RISK BASED AND SITE-SPECIFIC DOMESTIC USE WATER QUALITY GUIDELINES

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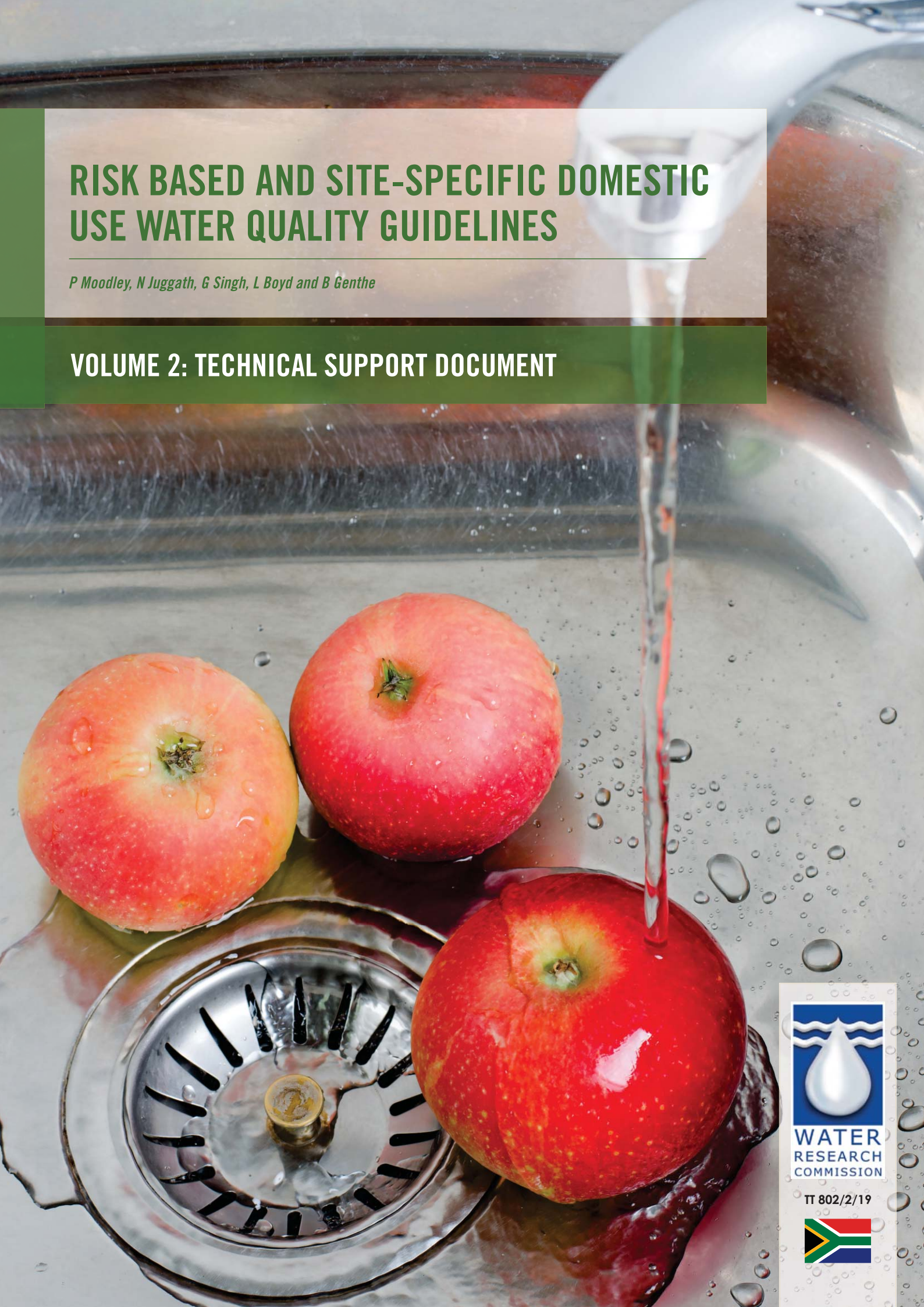
VOLUME 2: TECHNICAL SUPPORT DOCUMENT



WATER RESEARCH COMMISSION

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VOLUME 2: TECHNICAL SUPPORT DOCUMENT

Report to the
Water Research Commission

by

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EXECUTIVE SUMMARY

BACKGROUND

The ultimate objective of drinking water quality guidelines (human consumption) is the protection of public health. Therefore, every effort needs to be taken to ensure that water intended for human consumption is safe to use. Water quality guidelines for the domestic environment are also necessary to ensure that water is suitable for non-consumptive uses such as bathing and household use. Domestic water quality guidelines relate to the concentrations of physical, chemical or microbiological contaminants in water, allowable for consumption by humans during their lifetime, without the risk of significant health effects, or for non-consumptive uses without significant economic and physical effects. Guidelines for some contaminants are set to be protective for susceptible subpopulations, but these guidelines are also protective of the general population over a lifetime (DWAF, 2008). A water quality guideline is a recommended numerical concentration level (e.g. of a contaminant) or a descriptive statement (e.g. visual appearance of a water body) that will support and maintain the designated use of a particular water.

The most effective means of ensuring safe drinking (and domestic) water supply should encompass a risk management approach at all steps in the water supply chain, from catchment to consumer. The Framework for Safe Drinking water included in the WHO Drinking Water Guidelines (2017) adopts such a risk management approach by means of Water Safety Plans (WSPs). The WSP adopts the multi barrier approach, hazard and risk assessment and control measures as the key principles and concepts of risk management in protecting drinking water. The WHO guidelines include operational recommendations that focus on source water protection and adequate treatment in ensuring good and safe water supply. In South Africa, the WHO WSP approach to the delivery of safe water supply to domestic users has been adopted. This is included as a component of the Blue Drop Certification process. This endeavour would serve as a supporting risk management tool to assess the quality of domestic water supply. It aims to provide a risk-based decision framework where possible, to help the user refine guideline trigger values for local, regional and or catchment uses, and inform actions to address water quality risks and assess performance.

The 1996 South African Water Quality Guidelines (Volume 1) (SAWQGs) has been used by water quality managers and water resource managers as a primary source for decision-making to judge the fitness for use of water for domestic use. The guidelines are essentially a user needs specification of the quality of water required for drinking, food and beverage preparation, bathing and person hygiene, laundry, household washing (dishes), hot water systems and for gardening in the domestic environment. Target Water Quality Ranges per constituent are presented by assuming lifelong exposure and incorporate a margin of safety. The target water quality ranges were set as equal to the no-effect range which is defined as the concentration at which the presence of the constituent would have no known or anticipated effect on the fitness of the water for the domestic user/consumer. It reflected the scientific thinking at the time it was produced. Subsequently, the decision support function of water quality guidance has grown and become more complex. Increased scientific understanding of the complexity of water ecosystems and adaptive catchment management processes has led to new ways of managing water quality.

Traditional scientific and management approaches may not deal well with contemporary water quality issues. In their place, holistic, best-practice approaches need to be taken to ensure that water resources are managed sustainably. In 2007 a number of specific issues came to the fore that made it necessary to re-examine the philosophical basis used for determining and applying the water quality guidelines. These included *inter alia* the classification of water resources and Reserve determination under the National Water Act

(Act No. 36 of 1998), the concept of risk as potential common basis for decision making in various contexts, site specificity, advancements in guideline determination internationally and the need to include additional water quality constituents. Additional factors that have influenced the optimal use of the SAWQGs include the misapplication of the guidelines (e.g. guideline values are used interchangeably) or confusion in interpretation of terminology (e.g. guidelines versus standards).

