ASROCKS-project is partly funded by EU's Life+ Environment Policy and Governance –programme. Partners of the project: Geological Survey of Finland, Tampere University of Technology and Finnish Environment Institute.

Preliminary risk assessment for rock aggregate production and construction sites

- Building a Conceptual Site Model and defining worst case scenarios

Jaana Sorvari and Heli Lehtinen
Finnish Environment Institute (SYKE)
P.O.Box 140, FI-00251 HELSINKI, Finland

Preliminary risk assessment - Building a Conceptual Site Model and defining worst case scenarios

Jaana Sorvari and Heli Lehtinen, Finnish Environment Institute

Introduction

Risk assessment (RA) is a procedure where the risk posed to human health and/or the environment by the actual or potential presence or release of hazardous substances, pollutants or contaminants is evaluated either qualitatively or quantitatively. The existence of risks requires the involvement of all its elements, that is, a source, transport pathway, exposure route and recipient (Fig. 1).

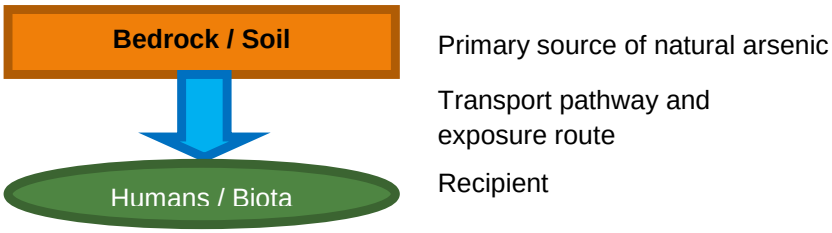


Figure 1. Formation of risks caused by naturally occurring arsenic.

RA procedure starts with the formulation of a Conceptual Site Model (CSM) that addresses all these elements. CSM defines the objectives and boundaries of RA and it can be updated and specified along the RA process when more data become available. The ultimate goal, as well as the resources and data that are available, determine the RA approach, i.e. whether RA will be based on realistic or worst case assumptions. Realistic RA requires more precise data than the worst case RA.

The occurrence of the source, i.e. the amount of a harmful substance and the spatial extent of its occurrence in the environment are crucial factors in the formation of risks. Other relevant factors include soil, groundwater and surface water conditions and their characteristics, land use pattern, barriers that limit contaminant transport and any channels or fractures that could enhance transportation. The worst case approach is based on the definition of scenarios where the source, transportation, exposure and recipients are defined to indicate the highest possible risks.

Environmental fate and toxicity of arsenic

Humans

Drinking water resources located within or in the vicinity of sites with high environmental arsenic concentrations may be an important contributor in the formation of human health risks. In the general population, the primary route of arsenic exposure is through food ingestion, though. Hence, food is considered the main contributor to total arsenic intake. In the group of imported foodstuffs, rice is probably the most important source of arsenic. Finnish food items generally contain only low concentrations of arsenic, with the exception of some Baltic fish species. It is worth noting that inorganic arsenic has rarely been found to substantially accumulate in biota, however. Arsenic in seafood is mainly in the form of non-toxic arsenobetaines.

Arsenic is both genotoxic and a well-known human carcinogen. Arsenite (As^{3+}) is considered to be a more potential carcinogen than arsenate (As^{5+}). The principal target organs of carcinogenic response to arsenic are the lungs, skin and bladder. Lung cancer appears to be a critical effect following chronic inhalation exposure of arsenic. Arsenic is transported in air mainly via particles since gaseous arsenic compounds only form in very specific conditions.

Also non-cancer toxicity in long term oral arsenic exposure has been verified. Only few data is available on non-cancer effect in humans exposed to inorganic arsenic by inhalation. Effects are expected to be improbable below a concentration of about 0.1-1.0 mg As/m^3.

Biota

Studies have shown that aquatic biota can experience adverse effects already in very small concentrations of arsenic. Attention should therefore be paid particularly to ecological risks to receptors in the nearby water bodies. Arsenic has been claimed to be one of the most toxic elements to fish with chronic exposures resulting in accumulation up to toxic levels. Chronic toxicity in long-term exposure to various aquatic organisms has been verified in laboratory tests and growth inhibition in algae and water flea can occur already in very low concentrations (see Table 1). Acute toxicity is mostly rather low, however.

Table 1. Toxicity of arsenic to aquatic biota.

Target organism and species	**Effective concentration**	**Explanation**	**Source**
Midge, larva (*Chironomus riparius*) (*Tanytarsus dissimilis*) *Chironomus sp.*	97,0 mg/l 358000 / 435000 µg/l	LC_{50} (48 h) LC_{50} (96 h)	Holcombe et al., 1983 ref Sample et al.1997 Jeyasingham & Ling, 2000
Amphipod, crustacean (*Hyalella azteca*)	483 / 581 µg/l	LC_{50} (7 d) H_3AsO_4 di-Na-salt	Borgmann et al., 2005
Copepods	100 µg/l	NOEC (14 d), H_3AsO_4 di-Na-salt, *Tigriopus japonicus*, reproduction and lethality	Lee et al., 2008
Aquatic plants - lemna - waterweed (*Hydrilla verticillata*)	1000 µg/l 63200 µg/l 8180 50 µmol/l[a] (= 9400 µg/)	NOEC (20 d), H_3AsO_4 mono-Na-salt, *Lemna gibba*, growth EC50 (7 d), H_3AsO_4 di-Na- salt, *Lemna minor*, growth EC50 (7 d)), H_3AsO_4 di-Na- salt, *Lemna minor*, chlorophyl, NOEC (4 h), H_3AsO_4 di-Na- salt biochem. & entzymatic changes	Mkandawire et al., 2006 Naumann et al., 2007 “ Srivastava & D'Souza, 2010
Algae - bluegreen algae *Microcystis sp.*, - green algae *Stichococcus bacillaris*	 0,1..100 µmol/l[a] (= 19…19000 µg/l) 100 µmol/l 100 µmol/l	NOEC (14 d), H_3AsO_4 di-Na-salt, biochemical changes ”, population growth rate NOEC (48 h), H_3AsO_4 di-Na-salt, population biomass	Gong et al., 2009 Pawlik-Skowronska et al., 2004
Water flea (*Daphnia* sp., *Ceriodaphnia* sp., *Simo cephalus*)	5200…15000 µg/l 7400 µg/l 38 / 20 µg/l 5500 µg/l 2600 µg/l 520 / 1400 µg/l 1500…4300 µg/l	EC_{50} (48 h), H_3AsO_4, *D. magna*, mobility LC_{50} (24 h), H_3AsO_4, *D. magna* LOEC/NOEC (21 d), H_3AsO_4, *D. magna*, growth NOEL (24 h), H_3AsO_4, *D. magna*, lethality NOEL (48 h), H_3AsO_4, *D. magna*, mobility LC_{50} (48 h), *D. magna*, *D. pulex*, *Ceriodaphnia reticulata*	Pesticide Ecotoxicity Database, 2013 « « « « « «
Molluscs - clam *Mercenaria mercenaria* - snail *Biomphalaria glabrata* - blue mussel *Mytilus edulis*	 14000 12000 50 / 100 µg/l 3000 µg/l	 EC_{50} (48 h), H_3AsO_4, mobility NOEL (48 h), H_3AsO_4, mobility NOEC (96 h), As-oxide, biochemical changes EC_{50} (48 h) As_2O_5, development	 Pesticide Ecotoxicity Database, 2013 Ansaldo et al., 2006 Martin et al., 1981
Amphibians, *Euphlyctis hexadactylus*	270 / 249 µg/l	LC_{50} (72 h / 96 h) As-oxide,	Khangarot et al., 1985
Fish - species not defined - different species - salmon (*Salmo gairdnerii)* - rainbow trout (*Oncorhynchus mykiss*)	 10000…18000 µg/l 550 µg/l 50500…72000 3400 / 32000 µg/l 23300…26600 µg/l	 C (24-48 h), As_2O_5 LC_{50} (24 h), As_2O_5 LC_{50} (96 h), H_3AsO_4 NOEL (96 h), H_3AsO_4 LC_{50} (96 h), As III	 Suter & Tsao, 1996 Nikunen et al., 2000 “ Pesticide Ecotoxicity Database, 2013 Spehar et al. 1980, ref. Sample et al. 1997

