirect governmental expenditures and, hence, summed up to approximate the total cost pre remediation. The underlying practice is the same for *total remediation cost* (see Table 6). That is, all funds allocated to bear contract work costs are summed up. Yet, to avoid underestimation of on-going project expenses, two different cost categories are applied. For completed sites, the total remediation cost is based on *issued funds for remediation contract work*, whereas the category *approximated final costs* is used for sites in progress.

[20] Here the defintion of contract work refers to the actual site clean-up, excluding costs for preceding site investigations, risk assessment etc.

[21] Own translation.

[22] The LIP, in effect between 1998 and 2002, was an investment subsidy programme with a dual purpose: to speed up Sweden's transformation into an ecologically sustainable society and to reduce unemployment.

Table 5 Total remediation cost per site

Object	Status[a]	Total pre remediation cost[b]	Total remediation cost[b]	Total cost[c]
Akterspegeln	Completed	785.000	22.400.000	23.185.000
Robertsfors	In progress	1.448.000	57.985.934	59.433.934
Burträskbygden	In progress	680.350	6.940.000	7.620.350
Tvärån	Completed	59.000	15.435.619	15.494.619
Svartbyn	Completed	172.176	1.950.000	2.122.176
Sjösa	In progress	1.748.762	31.000.000	32.748.762
Lyshälla	Completed	250.000	977.383	1.227.383
Mjölby	In progress	703.250	2.000.000	2.703.250
Rimforsa	In progress	1.220.099	8.600.000	9.820.099
Hjulsbro	In progress	419.711	800.000	1.219.711
Glasbrukstomten	In progress	0	88.000.000	88.000.000
Grimstorp	In progress	4.110.779	122.800.000	126.910.779
Elnaryd	In progress	1.084.848	83.750.000	84.834.848
Högsby - Ruda	Completed	4.623.651	70.776.349	75.400.000
Tröingeberg	In progress	1.850.919	7.500.000	9.350.919
Oxhult	Completed	200.000	2.653.000	2.853.000
Gudarp	In progress	0	73.666.537	73.666.537
Konsterud	Completed	0	9.087.563	9.087.563
Kramfors	Completed	803.290	14.291.405	15.072.604
Svanö	In progress	0	34.080.000	34.080.000
Svartvik	In progress	1.058.106	83.874.592	84.932.698
Forsmo	Completed	486.947	24.171.485	24.658.432
Fagervik	In progress	6.539.845	90.000.000	96.539.845

[a] For more information on "status", see Table 1.[b] The data is based on the quarterly report (quarter 4, 2007) provided by the country administrative boards by order of the Swedish EPA.[c] Sum of total pre remediation cost and total remediation cost.

6. Concluding remarks

By presenting data from governmentally funded arsenic remediation projects, this memo provides information that will be used in a forthcoming analysis on governmental preferences for risk reductions at contaminated sites in Sweden.

Appendix A

Quantification of extra cancer risk posed by arsenic contaminated air on the basis of the concentration in soil.

The exercise is based on the assumption that a site's average arsenic concentration is 163 mg/kg. The site and its surroundings are used for recreational purposes and the number of individuals visiting the site is 100 per day.

Relevant parameters for approximating air exposure are the number of *soil particles in inhaled air*, the *respirable particle fraction* and the *time of exposure* (Swedish EPA, 1997).

The soil particles in inhaled air in our study refer to the concentration of inhalable dust. To control for the fact that fine particles in the air may contain higher concentrations than a relatively larger sample of soil, a concentration factor of 1-5 is applied.

The excess inhalable particle concentration tells how much of the total dust in the air that originates from the contaminated site. That is, the parameter depends on the soil characteristic (i.e. grass, sand, soil) and is assumed to vary from 1 to5 μg/m^3.

The time of exposure is based on the land use. Approximating recreational activities to one hour a day, the number of individuals exposed 24 hours/day is given by $(1\,h \div 24\,h) \times 100 = 4{,}16 \approx 4$.

Given the information above, the arsenic concentration in inhaled air can be calculated as:

$$1\text{-}25\,mg/m^3 \times 0{,}163\,ng/mg = 0{,}163\text{-}4{,}075\,ng/m^3 \approx 0{,}16\text{-}4{,}1\,ng/m^3 .$$

As emphasised in Table 4 (see page 13), the exposure-response function applied to quantify the number of cancer cases from air inhalation over a lifetime at or adjacent to the site is $1{,}5 \times 10^{-6}$ per ng/m^3. The effect (cancer risk) is given by:

$$0{,}16\text{-}4{,}1\,ng/m^3 \times 4\,individuals \times (1{,}5 \times 10^{-6}\,ng/m^3) = 0{,}96 \times 10^{-6}\text{-}24{,}6 \times 10^{-6} \approx 1\text{-}25 \times 10^{-6} .$$

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