Since the evolvement of water resource management within South Africa, the water quality guidelines have become decision support tools rather than a list with numbers. In 2008 the then Department of Water Affairs (DWA) undertook an investigation on the need for the review of the 1996 version of the SAWQGs Fresh Water series, specifically on how guidelines are applied. The outcomes of this investigation highlighted the necessity to review the water quality guidelines and the significance of producing a software decision tool to support the decision processes relating to the assessment of fitness for use and numerical water quality objective setting in, primarily, fresh water resources. The review included among other recommendations, that the water quality guidelines should support site specificity, be risked based, provide for a tiered fitness for use assessment and consider a software-based decision support tool. In light of these recommendations the Water Research Commission (WRC) initiated an overarching project that has seen the commissioning of a series of projects to develop risk-based decision support tools per water user group.

This project addresses the 'Development of a Risk based Methodology and Decision Support System for Domestic Water Use" as part of the series. The project objective was to develop a risk-based methodology for determining water quality guidelines for domestic use enabled through a user-friendly and practical decision support system (DSS). The specific aspects that have been addressed in terms of meeting this objective include firstly, the development of the approach and methodology for the risk calculations based on supporting science to be included in the technology demonstrator; and secondly the development of the informatics for a demonstrator decision support system that addresses the main decision contexts for the use of the guidelines. The intention is that the guidelines will no longer represent a simple pass-fail number, which ignored spatial and temporal variability. The risk science and the approach adopted for the domestic user water quality guidelines considers a combination of qualitative and quantitative risk assessment. At the core of the guidelines is a quantified risk estimate (probability of a risk), which is assessed in terms of threshold criteria that relates to fitness for use categories or water quality requirements.

APPROACH

The approach undertaken for the guideline development has incorporated the concepts of risk and site specificity into the methodology to provide the risk-based water quality guidance to the user. Based on the selected domestic use type, risk quantification is applied as a basis to the assessment of fitness for use, accounting for the nature of the water resource and the nature of the water user as the site specificity components. The ability of the user to provide some water quality input to the risk assessment process and the flexibility to select the domestic water use type and adjust the receptor information, supported the requirement of presenting the guidelines as a software product rather than a static document.

Risk

In the course of deriving the guidelines the risk refers to the probability of the adverse/undesired effects to the domestic user of using water containing a potential hazard, including the severity of the consequences. The hazard in this context refers to a range of water quality constituents that may be present in the water that renders it less fit for use, and its consequences based on the how the water is to be used within the domestic environment. Thus, risk is a function of hazard and exposure. Where *hazard* = biological, chemical or radiological agent that has the potential to cause harm, *hazard effect* = adverse impact on human

health/appliances/household items that can result from exposure to a substance and *exposure* = contact between a substance and an individual or a population. The threat caused by a hazard depends not only on the severity of its effect but also on whether or not the effect is reversible.

Acceptable Risk

Risk is generally taken to be the probability of injury, disease, or death under specific circumstances (WHO, 2001). Acceptable risk decisions are rarely easy. The subject of what constitutes an acceptable risk is an extremely complex issue and must be handled from a policy perspective. In determining acceptability, it is however largely the perceived risk that determines the basis of what can be tolerated. Acceptable risk is very location-specific, and in some cases culturally specific. For this reason it plays an important role in adapting guidelines to suit local circumstances, where local stakeholder involvement and available data is vital. For purposes of the risk based guidelines acceptable risk applied includes internationally applied risk levels derived from the probability approach, the tolerated approach and disease burden approach.

Site specificity

The site-specific components of the risk-based methodology relates to what and how the water is exposed to the domestic user. This considers the composition of the water quality that is entered as an input (constituents and concentrations), the selected the conditions of the exposure (duration, volume, route) and the characteristics of the receptor (e.g. human – age, body weight). The risk based water quality guidance provided by the decision support system is a combination of the intrinsic risk of the water quality constituent (the hazards, its toxicity and known adverse effects) and the extrinsic risk of the nature of the water and the nature of the water user (route of exposure, receptor, exposure conditions) that is derived through a mathematical approach (calculation methodology) comprising the risk assessment that is reported as a fitness for use.

Risk estimation

The building blocks comprising the risk approach and how these are applied in the deriving the domestic use water quality guidelines is as follows:

- ***Selection of the water use type*** – The description of the exposure scenarios for the domestic user on how the water may be encountered. This comprised the types of domestic water use typically encountered in the domestic environment. This was guided by the 1996 SAWQGs Volume 1, working committee and the reference group. The domestic use categories incorporate what the water is to be used for:
    - Drinking;
    - Food and beverage preparation;
    - Bathing and personal hygiene use;
    - Laundry;
    - Household washing;
    - Appliances/Plumbing;
    - Gardening; and
    - Pour flushing.

Depending on the domestic use type and nature of the exposure, the adverse effects of using less than target water quality may manifest as either human health, aesthetic quality and/or a physical risk. The identification of whether a water quality constituent presented as a human health, aesthetic quality and/or a physical risk to a domestic user directed the calculation methodology of how the risk is assessed.

- ***Hazard identification (water quality constituents)*** – Hazard identification (in this context the water quality constituent), in which a determination is made as to whether a water quality constituent has the potential to cause harm to human health, and/or potential to result in physical effects to property and/or potential to reduce the desirability (aesthetics). It is the process of determining whether exposure to the constituent in question can cause an increase in the incidence of specific adverse health effects (e.g. cancer, birth defects). It is also whether the adverse health effect is likely to occur in humans. Hazards include microbial, chemical, physical and radiological agents. Assessment of the chemical, microbial and physical water quality constituents, their characterisation of effects and how they are experienced based on the exposure conditions was undertaken. The characterisation of the hazard risk (effect of the water quality constituent) comprised largely of the review and interrogation of the available literature of risk, exposure and toxicological assessment data to determine the individual adverse effect end pints. This relied on dose assessment relationships (examination of exposure and effects) and exposure assessments in which what is known about the frequency, timings, magnitude, and levels of contact with the hazard is examined. This was a fundamental component to the risk-based guideline development process. For the purposes of the development of a technology demonstrator the range of water quality constituents (hazards) addressed were limited to 50 constituents, comprising the different types of hazards. These hazard types dictate how risk is calculated, and include the following:
    - Carcinogens (non-threshold – those that do not appear to have a threshold)
    - Toxicants (effects are observed only above a certain threshold dose, with no effects observed at doses below this threshold even with lifetime exposure)
    - Infectious agents (microbiological – disease burden quantification)
    - Physical properties (aesthetic acceptability and physical damage); and
    - Chemical properties (damage to subsistence garden crops).

- ***Quantification of the Risk –*** The probability of occurrence of the risk (risk estimate) is derived through a mathematical approach (calculation methodology) comprising the risk assessment. The risk estimate that is obtained as an output, provides guidance about the nature and extent of the risk from exposure to the water quality constituent. Risk is assessed based on the computation of the following components:
    - The exposure scenario – domestic use type:
        - Selection of domestic use category
    - The water quality constituent:
        - The water quality constituent (s) of interest selected (hazard).
    - The assessment conditions (site specific components):
        - The water quality composition (concentrations)
        - The exposure route (how)
        - The receptors (human/ physical)
        - The exposure conditions (magnitude, duration, frequency and volume)
        - Human health, Physical or Aesthetic (effect end points).