LC50 = lethal concentration, concentration that kills 50% of test organisms, NOEC/NOEL = no observed effect concentration/level, EC50 = effective concentration where 50% of test organisms experience specified adverse effects (e.g. reduced mobility, growth, reproduction etc.)

The toxicity of arsenic to soil organisms and terrestrial animals varies widely (Table 2).

Table 2. Toxicity of arsenic to some plants and animals. Doses refer to oral intake.

Target organism and species	Concentration / Dose	Explanation	Source
honeybee *Apis mellifera*	157 µg	LD_{50} (96 h), DMA	Pesticide Ecotoxicity Database, 2013
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*)	22 mg/kg-dw in soil	geometric mean of LOAEC and NOAEC, growth	Jiang and Singh, 1994[1]
- blueberry (*Vaccinum angustifolium*)	55 mg/kg-dw in soil¨	"	
- spruce	1000 mg/kg-dw in soil		Anastasia and Kender, 1973[1]
	97 mg/kg-dw in soil	LOEC (335 d), As_2O_3, height	Rosehart and Lee, 1973[2]
	22 mg/kg-dw in soil	geometric mean of LOAEC and NOAEC, growth	Jacobs et al., 1970[1]
- potato (*Solanum tuberosum*)		"	Jiang and Singh, 1994[1]
- barley (*Hordeum vulgare*)			
Birds			
- mallard duck (*Anas platyrhynchos*)	3.72 / 17.3 mg/kg-bw/d	NOAEL (10 w / 4 w), mortality, juveniles	Camardese et al., 1990[1] / Hoffman et al., 1992[1]
	0.410 mg/kg-bw/d	LOAEL (10 w), enzyme activity (acetylcholine-esterase), juveniles	Camardese et al., 1990[1]
	1.49 mg/kg-bw/d	LOAEL (2 w), growth, juveniles	
	386 mg/kg	LD_{50} (single dose), $NaAsO_2$	Camardese et al., 1990[1]
- pheasant (*Phasianus colchicus*)		LC_{50}, arsenic compounds	Sample et al., 1996
	17.4…3,300		
- various species			Eisler, 1988
Terrestrial mammals			
- rat (*Rattus norvegicus*)	1.39...32 mg/kg-bw/d	NOAEL (varying exposure time), mortality, juveniles	studies reviewed in USEPA, 2005
	5.0…20.6 mg/kg-bw/d	LOAEL (varying exposure time), growth, juveniles	"
	0.447...10.3 mg/kg-bw/d	NOAEL (varying exposure time), growth, juveniles	"
	1.2 mg/kg-bw/d	LOAEL (6 w), enzyme activity (general changes), juveniles	Wood and Fowler, 1978[1]
			Nemec et al., 1998[1]
- rabbit (*Oryctolagus cuniculus*)	0.750 / 3.0 mg/kg-bw/d	NOAEL / LOAEL (12 d), mortality, gestation	Sample et al., 1996
- mouse (species not specified)	0.44...19	LOAEL (lifetime), varying exposure dose and toxic effect, As III	Opresko, 1994
- meadow vole (species not specified)	0.114 mg/kg-bw/d	NOAEL, arsenite (derived from test species: mouse)	Sample et al. 1996
- short-tailed shrew (species not specified)	0.150 mg/kg-bw/d	NOAEL, arsenite (derived from test species: mouse)	Sample et al. 1996
Domestic animals			
- dog (*Canis familiaris*)	2.25 / 5.62 mg/kg-bw/d	NOAEL / LOAEL (2 yr), $NaAsO_2$, mortality, juveniles	Byron et al., 1967[1]
	1.04 / 1.66 mg/kg-bw/d		
	24.0 / 48.0 mg/kg-bw/d	NOAEL / LOAEL, (8 w), growth, juveniles	Neiger and Osweiler, 1989[1]
- mouse (*Mus musculus*)	2.84...7.69 /5.69…32.4mg/kg-bw/d		Nemec et al., 1998[1]
	0.00650; 0.548 4mg/kg-bw/d	NOAEL / LOAEL (9 d), mortality, gestation	studies reviewed in USEPA, 2005
		NOAEL /LOAEL (varying exposure time), growth, juveniles	
		LOAEL (91 d; 6 mo), reproduction, juveniles	Healy et al., 1998[1]; Schroeder and Mitchener, 1971[1]

LD50 = lethal dose, Dose that kills 50% of test organisms, LOEC/LO(A)EL = lowest observed effect concentration/(adverse) effect level, LCT = lowest concentration tested, NOAEC/NOAEL = no observed adverse effect concentration/level, EC50 = effective concentration where 50% of test organisms experience specified adverse effects (e.g. reduced mobility, growth, reproduction etc.)