Water quality composition: Input of water quality analysis data (once off or recorded time series data – however a single data input is not preferred)

Exposure Route: The exposure route means of entry of the hazard. The exposure route is generally further described as intake (as eating, drinking, or inhaling) or uptake (absorption through skin or eye) on contact. Five pathways are assessed operable for each receptor identified.

- ingestion
- inhalation
- dermal
- aesthetic acceptability (contact)
- physical/chemical contact household items/objects, gardening crops

The Receptor: In assessing risk, exposure assessment is the process of estimating the exposure of a human receptor/situation to a substance under a given scenario. The most susceptible receptor varies depending on the expected water use. Three receptors are considered with respect to domestic use:

- humans (health and aesthetics)
- household items/plumbing/appliances, laundry
- subsistence crops

The Exposure conditions: considers aspects such frequency, duration, magnitude and levels of contact of the receptor with the hazard (water quality constituent). Default exposure conditions applied to the specification of the generic water quality requirements and a range of pre-defined exposure conditions are included for the fitness for use assessments. The user is presented with the functionality in the fitness for use assessment to adjust these based on the site-specific circumstances or as an option to revise the calculation algorithm.

The Calculations: Quantification of the risk incorporates six calculations which are dependent on whether the risks are human health, aesthetic or physical associated adverse effects. Each of which is a mathematical formula that is run. The health-related acceptable risk values (for chemicals) are conservative, incorporate a range of safety factors and are based on reference toxicological data. Characterization of the hazard as either a threshold (toxicant) or non-threshold (carcinogen) chemical is important as different approaches are used for the quantification of the risk estimate. The use of threshold criteria has been applied to quantifying risk of the aesthetic and physical effects of the chemical constituents based on exposure and threshold tolerance levels for each. The health risk associated pathogenic bacteria, viruses and parasites present in water for domestic use is determined by a quantitative microbial risk assessment (QMRA), an approach adopted by the World Health Organisation in the Drinking Water Quality Guidelines (WHO, 2017). The QMRA provides an estimate of the probability of infection based on the number of pathogens ingested (dose). The QMRA is the adopted calculation methodology.

The six calculations that are applied based on the routes of exposure and type of hazard are as follows:

Calculation 1 – Calculations associated with ingestion of water:

- Chemical – Toxicant
- Chemical – Carcinogen
- Microbiological – Infectious agent

Calculation 2 – Calculations associated with inhalation
Calculation 3 – Calculations associated with dermal exposure
Calculation 4 – Calculations associated with physical effects
Calculation 5 – Calculations associated with aesthetic acceptability
Calculation 6 – Calculations associated with gardening.

For the purposes of the domestic use risk-based water quality guidelines, the risk to the domestic user who relies on subsistence crops is included at a reference level as generic risk-based water quality requirement.

The domestic use guidelines have adopted the generic fitness for use criteria of the Irrigation Risk Based Water Quality Guidelines (conservative limits). For further risk-based site-specific guidance the user is directed to the Irrigation Risk Based Water Quality Guidelines (Du Plessis *et al.*, 2017).

DECISION SUPPORT SYSTEM (DSS)

A software-based decision support system (DSS) offers the advantage to improve the way in which the guidelines are used because the focus is directly on supporting decisions in specific contexts; by applying the supporting science rather than producing simple numeric guidance. The DSS provides a structured approach necessary for assessing fitness for use and determining water quality requirements based on a qualitative or quantitative risk assessment. The DSS is done through a software demonstrator/ prototype system for the purposes of this project using MS Excel as the user platform. A three-tiered system is defined. Each tier provides an output that has to comply with the applicable level of risk assessment. The difference between the tiers lies primarily in the degree of site-specificity required to produce an output. The DSS has been designed to assess:

- A quantitative risk – as a percentage probability of occurrence of the adverse effect, or as
- A qualitative risk – reported as a water quality requirement based on the risk threshold criteria at which the adverse effect is expected to manifest.

The risk-based water quality guidelines for domestic use is presented as a software decision support tool and includes a tiered system of assessment which operates at two levels of functionality. The difference between the levels lies primarily in the degree of site-specificity required to produce an output. The assessment system accommodates for the needs of the novice, intermediate and expert user respectively includes three tiers. The following definition of the assessment levels informs the basis of design for the DSS informatics.

Fitness for Use		Water Quality requirement
Site specific		Generic
Tier 3	**Tier 2**	**Tier 1**
The most site-specific guidance. A risk assessment protocol, requiring highly skilled input and output interpretation. Allows for the adjustment of the algorithm and reference data. Default site specific component options that can be changed to suit site specific circumstances (more specific models and parameters). Functionality/permissions to adjust the calculation methodologies, reference databases and algorithms to provide the detailed site-specific risk quantification for the scenario.	Moderately site-specific, requiring some skills, but largely uses pre-defined water use scenarios and limited site characterisation choices with common field observation and or measurement input required from the user for scenarios manipulation. Rule-based output interpretation. Calculations are specific to the domestic water use categories and are based on the detail of the site-specific information entered	Most generic (and by implication the most conservative) approach to risk guidance. Minimum user input required and simple output provided. Simplified generic conservative assumptions used and totally reliant on the default datasets (worst case exposure). Does not involve rigorous calculation methodology.
Output: Presentation of the adjusted risk estimate (probability of occurrence) and associated fitness for use based on the revised site-specific exposure scenarios and methodology.	*Output:* Presentation of the risk estimate (probability of occurrence) and associated fitness for use based on the water quality input and selection of the pre-defined exposure scenarios.	*Output:* Descriptions and risk-based thresholds of levels of water quality requirements (most conservative and generic) per domestic use category.

The generic risk-based water quality guidelines are reported as water quality requirements, as no site-specific components are inputted, the system reports on what would be the required water quality for an intended type of domestic use. The DSS produces risk-based water quality guidance at two levels, either:

- as a water quality requirement, *i.e*. generic conservative threshold risk criteria per constituent for a selected domestic use category, or
- as a quantified risk estimate of fitness for use expressed for a selected domestic use category based on an input and selected exposure conditions.

Figure E1 depicts the overall structure of the DSS.

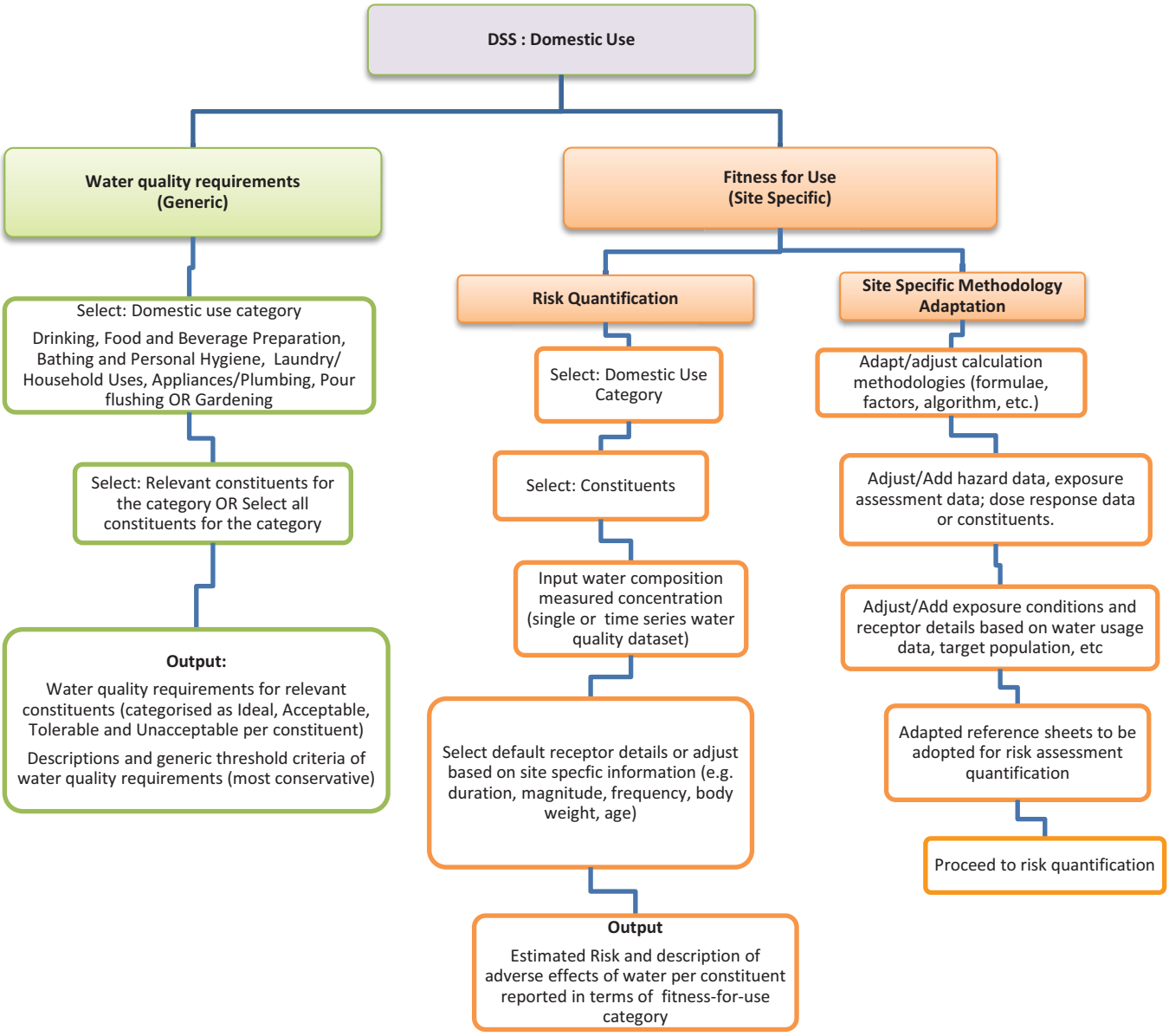

Figure E1: Functional Structure of the DSS

A ‘risk-based guideline’ (the probability of adverse effect occurring) is generated based on the computation of the following hazard and exposure input parameters through the algorithm for the assessment levels run in the DSS:

For Generic (Water Quality Requirement – threshold risk criteria):

- selected domestic use category;
- selected of water quality constituent(s) of interest;

For Site Specific (Fitness for Use – risk estimate):

- selected domestic use category;
- selected of water quality constituent(s) of interest;
- entry of water quality input (water composition either as a single entry or a time series);
- Selection of exposure scenarios (receptor, route, duration, magnitude, frequency).
- *Further for expert site specific*
- functionality to revise the algorithms, methodology and reference databases (and rerun as above).

The two assessment levels of water quality guidance and as well option to adjust the risk calculation methodology is described below:

RISK REPORTING – RISK BASED WATER QUALITY GUIDELINES

“Risk based” guidelines simply allow the suitability of the water to be interpreted in terms of risk of specific adverse effects. The DSS reports on the risk of the likelihood of adverse effects that may be experienced when using the water for a domestic use in a given context. Water quality is therefore expressed in terms of the likelihood of the potential risk. A colour coded generic fitness for use categorization system and a quantified risk estimate (as a percentage) is reported in the DSS as the potential risk. The DSS uses a two-type reporting system, either a four category or two category system which is dependent on the selected water quality constituent(s). The two-category system is applied to carcinogens and microbial infectious agents and reports a fitness for use either as (1) above or (2) below an acceptable risk target. The four-category system is applied to toxicants and physical and aesthetic constituents and reports the fitness for use as (1) ideal, (2) acceptable, (3) tolerable or (4) unacceptable. This is aligned with the standard practice within DWS (and in line with the irrigation and recreational risk-based water quality guidelines recently developed).

The four-category system is in harmony with a risk-based assessment of water quality in that the ‘Ideal’ category represents a no risk scenario (safe level), while the ‘Unacceptable’ category represents a high-risk scenario (likely presence of the adverse effects). The two-category system is aligned to the WHO (2017a) health-based target guidelines that is based on the health outcome type. The first outcome considers the burden of disease associated with different water-related hazards, taking into account varying probabilities, severities and duration of effects, and uses the disability-adjusted life years (DALY) tolerable burden of disease target as the metric. The second outcome considers the incidence of cancer and includes an acceptable risk target level (no adverse effect or negligible risk). This fitness for use categorisation represented by the colour scheme is shown in Table E3 and Table E4. The same colour scheme is also used throughout the DSS to depict the fitness for use based on risk.

Table E3: A generic description of the fitness for use categories used for risk reporting

Reported Category	Description
Ideal	A water quality fit for a lifetime of use.
Acceptable	A water quality that would exhibit minimal impairment to the fitness of the water for its intended use. No observed adverse effects.
Tolerable	A water quality that would exhibit some impairment to the fitness of the water for its intended use. Minor risk of adverse effects presenting themselves.
Unacceptable	A water quality that would exhibit unacceptable impairment to the fitness of the water for its intended use. Significant risk of adverse effects, presenting themselves.

Table E4: A generic description of the of the fitness for use categories used for tolerable burden of disease or cancer risk reporting

Reported Category	Description
Below acceptable risk target	< the upper limit target DALY tolerable burden of disease < the acceptable risk for cancer
Above acceptable risk target	> the upper limit target DALY tolerable burden of disease > acceptable risk for cancer

The categorisation is based on threshold risk criteria as obtained from scientific literature and risk databases that include exposure assessment data for each constituent or on acceptable risk levels.
The risk-based water quality guidelines are reported at two levels based on whether the user selects generic or site specific (input based) guidance, as follows:

- ***For Water Quality Requirements (generic)*:** The DSS report screen reports *all* risk threshold criteria and associated fitness for use levels (*i.e*. ideal, acceptable, tolerable or unacceptable) for the specific constituent(s) selected.
- ***For Fitness for Use (site specific)***: The DSS report screen reports only the fitness for use category within which the quantified risk estimates falls (ideal OR acceptable OR tolerable OR unacceptable OR >DALY OR <DALY) together with the risk estimate value, the exposure concentration of the specific constituent and the description of the associated adverse effect end points.