[1] Ref. in USEPA, 2005

[2] Ref. in Will and Suter, 1995

In the surrounding areas of a quarry site, plants can be exposed to arsenic via wet or dry deposition or through root uptake from soil. In general, plants are more sensitive to arsenic than animals, the water-soluble forms being the most phytotoxic and arsenite more toxic than arsenate. On the other hand, some plant species are highly tolerant to arsenic. A few hyperaccumulators, such as some ferns (*Pteris vittata*)

have also been identified. Arsenic ions are toxic to most micro-organisms. However, due to their capability of adapting to varying environmental conditions and developing resistance mechanisms, a wide range of micro-organisms can also survive even in habitats with high concentration of arsenic. Microbes can also transform inorganic arsenic to methylated compounds. Hence, interactions of microbes with arsenic species in soil and water affect the environmental fate of arsenic.

Besides arsenic speciation, several abiotic factors such as, temperature, pH, redox-potential, organic matter content, phosphate concentration, adsorption to solid matrices, the presence of other substances and toxicants, as well as the duration of exposure, affect the sensitivity of biota to arsenic. Therefore, comparison of environmental concentrations with the toxicity thresholds can merely be considered to provide an indication of potential effects.

Quarry sites[1]

Life cycle of a quarry

The life cycle of a quarry producing bedrock aggregates includes several phases (Fig. 2). The activities at the site differ at these stages and consequently, the formation and magnitude of risks also differ. This means that risk management actions are not uniform either.

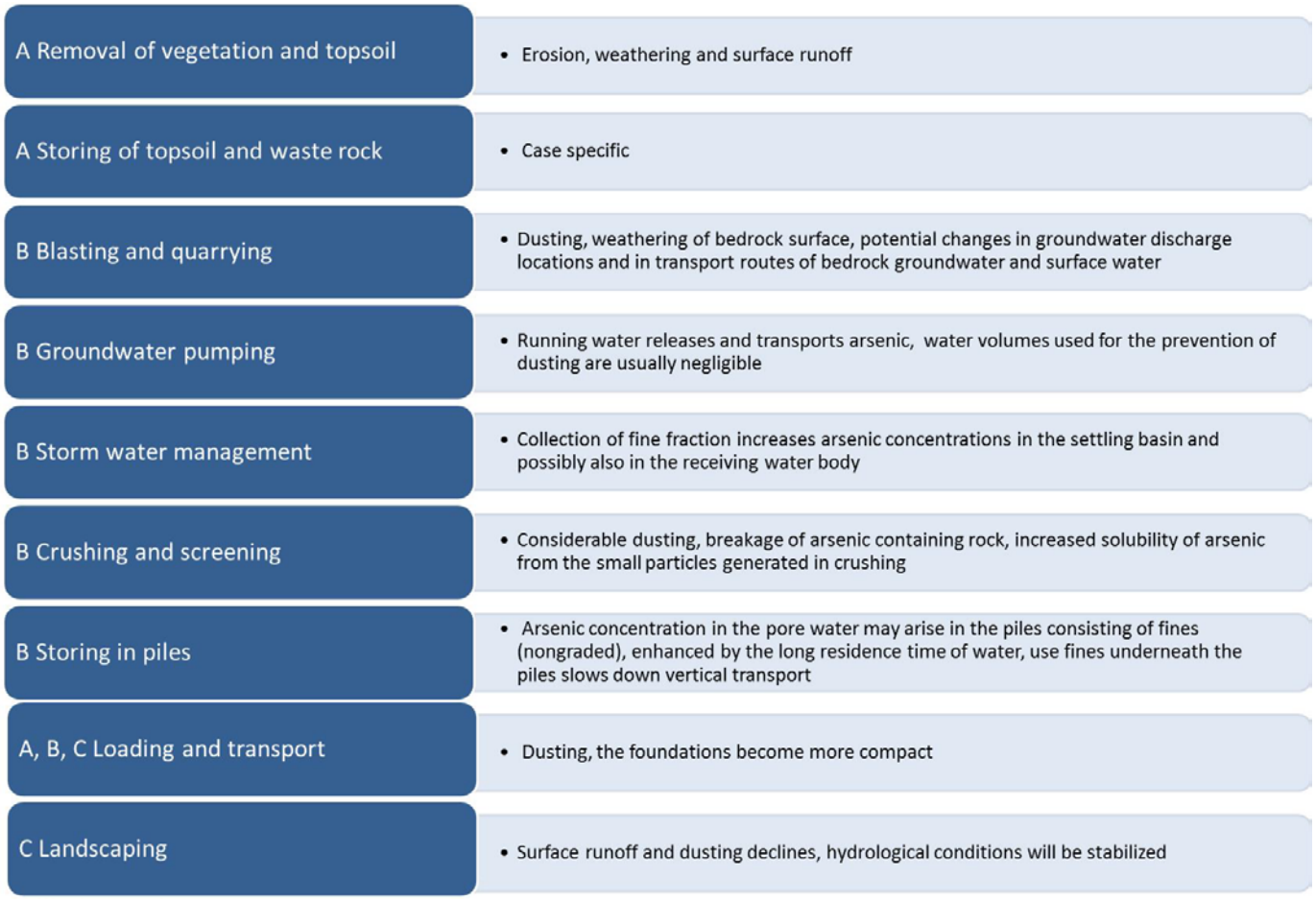


Figure 2. Activities and the potential emissions and release mechanisms of arsenic during the life cycle of a quarry. A. Planning and foundation, B. Operation, C. Closing and aftercare.

[1] This guidance focuses on bedrock aggregates, and hence extraction of sand or gravel is not considered. According to the ASROCKS project, in sites producing sand and gravel based aggregates arsenic only occurs in low concentrations that do not cause significant risks to the environment or human health.

The short-term preparatory works such as removal of plants and top soil are expected to cause only very minor health or environmental risks compared to the risks during the operation. Storing of waste rock and surplus soil from the preparatory works can increase arsenic emissions from these matrices during the operating time. In Finland quarries generally operate at least 10 years.

In practice, risk assessment should also consider not only the current situation, but also any known future land use after the closing of the quarry. Such risk assessment follows the generic procedure described in many RA guidelines of contaminated sites. The focus in this guidance is therefore on an operating quarry.

Source and transport pathways of arsenic

In an operating quarry, the main source of arsenic to be studied is the rock material. As a result of blasting and crushing, arsenic that is bound to rock material is exposed to aerobic conditions which can increase its solubility. Dissolution from sulfide minerals is then one of the most relevant release mechanisms of natural arsenic. Dissolved arsenic can be further transported to the environment by seepage water or via surface runoff.

The chemistry of arsenic in natural waters is very complex and affected particularly by pH and redox potential. Therefore, prediction of the environmental fate and consequences of arsenic in natural waters is in fact difficult. In aquatic environments typical to Nordic countries, particularly the formation of complexes with iron or binding to humus or particles can reduce the toxic effects of arsenic.