Threshold limit criteria (for toxicants, physical and aesthetic constituents) are applied to each category and represent how the adverse effects and likelihood of occurrence of the risk are linked to the fitness for use each category. A risk estimate (as a percentage) has been defined based on these threshold limit criteria and represents the probability of occurrence and severity of adverse effect as follows:

Risk estimate (Percentage)	Probability of Occurrence	Severity of the effect
<1	None	None
1-5	Rare	Negligible
>5-15	Possible	Minor
>15-100	Certain	Significant

CONCLUSION AND RECOMMENDATIONS

The project aim was successfully achieved, with the DSS as a product fulfilling the requirements of the technology demonstrator for risk based domestic use water quality guidelines. However, the following is required and recommended to develop the product further to a fully functional system to be utilised within the water resource management sector in South Africa:

- The further development of the domestic user DSS methodology in the next phases would need to address:
  - The functionality of the water quality objective setting at Tier 2;
  - Expansion of the water quality constituent database to include all constituents relevant to domestic use; specifically, within the South African context;
  - The consideration of synergistic and antagonistic effects of constituents and expansion of the calculation methodology to address this;
  - The update of the methodology to include the assessment of multiple constituents simultaneously;
  - Endpoint confirmation of all hazards;
  - The incorporation of local domestic water use pattern information where applicable to improve site specificity, calculation methodology and receptor information;
  - Processes and procedures for the updating of the methodologies and exposure assessment data, based on the best available science information as it becomes available;
  - Functionality that allows export of water quality monitoring data from national and local monitoring programmes directly into the DSS;
  - A structured procedure applicable to Tier 3 users should be developed to control and maintain the original product while providing the user with a clear method of detailed analysis; and
  - Currently the DSS tool has been demonstrated using MS Excel, however in going forward to full scale application, it is recommended that available on-line databases be tested to select a software suitable for the DSS for the guideline series.
- Wider stakeholder buy-in and guidance is required to gain acceptance of the risk-based approach for the assessment of water quality. Users may be hesitant to want to take decisions on the basis of a risk quantification that the DSS provides, without requisite understanding of the support it is meant to provide. More engagement is required to get users to accept the philosophy and approach;
- Further testing with the wider stakeholder user groups is required to refine the product and to update the DSS to improve user-friendliness and utility, based on feedback from users.
- A DSS tool that is available through an on-line platform is recommended.
- Next phases of the project require the integration with the user guidelines that needs to consider the selection of coding platform, intellectual property issues, controlled access to software system, version controls as well as processes and procedures on the updating of the methodologies and functionality of the DSS for the water user groups.
- Such a system places stringent demands on the custodianship of the product. An owner and champion within the DWS are required to spearhead the next phases of the DSS, its integration, its promotion and maintenance.

The development of risk-based approach and a technology demonstrator DSS for domestic water quality guidelines was a challenging undertaking, requiring a shift in thinking and approach and innovation in conceptualisation and development. It however proved to be exciting and forward thinking, with the resultant DSS product presenting a novel and revolutionary manner of how domestic water quality may be expressed in supporting the multifaceted dimensions and complexities to water quality management decision making in South Africa.

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GLOSSARY

Acceptable daily intake (ADI): Estimated maximum amount of a chemical, biological or physical agent, expressed on a body mass basis, to which individuals in a (sub)-population may be exposed daily over their lifetimes without appreciable health risk. Related terms: Reference dose, Tolerable daily intake. Usually expressed as milligram/kilogram of body weight/day.

Acceptable risk: Used in risk management to reflect the highest risk that can be tolerated for the specified end-point and target population. It depends on scientific data, social, economic, and political factors, and the perceived benefits arising from exposure to an agent.

Adverse effect: Change in the morphology, physiology, growth, development, reproduction, or life span of an organism, system, or (sub)-population that results in an impairment. Equivalently, an undesirable response of a receptor-effector mechanism.

Benchmark Dose (BMD): A dose that produces a predetermined change in response rate of an adverse effect (called the benchmark response or BMR) compared to background.

Cancer: A disease in which altered cells (mutations) divide uncontrollably (neoplastic growth) resulting in tumours (neoplasms) that may be benign (inert) or malignant (proliferate). Common types of cancer include: Leukaemias (white blood cells and derived tissues), Lymphomas (lymphatic system), Sarcomas (connective tissue) and Carcinomas (epithelial tissues).

Carcinogenicity: The extent to which a substance can cause cancer.

Concentration: Amount of a material or agent dissolved or contained in unit quantity in a given medium or system

Dosage: Amount of toxicant per unit of animal (organism) mass or weight, or weight per unit of time (*e.g.* 2 mg/kg/day). Can also incorporate frequency (*e.g.* 2 mg/kg/day for 2 years).

Dose: Total amount of an agent administered to, taken up by, or absorbed by an organism, system, or (sub)-population.

Dose-response: The response that manifest in an organism, system, or (sub)population caused by an amount of an agent (the dose) administered to, taken up by, or absorbed by the organism, system, or (sub)population. *Related terms*. Dose-effect, Concentration-effect.

Dose-response assessment. Dose-response assessment examines the relationship between exposure and effects, and inferences derived from such an analysis with respect to the entire population. Dose-response assessment is the second of four steps in risk assessment.
Dose-response curve: Graphical presentation of a dose-response relationship.

Dose-response relationship: The numerical relationship between the administered dose (exposure) and the response it causes (effects).

Effect: Change developed in the state or dynamics of an organism, system, or (sub)-population in reaction to exposure to an agent. (Synonymous with response.)

Exposure: Concentration or amount of a particular agent that reaches a target organism or (sub)-population with a specific frequency and defined duration.

Exposure assessment: Exposure assessment examines what is known about the frequency, timing, and levels of contact with a stressor.

Exposure duration: The length of time an organism is exposed to a chemical.

Exposure scenario: A set of conditions or assumptions about sources, exposure pathways, amounts or concentrations of agent(s) involved, and exposed organism or(sub)population (*i.e.* numbers, characteristics, habits) used to aid in the evaluation and quantification of exposure(s) in a given situation.

Fitness for use: A scientific judgement, involving objective evaluation of available evidence, of how suitable the quality of water is for its intended use or for protecting the health of aquatic ecosystems.

Hazard: Inherent property of an agent or situation having the potential to cause adverse effects when an organism, system, or (sub)-population is exposed to that agent.

Hazard assessment: A process designed to determine the possible adverse effects of an agent or situation to which an organism, system, or (sub) population could be exposed. The process includes hazard identification and hazard characterisation, the first two of four steps in risk assessment.

Hazard characterisation: The qualitative and, wherever possible, quantitative description of the inherent property of an agent or situation having the potential to cause adverse effects. This should, where possible, include a dose-response assessment and its attendant uncertainties. Hazard characterisation is the second of two steps in hazard assessment and the second of four steps in risk assessment.

Hazard identification: Examines whether an agent has the potential to cause harm to humans and/or ecological systems, and if so, under what circumstances. The identification of the type and nature of adverse effects that an agent has an inherent capacity to cause. Hazard identification is the first of two steps in hazard assessment and the first of four steps in risk assessment.

Lowest Observed Adverse Effect Level (LOAEL): The lowest dose at which a statistically significant adverse effect (frequency or severity) could be found.

No Observed Adverse Effect Level (NOAEL): The highest exposure level (dose) at which there are no significant increases in the frequency or severity of adverse effects in the exposed population. Some effects may be produced at this level, but they are not considered adverse or precursors of adverse effects.

Reference dose (RfD): An estimate of the maximum daily exposure dose that is likely to be without deleterious effect even if continued exposure occurs over a lifetime. *Related term*. Acceptable Daily Intake (ADI). The ADI is based on NOAELs with ADIs significantly lower than NOAELs (due to safety factors). The reference dose is a surrogate for the ADI and may be even lower than the ADI as it can use safety factors for hypersensitivity, individual variation, extrapolation from animals to humans during experimentation. Reference doses are therefore actually based on NOAELs and used to quantify noncarcinogenic risk.

Reference concentration (RfC): An estimate of the continuous maximum inhalation exposure to the human population that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used. Generally used in used to quantify noncarcinogenic risk.

Risk: The probability of an adverse effect in an organism or (sub)-population caused under specified circumstances by exposure to an agent.

Risk assessment: The process to estimate the nature and probability of adverse health effects in humans who may be exposed to chemicals in contaminated water, now or in the future. Taking account of the inherent characteristics of the agent of concern as well as the characteristics of the specific target receptor. Risk assessment provides information on potential health or ecological risks. The risk assessment process includes four steps: hazard identification, hazard characterisation, exposure assessment, and risk characterisation.

Risk characterisation: Risk characterization examines how well the data support conclusions about the nature and extent of the risk from exposure to environmental hazards. Risk characterisation is the last of four steps in risk assessment.

Risk estimation: Quantification of the probability, that specific adverse effects will occur in an organism, or (sub)-population due to actual or predicted exposure.

Risk management: The decision-making process based on the quantitative value obtained from risk assessment models coupled with insight, experience, and judgment. Forms an integral part of risk communication.

Safety factor: Composite (reductive) factor by which an observed or estimated No Observed Adverse Effect Level (NOAEL) is divided to arrive at a criterion or standard that is considered safe or without appreciable risk. The value used is typically a management or policy decision and is usually somewhat subjective. *Related terms*. Assessment factor, Uncertainty factor.

Slope factor: The slope of the linear extrapolation to the origin. An upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate is generally reserved for use in the low-dose region of the dose-response relationship that is for exposures corresponding to risks less than 1 in 100.

Stressor: A stressor is any physical, chemical, or biological entity that can induce an adverse response.

Target water quality range: Defined for the 1996 South African water quality guidelines as the concentration range of a water quality constituent for which no (adverse) effects are observed.

Threshold dose: The highest dose of a toxicant at which toxic effects are not observed.

Tolerable daily intake (TDI): An estimate of the amount of a substance drinking-water, expressed on a body weight basis (milligram or microgram per kilogram of body weight), that can be ingested over a lifetime without appreciable health risk, and with a margin of safety. The term “tolerable” is used for agents that are not deliberately added, such as contaminants water. TDI signifies permissibility rather than acceptability.

Toxicant: A chemical substance capable of exhibiting a toxic effect.

Uncertainty: Uncertainty refers to our inability to know for sure – it is often due to incomplete data. For example, when assessing the potential for risks to people, toxicology studies generally involve dosing of sexually mature test animals such as rats as a surrogate for humans. Since we don't really know how differently humans and rats respond, risk assessment often employs the use of an uncertainty factor to account for possible differences. Additional consideration may also be made if there is some reason to believe that the very young are more susceptible than adults, or if key toxicology studies are not available.

Uncertainty factor (UFs): One of several, generally 10-fold, default factors used in operationally deriving the Reference dose and Reference concentration from experimental data. The factors are intended to account for (1) variation in susceptibility among the members of the human population (i.e. inter-individual or intraspecies variability); (2) uncertainty in extrapolating animal data to humans (i.e. interspecies uncertainty); (3) uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure (i.e. extrapolating from subchronic to chronic exposure); (4) uncertainty in extrapolating from a LOAEL rather than from a NOAEL; and (5) uncertainty associated with extrapolation when the database is incomplete.

Variability: Refers to the range of toxic response or exposure. For example, the dose that might cause a toxic response can vary from one person to the next depending on factors such as genetic differences, pre-existing medical conditions, etc. Exposure may vary from one person to the next depending on factors such as where one works, time spent indoors or out, where one lives, how much people eat or drink, etc.

Water quality: The physical, chemical, radiological, toxicological, microbiological, biological and aesthetic properties of water that (1) determine its fitness for use or (2) that are necessary for protecting the health of aquatic ecosystems. Water quality is therefore reflected in (a) concentrations of substances (either dissolved or suspended) or microorganisms, (b) physico-chemical attributes (*e.g.* temperature), (c) levels of radioactivity and (d) biological responses to those concentrations, physico-chemical attributes or radioactivity.

Water quality constituent: Any of the properties of water and / or substances dissolved or suspended in the water. The term constituent is used interchangeably with variable, determinand or characteristic.

Water quality criteria: Numerical and qualitative descriptors for a given water quality constituent describing its potential effects on selected endpoints and water users.

Water quality guideline: A recommended numerical concentration level (e.g. of a constituent) (quantitative measure) or a descriptive statement (e.g. visual appearance of a water body) that will support and maintain the designated use of a particular water. Water quality guidelines are provided for chemical and physical constituents of water, as well as biological indicators.

CHAPTER 1: BACKGROUND

1.1 INTRODUCTION

The National Water Act (Act No.36 of 1998) emphasises on the need to protect our fresh water ecosystems, which are under threat because of pollution from many sources. Being a water scarce country, water resources in South Africa requires careful management in order to enable provision of basic water services and equitable allocation, while meeting the needs of inclusive economic growth without threatening the integrity of the aquatic ecosystem. Though the concept of risk management in the context of water resource management is not explicitly stipulated in the National Water Act (Act No.36 of 1998), its role in supporting decision making with regards to resource classification and fitness for use is well recognised. While there is no legal obligation requiring the use of risk approaches or risk science in water resources management, the concept of risk offers a scientifically tenable approach to assess impact of different qualities of water. Consistent with global practice, the concept of risk has been used as basis for the development of South African Water Quality Guidelines (SAWQGs) (Jooste and Claassen, 2001). As such, the 1996 SAWQG series have been used by water quality managers and water resource managers as a primary source for decision-making to judge the fitness for use of water for different purposes.

In 2007 a number of specific issues came to the fore that made it necessary to re-examine the philosophical basis used for determining and applying the water quality guidelines. These included *inter alia* the classification of water resources and Reserve determination under the National Water Act (Act No. 36 of 1998), the concept of risk as potential common basis for decision making in various contexts, site specificity, advancements in guideline determination internationally and the need to include additional water quality constituents. Additional factors that have influenced the optimal use of the SAWQGs include the misapplication of the guidelines (e.g. guideline values are used interchangeably) or confusion in interpretation of terminology (e.g. guidelines versus standards). In 2008 the then Department of Water Affairs (DWA) undertook a formal study to review approach used in the 1996 version of the SAWQGs. The outcomes of this investigation supported the view and need to revise the approach and the significance of producing a software decision tool to support the decision processes relating to the assessment of fitness for use and numerical water quality objective setting in, primarily, fresh water resources.

In addition, it was recommended that the revised water quality guidelines should support site specificity, be risked based, and provide for a tiered risk assessment approach through a software-based decision support tool. In light of these recommendations the Water Research Commission (WRC) initiated an overarching project that has seen the commissioning of a series of projects to revise the approach in order to guide the development of risk-based and site-specific water quality guidelines and decision support tools for different water use groups. Outcomes of the review undertaken by the Department of Water and Sanitation (DWS) in 2008 (the then Department of Water Affairs) were used as basis for developing the Terms of Reference of the WRC projects. The proposed change in the current guidelines is that both the fitness for use and water quality requirement assessment now relates to risk, which combines hazard and exposure, rather than the hazard predominantly, as applied in the 1996 guidelines. In addition, the revised risk assessment approach should take into account the specific water use/requirement context. This project addresses the ‘Development of a Risk based Methodology and Decision Support System for Domestic Water Use” as part of the series.