Figure 3. Transport with surface water was identified as the major mechanism spreading arsenic in the environment at rock aggregate production sites.

At a quarry site, air is also a potential transport route for arsenic, transport via air particles being the main mechanism. Arsenic only volatilizes in a very specific conditions, hence, it is not expected to be released to the environment in gaseous form at quarrys.

Recipients at production sites

At quarries, the major health risks to humans are occupational risks arising from dusting. However, according to the Finnish Institute for Occupational Health, instead of any chemicals like arsenic,.the inhaled fine quartz particles cause the most significant health risks related to the dusting. So far, no

evidence exists of significant exposure of arsenic via dusting among the operators of quarrying sites. Moreover, occupational health and safety measures should eliminate or minimize these risks.

Some aggregate producers utilize deep bedrock groundwater available on site mainly for dust prevention in the crushing and screening phase. This water can contain high levels of arsenic of natural origin, and should therefore not be used for human consumption.

No ecological recipients are expected to permanently occupy operating quarry sites. Occasional visits of migratory birds and other animals with broad habitats would not pose significant risks to these fauna due to the short exposure times.

Recipients within the impact area

Depending on the land use of the surrounding area and environmental conditions (e.g. wind speed and direction, vegetation), some human recipients could be exposed outside the site to air dust originating from the quarry. Nearby residents may also receive arsenic in domestic water that is prone to emissions from the quarry. It is therefore worth noting any use of local water for human consumption, though such situations are expected to be rare. Identification and mapping all potential domestic water sources in the vicinity of the site should in fact be included in the permit procedure related to the establishment of a quarry.

No significant human health risks are expected from the consumption of local food items contaminated by arsenic released in aggregate production although high concentrations of arsenic have been found in berries and mushrooms growing in the vicinity of mine sites. Due to the similarity of the activities, these food items could be the most potential sources of to human intake related to quarries. The concentrations of arsenic in rock material are, however, several orders of magnitude lower at the production sites studied in the ASROCKS project compared to the mines with arsenic-bearing ore. Residential areas may include small scale cultivation of vegetables in private gardens. Wet or dry deposition could bring about arsenic to such plants. Root intake is not a probable transport mechanism since the topsoil is normally replaced with clean soil in built areas. Several studies also show that arsenic mainly stays in the surface of the plant and would in most part be removed by washing or peeling. Overall, the low average arsenic concentrations and land use in the surroundings suggest that plant intake would not pose significant risks to human health in the study areas of the ASROCKS project.

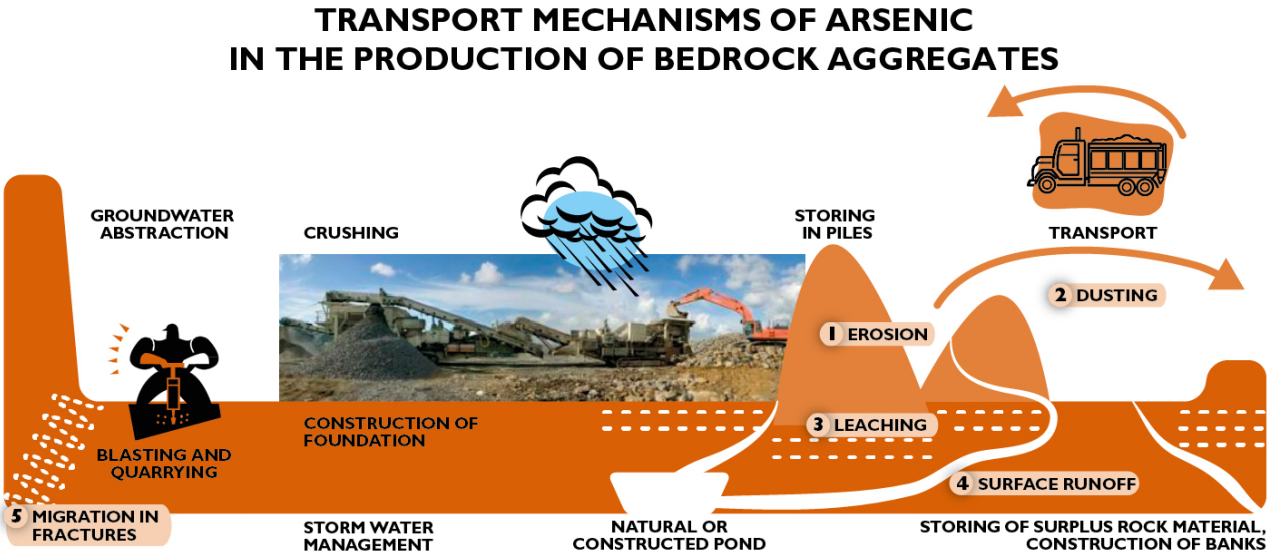
Skin absorption of arsenic bound to solid particles, e.g. dust, is negligible and would not cause any significant risk neither to humans or animals. Fine dust spreading to the environment could however expose humans and above-ground animals to arsenic through inhalation. From the ecological recipients, small mammals are expected to be the most sensitive target organisms (see Table 2). Again, the concentrations found in the ASROCKS project imply no significant human health or ecological risks related to inhalation of dust.

Considering any ecological effects it is most important to identify any protected ecosystems or species within the impact area This should be done already in the planning phase of a quarry in order to determine the necessary risk management measures. In the ASROCKS project, migration of arsenic via waterways was identified as the most important transport mechanism that could lead to adverse environmental effects. Sensitive recipients could therefore include a specific protected species living in a water body that is connected to the primary receiving watercourse of quarry effluents.

Conceptual Site Models for quarries and construction sites

Figure 4 summarizes the potential transport pathways at an operating, aggregate producing quarry and a construction site. The generic CSM should consider all these identified potential transport pathways.

A.



B.