1.2 PROJECT AIM AND OBJECTIVES

The main aim of this project was to develop a risk-based methodology for determining water quality guidelines for domestic use enabled through a user-friendly and practical decision support system (DSS). The specific aspects that have been addressed in terms of meeting this objective include firstly, the development of the approach and methodology for the risk calculations based on supporting science to be included in the technology demonstrator; and secondly the development of the informatics for a demonstrator decision support system that addresses the main decision contexts for the use of the guidelines. The following were the aims of the project:

1. To establish a working committee and international review panel whose function is to provide expert advice during the guideline development process
2. To carry out a systematic review of relevant literature to identify and critically appraise best available evidence.
3. To develop an intermediate technology demonstrator that demonstrates the most important features of risk estimation and reporting
4. To engage with stakeholders to elicit comment and recommendations and maximise synergy with parallel projects on the development of water quality guidelines for other water users.
5. To develop a fully-functioning decision support system for domestic water use

1.3 SCOPE AND LIMITATIONS

The intention is that the revised guidelines will no longer represent a simple pass-fail number, which ignored spatial and temporal variability. The risk science and the approach adopted for the domestic user water quality guidelines considers a combination of qualitative and quantitative risk assessment. At the core of the guidelines is a quantified risk estimate (probability of a risk), which is assessed in terms of threshold criteria that relates to fitness for use categories or water quality requirements. The overall project objective is to develop risk-based and site-specific water quality guidelines for domestic use derived through a user-friendly decision support system. A software-based decision support system (DSS) offers the advantage to improve the way in which the guidelines are used because the focus is directly on supporting decisions in specific contexts; by applying the supporting science rather than producing simple numeric guidance. In addition, the DSS provides a structured approach necessary for assessing fitness for use and determining water quality requirements based on a qualitative or quantitative risk assessment. The DSS caters for risk assessment through a three-tiered approach. Each tier provides an output that has to comply with the applicable level of risk assessment. The difference between the tiers lies primarily in the degree of site-specificity required to produce an output.

The water quality constituents to be included in the technology demonstrator include the suite of constituents comprising the 1996 Domestic Use Water Quality Guidelines (Volume 1), the relevant constituents included in the current South African Drinking Water Quality Standards (SANS 241:2015) which were not included in the 1996 edition, and other relevant constituents adopted from the 4th edition of the WHO Drinking Water Quality Guidelines (WHO, 2011). The fact sheets for each of the constituents considered in this edition are included in this report. The main consideration for selection of constituent's inclusion was dependant on the availability of toxicological assessment and empirical data, which was required to support the risk assessment. However, constituent lists in the DSS may be easily expanded in future and extended to include other known and emerging substances of concern.

CHAPTER 2: MANAGING RISKS IN DOMESTIC WATER SUPPLIES

2.1 INTRODUCTION

Water quality guidelines for the domestic environment are also necessary to ensure that water is suitable for both consumptive (e.g. drinking) and non-consumptive uses such as bathing and household use. The ultimate objective of drinking water quality guidelines is the protection of public health. Therefore, every effort needs to be taken to ensure that water intended for human consumption is safe to use. Domestic water quality guidelines relate to the allowable concentrations of physical, chemical or microbiological contaminants in water for the different domestic uses that upon exposure do not pose significant human health effects, or cause significant economic and physical effects within domestic settings. The greatest risks to consumers of drinking water are pathogenic microorganisms. Protection of water sources and treatment are of paramount importance and must never be compromised. Disinfection is the single process that has had the greatest impact on drinking water safety. There is clear evidence that the common adoption of chlorination of drinking water supplies in the 20th century was responsible for a substantial decrease in infectious diseases. In general, the highest priority guidelines are those dealing with microbiological contaminants, such as bacteria, protozoa and viruses. Guidelines for chemical and physical parameters can either be health based (usually listed as a maximum acceptable concentration); based on aesthetic considerations or established based on operational considerations (NHMRC, NRMMC, 2011).

The most effective means of ensuring safe water supply should encompass a risk management approach at all steps in the water supply chain, from catchment to consumer. This requires achieving a steady balance between the extremes of failing to act when action is required and taking action when none is necessary. Lack of action can compromise public health, whereas excessive caution can have significant social and economic consequences. Corrective action should be undertaken in a considered, measured and consultative manner. Risk management is about taking a carefully considered course of action. As the obligation is to ensure safe water and protect public health, the balancing process most often favours a precautionary approach (NHMRC, NRMMC, 2011). Thus, a guideline is a recommended numerical concentration level (e.g. of a contaminant) or a descriptive statement (e.g. visual appearance of a water body) that will support and maintain the designated use of a particular water.

2.2 WATER QUALITY RISK MANAGEMENT

2.2.1 Definition of risk

The definitions of risk vary considerably. Risk is a statistical concept defined as the expected likelihood or probability of undesirable effects resulting from a specified exposure to a known or potential environmental concentration of a material. A material is considered safe if the risks associated with its exposure are judged to be acceptable (EPA Victoria, 2004). A risk is posed when there is a source, a potential exposure pathway and a receptor (receiving environment, for example, ecosystems and/ or humans). It is important to note that risk is not a concentration, dose, other value-based point, or even non-value-based levels. Risk is the probability that a particular adverse effect occurs during a stated period of time (DWAF, 2005). Risk-based can therefore be defined as recognising the risk factors in giving effect to risk objectives. Risk is sometimes

defined in toxicology applications as the expected frequency of the occurrence of an undesirable effect arising from exposure (DWAF, 2008). Description of the risk, therefore requires an assessment that provides answers to the following three questions (Jooste, 2015):

- What can happen (the scenario) (dependent on the way/circumstances the water is used)
- How likely is this to happen (probability); and
- If it does happen, what are the consequences (effects of the hazard, in the case of the water quality guidelines)?

Definition of the risk is therefore a description of water user scenarios, its consequences and the likelihood of each occurring (based on the conditions of exposure), in this case in context of the domestic user.

2.2.2 Risk assessment

Risk assessment plays an inherent part of an overall risk management strategy, because it allows for a structured approach to; identify hazards and risk factors that have the potential to cause harm (hazard identification); analyse and evaluate the risk associated with that hazard (risk analysis, and risk evaluation). Risk assessment is a process by which the extent of exposure is compared against the hazard (intrinsic toxicity) of the contaminant to determine whether it is likely to result in harm to the exposed individual(s). Exposure to a contaminant can be by oral, inhalational or dermal routes (WHO, 2006). In general terms, risk depends on the following 3 factors:

- How much of a contaminant is present in the water?
- How much contact (exposure) a person or other receptor has with the contaminated water, and
- The inherent toxicity of the contaminant.

The elements of the risk assessment process, as it pertains to the development of the health-based water quality targets are discussed in the sub-sections below.

2.2.2.1 Hazard identification

Hazard identification (presence of the hazards), in which a determination is made as to whether a stressor (the water quality constituent) has the potential to cause harm to human health, and/or potential to cause physical damage to property and/or potential to reduce the desirability (aesthetics). It is the process of determining whether exposure to a stressor can cause an increase in the incidence of specific adverse health effects (e.g. cancer, birth defects). It is also whether the adverse health effect is likely to occur in humans. Hazardous agents include microbial, chemical, physical and radiological agents. In the case of chemical stressors, the process examines the available scientific data for a given chemical (or group of chemicals) and develops a weight of evidence to characterize the link between the negative effects and the chemical agent. Exposure to a stressor may generate many different adverse effects in a human: diseases, formation of tumours, reproductive defects, death, or other effects.

2.2.2.2 Hazard characterization

Hazard characterization involves the following;

- Dose-response assessment, in which the numerical relationship between exposure and effects are examined;
- Exposure assessment, in which what is known about the frequency, timings, magnitude, and levels of contact with the stressor is examined.