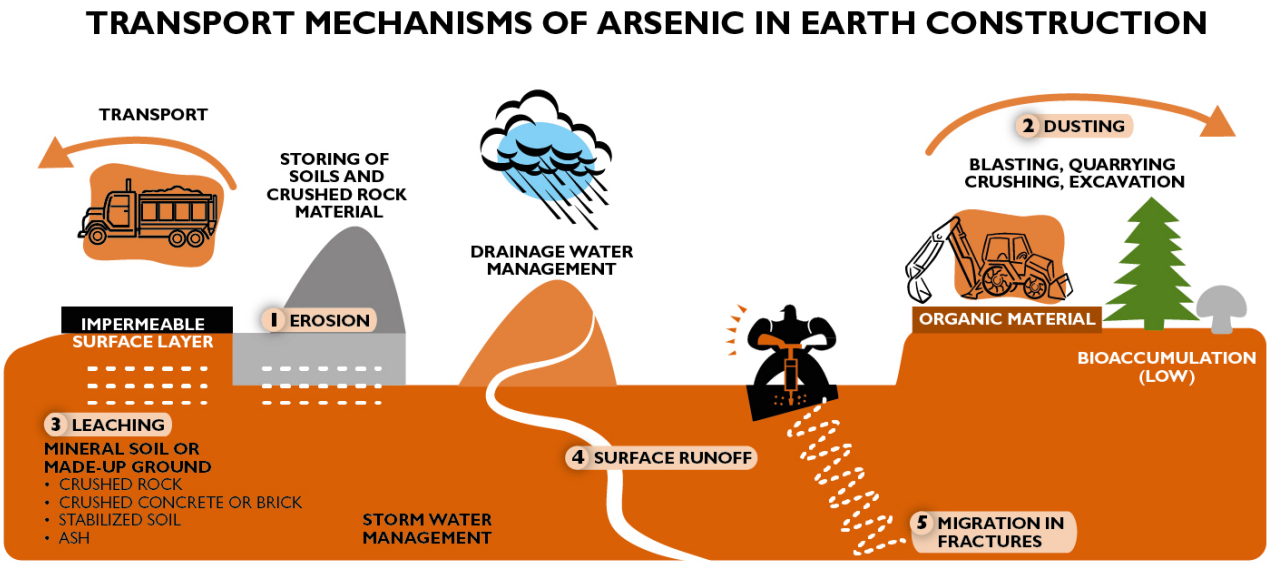


Figure 4. Potential environmental release and transport mechanisms (of arsenic) at an operating quarry (A) and at a construction site (B).

Figure 5 further presents the generic CSM that defines the objectives and boundaries for human health risk assessment. Some exposure routes can be assumed to be insignificant solely on the basis of literature data (e.g. skin contact).

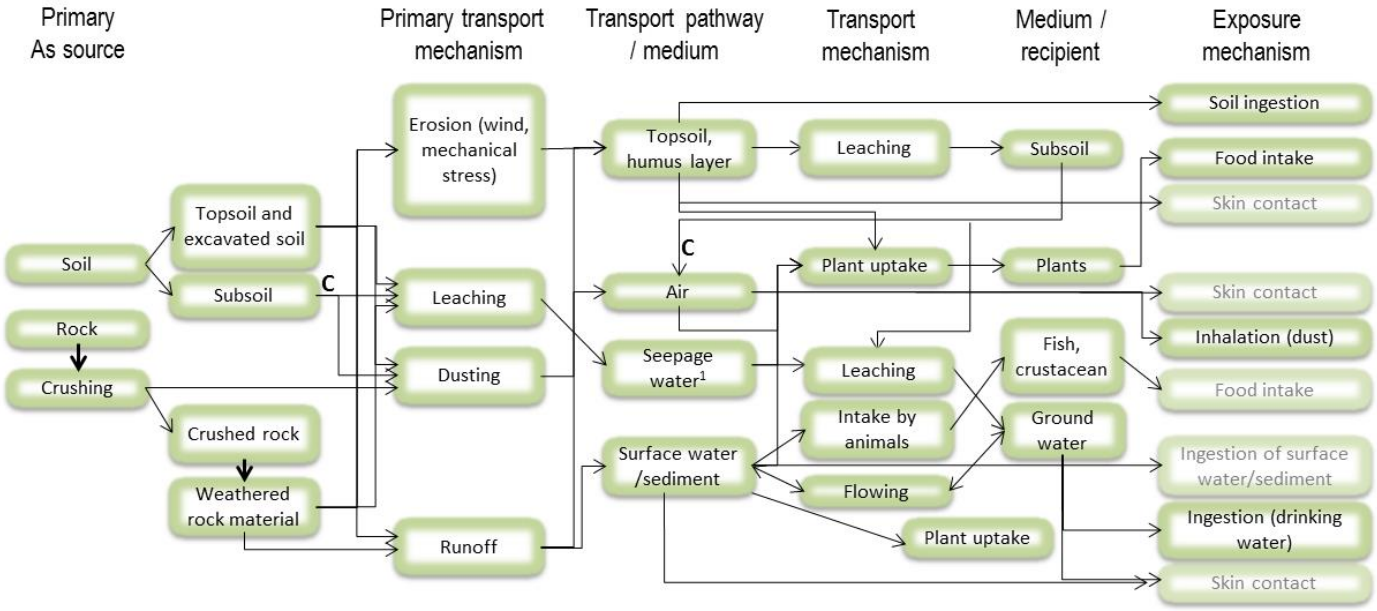


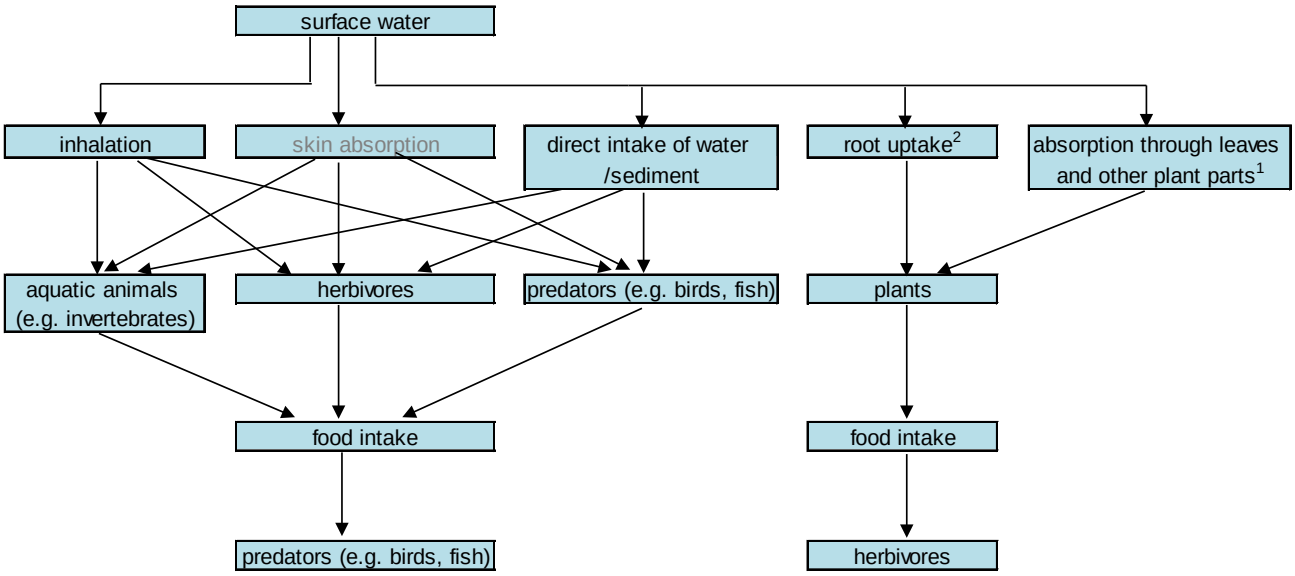
Figure 5. Formation of arsenic (As) induced human health risks at rock aggregate production sites or their surroundings and at construction sites, during or after termination of activities. Those pathways marked with C refer to construction only. Exposure mechanisms shown with dim font and border line are expected to be insignificant.

Based on the site data, some transport pathways and exposure routes could be further ignored in the risk assessment as being insignificant or non-existent. For example the studies in the ASROCKS project showed that arsenic emitted to surface water at the study sites quickly decreased down to the level of natural background concentration along with the increasing distance from the source. Due to this and the fact that the primary receiving and transporting waterways are small ditches or streams, the human intake via exposure to fish or other aquatic food sources potentially exposed to quarry-related arsenic is considered insignificant. In addition, considering the land use, soil ingestion is expected to be a relevant exposure route only at construction sites and when excavated arsenic-bearing soil material or fine rock material is used on site as topsoil.

Formation of ecological risks

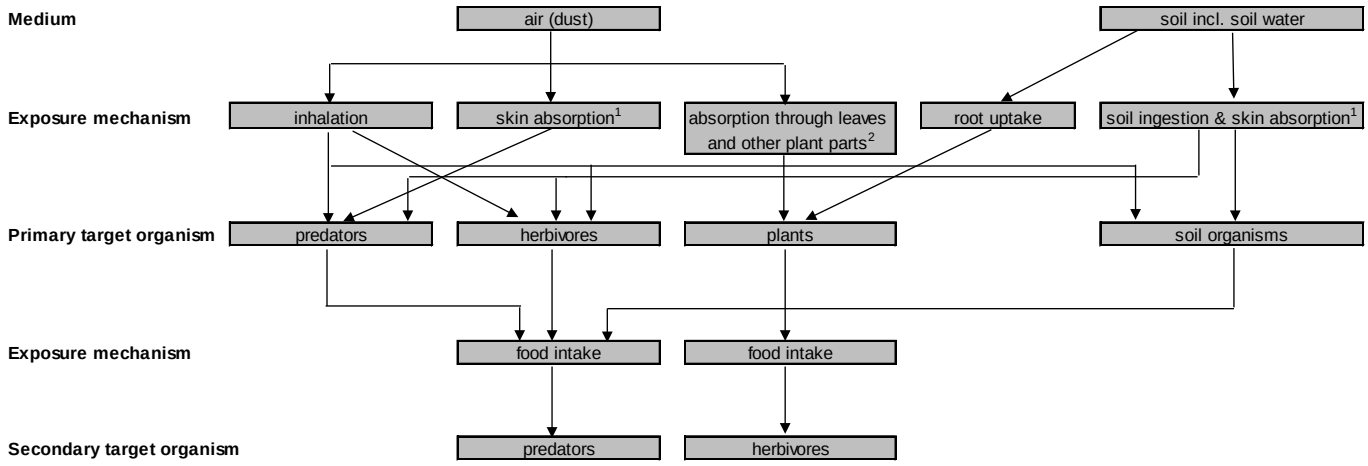
The CSM describing the formation of ecological risks should take into account the habitat type of the surrounding environment that can be affected by the aggregate production activities. Emphasis should be placed particularly in any protected ecosystems or species. When relevant, not only the primary target organisms but also the secondary exposure of animals in higher trophic levels that might be exposed through food intake or predation should be considered (Fig. 4).

A. Aquatic organisms



[1] particularly arsenic hyperaccumulators

B. Terrestrial organisms



[1] mainly only soft-bodied animals such as earthworms
[2] particularly arsenic hyperaccumulators

Figure 4. Formation of arsenic (As) induced ecological risks at rock aggregate production sites or their surroundings and at construction sites, during or after termination of activities. Exposure mechanisms shown with dim font and border line are expected to be insignificant.

Worst case scenarios

When data on the potential source, e.g. occurrence of arsenic, transport pathways, exposure routes or recipients is inadequate or when the aim is to determine the highest possible risks, it is a common practice to conduct the risk assessment based on the worst case scenario. It is worth noting that due to the conservative assumptions this approach easily leads to overestimation of actual risks.

Quarries

1. Arsenic source

Normally there is no exhaustive data on the extent and concentration level of arsenic in the bedrock since unfortunately, the existence of arsenic in rock material cannot be visually identified. In the ASROCKS project, sulphide-bearing gabbro and metavolcanic rocks tend to have higher arsenic concentrations than granite, granodiorite, tonalite and mica gneiss. However, elevated As concentrations were also measured in some sulfide-bearing veins and from the surfaces of cracks in bedrock in a quarry where the dominating rock type was mica gneiss.Therefore, some chemical studies are needed to find out whether and how much arsenic is present. Such studies could be conducted on site using a portable measuring device based on XRF.

If some arsenic analyses are conducted, the representativeness of sampling becomes the main issue, i.e. whether the samples represent the actual high end concentrations or not.

Besides the concentration in rock material, the solubility of arsenic is a crucial factor in the formation of risks. In the ASROCKS project, only a small part (maximum 2 %) of the *aqua regia* soluble arsenic in of crushed bedrock aggregates was soluble in the percolation test at the liquid/solid ratio of 10. While the easily leachable and potentially available arsenic determined by ammonium acetate-EDTA extraction was 14 %, at the maximum.

.

2. Transport pathways

In the ASROCKS project, surface runoff was identified the major transport mechanism potentially accounting for any ecological risks that might appear in the surroundings of a quarry site. In the worst case, the arsenic discharge could reach some sensitive or otherwise protected water bodies. The distance between the primary receiving water course (usually a small ditch or brook) and the sensitive water body as well as the characteristics of the latter, are then the key factors determining whether arsenic release will be a threat to any ecological recipients.

At quarries, arsenic transport via fractures in the bedrock can be an important transport pathway of arsenic along with waters. In the worst case, transport along fractures could lead to the contamination of an important groundwater reserve used as a domestic water source.

Arsenic can also be transported outside the aggregate production site via air dust. The worst case scenario related to this transport pathway should be based on the maximum operating time and production volume. Crushing techniques and equipment could also affect the formation of dust. The topography and shape of the quarry, climate conditions (e.g. wind speed and prevailing direction) and vegetative cover of the surroundings determine how far from the source the dust is transported. In the worst case, there would be no vegetation that would prevent the spreading of arsenic-containing dust to distant locations.

3. Toxicity, exposure and recipients

The chemical form and valence state significantly affects the toxicity of arsenic to both human and ecological recipients. In the RA based on worst case scenario and when there is no definite information on the actual speciation, it is generally assumed that the contaminant exists in its most harmful form. In reality arsenic can be present in several different forms and it can also transform to other species due to changes in the environment. In practice, it is extremely difficult to predict which would be the prevailing chemical form of environmental arsenic.

Residential area is the most sensitive land use from the viewpoint of human exposure since the exposure time is the longest. Then if risks are assessed quantitatively, RA should also consider sensitive recipients such as small children which are more sensitive to harmful substances than adults. In residential areas,

groundwater which can receive arsenic emissions from a quarry is also a potential exposure medium to humans. It is worth noting that the elevated levels of arsenic might be of natural origin i.e. not caused by the production activities per se.

A waterway can be sensitive due to, for example low water turnover rate or low depth. The aquatic ecosystem may be vulnerable due to presence of sensitive species. From the risk management viewpoint, water bodies are generally protected on the basis of their importance as a resource to human consumption or as a habitat of protected ecosystems or animal or plant species. Considering human health risks, the worst case scenario would involve using of arsenic containing surface water directly as domestic water. This scenario is highly improbable, however, since surface water would normally be treated before its use for human consumption. Recreational use (swimming, fishing) can be considered a more probable worst case scenario, depending on the type of the water body.

Dusting can result in dry or wet deposition of arsenic on the surface of food items. This could pose a risk to human health if there is at least small scale cultivation or recreational activities including picking of berries or mushrooms. It is however worth noting that only high concentrations of arsenic in the dust are expected to lead to significant human exposure through this route.

Although not all of the arsenic entering human or ecological recipients is bioavailable, a quantitative RA based on the worst case scenario generally assumes 100 % bioavailability. In calculating the human exposure, worst case scenario also means the use of exposure parameters that produce the highest possible intake estimate. USEPA and the European research institute JRC, among others, have compiled exposure parameter data for RAs that can be used to define the values that can be used in such a quantitative health risk assessment.

Construction sites

At construction sites, risks to human beings or ecological recipients during construction are expected to remain insignificant due to the brevity of the activities. The activities are mainly equivalent to those of aggregate production (blasting, crushing, transportations) and hence, air dust would be the most important medium to look at. In addition, potential runoff along with waters may transport arsenic outside the construction site.

Also, the same principles of the formation of risks described above for quarries are mainly applicable to construction sites. Moreover, risk assessment of a built site does not differ from the procedure used in the context of contaminated sites. Since many countries have issued guidelines for the risk assessment guidelines for contaminated sites, the approach for the worst case scenario is only briefly described here.

Arsenic source

1. Arsenic source

Besides the concentration of arsenic, the magnitude of risks after the finalization of construction depend on the location of arsenic-bearing materials, i.e. whether they have been used in the surface layer or in deeper layers. Generally speaking, surface soil can be expected to cause the highest risks since it allows a direct contact with the potential human and ecological recipients and arsenic uptake by plants. Surface soil is also prone to dusting which can lead to exposure via inhalation. Exposure from deeper soil layers is also possible during maintenance of cables or pipelines but would also be a minor contributor to the overall risks due to the brevity of the actions. From the viewpoint of topsoil as a source of arsenic, the worst case scenario would be the use of fresh excavated soil or crushed rock aggregate since in fresh material no weathering that normally causes dissolving and leaching of arsenic has not yet occurred.

Possible changes in the environment and how these changes might affect the speciation or solubility of arsenic should be taken into account when defining the worst case scenario. In particular, changes in the

pH value have been shown to affect the mobility of arsenic. On the basis of the results from the ASROCKS project, the solubility of As from crushed rock material was highest when the pH was below 4 or above 9. It is therefore important to identify the situations where pH could change so that it enables increased mobility of arsenic.

2. Transport pathways

In built areas, a key issue determining the importance of arsenic transport is whether the surface is paved or covered, for example by vegetation. Uncovered surface allows transport of arsenic by dusting by or surface runoff. At least in Finland, transport along with groundwater is not a probable transport pathway due to the limited spatial dimensions of construction sites and the amount of rock or soil material used in them. Relocation of arsenic-bearing topsoils on site may be a problem in some cases, but according to the ASROCKS project not really an issue at the Pirkanmaa study region.

3. Exposure and recipients

Land use is obviously the key determinant in the formation of risks since it defines the exposure time and recipients that can be exposed. Like in the case of a quarry, residential land use would be the worst case from the viewpoint of human exposure while ecological risks would involve the presence of some sensitive, protected species. In the case of topsoil containing arsenic, soil ingestion would be the main contributor to human health risks or ecological risks. In case of risks related to soil ingestion, the worst case should address young children and toddlers due to their hand-to-mouth behavior. Inhalation of air dust could be an exposure route to consider in the case of high arsenic concentrations. Built areas are not expected to be used for large scale cultivation of edible plants. Moreover, peeling, washing and cooking would remove most of arsenic from these.

In built areas, the natural habitat of biota has already been changed and no animals of higher trophic levels are expected to permanently dwell on site. The ecological risks would therefore mainly fall on soil organisms as maintainers of soil functions. Very often compensation by less sensitive organisms and recovery will ensure the important soil functions to continue even if elevated concentrations of arsenic are present. Most terrestrial organisms are also not particularly sensitive to arsenic so only rather high concentrations would cause any adverse effects to them (see also above).

Some remarks

In is worth noting that groundwater at or in the surroundings of aggregate production sites and construction sites can contain naturally high concentrations of arsenic. Such concentrations do not originate from any human activities. In practice it is very difficult to state whether the elevated concentration in groundwater has been caused by quarrying or construction activities or not. Any disturbance such as rock blasting, quarrying and soil excavation can however increase the mobility of arsenic by bringing the material to contact with oxygen or spreading the arsenic-bearing dust to the environment. This could consequently increase the magnitude of exposure. Finding out what is the contribution of human activities would require information on the groundwater concentration before starting the aggregate production activities (or construction). At least in Finland, such information is generally not available. In practice, Finnish authorities require the establishment of safety zones around aggregate production sites for the minimization of the spreading of dust.

Some specific occupations or hobbies, such as conservation of animals, might expose humans to arsenic. This, as well as intake of arsenic-containing food, such as rise, and smoking could add to the overall human exposure to arsenic and hence the risk of toxic manifestations.

Any contact with material that could change the pH value of arsenic-bearing material could increase the mobility of arsenic. For example contact with lime, ash or concrete could potentially cause an increase in the pH value and consequently increase the solubility or arsenic. The latter two materials are sometimes used in earth construction to replace primary aggregates whereas lime could be used as a soil amendment in parks and green zones.

Use of products, e.g. rock aggregates, and soil material outside the study sites was not considered in the ASROCKS project. The quality of building products is in fact controlled by CE marking system, which is currently being updated. The CE mark includes information of the technical properties of the product and information of the possible hazardous substances. Any building product, including aggregates, that might emit hazardous substance to soil, ground or surface water would then need to be tested. Any threshold values for hazardous substances would be defined at the national level, however. Potential large-scale utilization of mineral materials from construction sites, e.g. in noise barriers, was also not considered in ASROCKS. Such use would require case-specific assessment of the potential releases and consequent risks to the environment (and human health).

References

Ansaldo,M., Nahabedian, D.E., Holmes-Brown, E., Agote, M., Ansay, C.V., Guerrero, N.R.V., Wider, E.A. 2006. Potential Use of Glycogen Level as Biomarker of Chemical Stress in Biomphalaria glabrata. Toxicology224(1-2): 119-127.

Borgmann,U., Couillard, Y., Doyle, P., Dixon, D.G. 2005. Toxicity of Sixty-Three Metals and Metalloids to Hyalella azteca at Two Levels of Water Hardness. Environ. Toxicol. Chem.24(3): 641-652

Efroymson, R.A., Will, M.E., Suter, II G.W., 1997. Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Process: 1997 Revision. ES/ER/TM-126/R2. U.S. Department of Energy, Oak Ridge, USA.

Eisler, R., 1988. Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review. Biological Report 85(1.12). U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, USA.

Gong,Y., Song, L., Wu, X., Xiao, B., Fang, T., Liu, J. 2009. Effects of Arsenate on Microcystin Content and Leakage of Microcystis Strain PCC7806 Under Various Phosphate Regimes. Environ. Toxicol.24(1): 87-94.

IARC http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C-6.pdf

Jeyasingham,K. & Ling, N. 2000. Acute Toxicity of Arsenic to Three Species of New Zealand Chironomids: Chironomus zealandicus, Chironomus sp. a and Polypedilum pavidus (Diptera, Chironomidae). Bull. Environ. Contam. Toxicol.64(5): 708-715.

Joint Research Centre (JRC) http://cem.jrc.it/expofacts

Khangarot,B.S., Sehgal, A., Bhasin, M.K. 1985. "Man and Biosphere" - Studies on the Sikkim Himalayas. Part 5: Acute Toxicity of Selected Heavy Metals on the Tadpoles of Rana hexadactyla. Acta Hydrochim. Hydrobiol.13(2): 259-263.

Lee,K.W., Raisuddin, S., Hwang, D.S. , Park, H.G., Dahms, H.U., Ahn, I.Y., Lee, J.S. 2008. Two-Generation Toxicity Study on the Copepod Model Species Tigriopus japonicas. Chemosphere 72:1359-1365.

Martin,M., Osborn, K.E., Billig, P., Glickstein, N. 1981. Toxicities of Ten Metals to Crassostrea gigas and Mytilus edulis Embryos and Cancer magister Larvae. Mar. Pollut. Bull.12(9): 305-308.

Mkandawire,M., Taubert, B., Dudel, E.G. 2006. Limitations of Growth-Parameters in Lemna gibba Bioassays for Arsenic and Uranium Under Variable Phosphate Availability. Ecotoxicol. Environ. Saf.65(1): 118-128.

Naumann,B., M. Eberius, M., Appenroth, K.J. 2007. Growth Rate Based Dose-Response Relationships and EC-Values of Ten Heavy Metals Using the Duckweed Growth Inhibition Test (ISO 20079) with Lemna minor L. Clone St. J. Plant Physiol.164(12): 1656-1664.

Nikunen, E. 1993. Ympäristölle vaaralliset kemikaalit. Chemas Oy, Helsinki.

Nikunen, E., Leinonen, R., Kemiläinen, B. & Kultamaa, A., 2000. Environmental Properties of Chemicals, Volume 1, Finnish Environment Institute, Helsinki, Finland. 1165 p.

Pawlik-Skowronska,B., Pirszel, J., Kalinowska, R., Skowronski. T. 2004. Arsenic Availability, Toxicity and Direct Role of GSH and Phytochelatins in As Detoxification in the Green Alga Stichococcus bacillaris. Aquat. Toxicol.70(3): 201-212.

Pesticide Ecotoxicity Database. 2013. U.S. Environmental Protection Agency, and Office of Pesticide Programs

Rodushkina, I., Ödman, F., Holmström, H. 1999. Multi-element analysis of wild berries from northern Sweden by ICP techniques. Sci. Tot. Environ 231, 53-65

Sample, B.E., Opresko, D.M., Suter, II G.W., 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. ES/ER/TM-86/R3. U.S. Department of Energy, Office of Environmental Management, Oak Ridge, USA.

Sample, B.E., Suter, II G.W., Sheaffer, M.B., Jones, D.S., Efroymson, R.A., 1997. Ecotoxicological Profiles for Selected Metals and Other Inorganic Chemicals. ES/ER/TM-210. Oak Ridge National Laboratory, Oak Ridge, USA.

Sorvari, J., Schultz, E., Rossi, E., Lehtinen, H., Joutti, A., Vaajasaari, K. & Kauppila, T. 2007. Risk Assessment of Natural and Anthropogenic Arsenic in Pirkanmaa Region, Finland. Miscellaneous publications. Espoo, Geological Survey of Finland. 96 pp. + appendices. Available at: http://projects.gtk.fi/ramas/reports/.

Srivastava,S., D'Souza, S.F. 2010. Effect of Variable Sulfur Supply on Arsenic Tolerance and Antioxidant Responses in Hydrilla verticillata (L.f.) Royle. Ecotoxicol. Environ. Saf.73(6): 1314-1322.

Suter, II G.W., Tsao, C.L., 1996. Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota on Oak Ridge Reservation: 1996 Revision. ES/ER/TM-96/R2. Oak Ridge National Laboratory, Oak Ridge, TN. 104 p.

USEPA (United States Environmental Protection Agency). 2005. Ecological Soil Screening Levels for Arsenic. Interim Final. Office of Solid Waste and Emergency Response, Washington D.C, USA.

USEPA (United States Environmental Protection Agency). 1997. Exposure Factors Handbook. Vol I, II, III. EPA/600/P-95/002Fa. U.S. Environmental Protection Agency, Washington, DC, USA.

Will, M.E., Suter, II G.W. 1995. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1995 Revision. Oak Ridge, U.S. Department of Energy. ES/ER/TM-85/R2.

Zalaski, R., Gephart, L., 1999. NICOLE Exposure Factors Sourcebook for European Populations, with focus on UK Data.