- Exposure assessment considers both the exposure pathway (the course an agent takes from its source to the person(s) being contacted) as well as the exposure route (means of entry of the agent into the body). The exposure route is generally further described as intake (as eating, drinking, or inhaling) or uptake (absorption through skin or eye) (USEPA). Plausible pathways are assessed and evaluated to determine whether each pathway would be operable for each receptor.
- Exposure assessment is the process of estimating the exposure of a human receptor to a substance under a given exposure scenario. An exposure assessment is conducted for each potential hazard identified. For humans, exposure is determined as a dose and is called the estimated daily intake (EDI). The EDI is typically expressed as milligram (mg) of a chemical per kilogram (kg) of body weight per day, mg/kg-d. The EDI is calculated from site-specific concentrations of substances in each environmental medium, in this case, water, the amount of time a receptor spends in the area and receptor-specific parameters, such as body weight, ingestion rates and dietary preference.
- For human health risk assessment, the most susceptible receptor varies depending on the expected water use. For example, the very young, the elderly and the immunocompromised would be the most susceptible.

2.2.2.3 Risk characterization

Risk characterization, in which the exposure and dose-response assessments are combined to produce a quantitative risk estimate of the hazard/hazardous event. It examines how well the data support conclusions about the nature and extent of the risk from exposure to the hazards.

- In this step information that has been gathered from the exposure and toxicity assessments are combined to determine if a potential risk exists. Risks may be estimated qualitatively based on scientific judgement for a screening level risk assessment, or in a more detailed risk assessment, quantitatively assessed using exposure ratios (ER) for non-carcinogenic constituents or incremental lifetime cancer risk (ILCR) for constituents known or suspected of causing cancer.
- Establishing risk for a hazard will, by its very nature, consider typical mechanisms of exposure of the target organism to the water in question.

The level of risk for each hazard can be estimated by identifying the likelihood of occurrence (e.g. certain, possible, rare) and evaluating the severity of consequences if the hazard were to occur (e.g. insignificant, minor, moderate, significant). The aim is to distinguish between these ranges of risks.

2.2.3 The concept of risk management

The estimation of risk (probability of the risk occurring) constitutes the risk assessment process, which would then have to be taken by the user into the risk management phase to assess if the estimated risk is an acceptable one in the context of the situation. The risk assessment supports the risk management process, but the decision making will further also need to be based on target population, social concerns, public perceptions, economic issues or other related considerations. The differences between the processes of risk assessment and risk management are outlined in Table 2-1: (DWAF, 2005).

Table 2-1: Risk Analysis

	Risk Assessment	Risk Management
Actions	Identify Describe Measure	Evaluate and Judge Decide Implement
Influencing factors	Nature of effects Potency of agent Exposure Pathways Population at risk Average risk Cumulative risk	Social importance of risk De-minimise levels Acceptable risk – Regulatory Criteria/Policy Decision to reduce/not reduce risk Economics Priority of concern
	Sensitive groups Uncertainties of science Uncertainties of analysis	Legislative mandates Legal issues Risk perception

2.2.4 Water safety planning as a risk management strategy

The current edition of the WHO Drinking Water Quality Guidelines (WHO, 2017) emphasizes the need of consistently ensuring the safety and acceptability of domestic water supplies through the development and implementation of water safety plans (WSPs). The WSP approach advocates for proactive risk management from catchment to consumer. The key components of the WSP include a systems assessment, effective operational monitoring and management and communication (WHO, 2017) as part of a holistic risk management approach in all steps in the water supply chain, from catchment to consumer Figure 2-1). The USEPA, New Zealand, Australia and Canada adopt a similar approach, where treatment techniques such as filtration and disinfection are enforced, as a means to eliminate the health risk. In South Africa, the WHO WSP approach was formally adopted in 2008 through the introduction of the Blue Drop Certification programme, an incentive-based regulation programme for ensuring that the water supplied by the designated water service provides (WSPs) meet specified health-based targets, such as those set out in the South African Standards for Drinking Water (SANS 241: 2015).

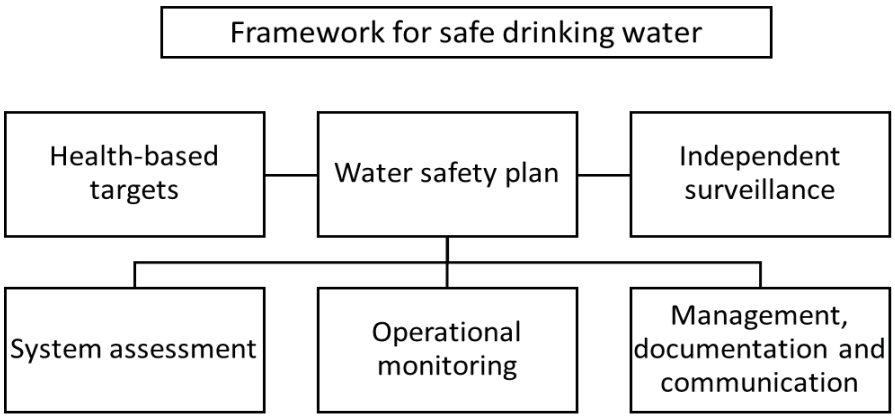


Figure 2-1: A framework that will produce potable and safe drinking water and consists of health-based targets, a water safety plan and independent surveillance (Davidson et al., 2005)

2.3 ASSESSING RISKS IN DOMESTIC WATER SUPPLIES

2.3.1 Overview

Risk assessment attempts to provide scientific estimates of health and environmental risks, and to identify sources of uncertainty inherent in scientific data. The concept of risk assessment is useful for supporting decision making, thereby providing guidance on the quality of domestic water supply as it aims to provide a risk-based decision on the fitness of water for a specific use. The 'bottom line' for a risk assessment often presents the possible adverse effect in the form of a certain (numerical) probability that there will be an increased risk, based on all the assumptions made in the analysis, for each segment of an exposed population and for each type of exposure, summed up as total risk (Leiss and Chiolco, 1994). To put it simply, a risk assessment analyses what can go wrong, how likely it is to happen, what the potential consequences are, and how tolerable the identified risk is. As part of this process, the resulting determination of risk (guidelines) may be expressed in a quantitative or qualitative fashion. Assessment of risks in domestic water largely revolves around protection of human health. The level of protection provided by guidelines depends entirely on the risk assessment criteria used. Thus, water quality guideline values can either generic, i.e. protective of the general population over a lifetime of use or can be set to be protective for specific contexts (DWAF, 2008).

2.3.2 The conventional generic risk assessment approach

Generic water quality risk assessment involves the use of using widely applicable assumptions about the characteristics and behaviour of water contaminant sources, pathways and receptors. The result from generic water quality risk assessment are health-based water quality targets (or guideline values) designed to be cautious and protective of a very wide range of populations, contexts/site conditions and receptor characteristics and behaviour. The 1996 South African Water Quality Guidelines: Domestic Use (Volume 1) were derived using a generic risk assessment approach and are essentially a user needs specification of the quality of water required for different domestic uses. The domestic user guidelines were to a fair extent based on a risk philosophy. Target water quality ranges per constituent were determined by assuming lifelong exposure and incorporate a margin of safety. The target water quality ranges were set as equal to the no-effect range which is defined as the concentration at which the presence of the constituent would have no known or anticipated effect on the fitness of the water for domestic use (DWAF, 2008).

The risk to the domestic water user was accounted for in terms of human health (short and long term), aesthetic (taste, colour, odours, staining, etc.) and economic (damage to appliances) effects. The risk posed to the user in terms of the stated potential impacts also considered the source of the water and the level of treatment. Thus the 1996 guidelines for domestic water use are considered a generic risk-based guideline, that does assess risk posed by different water quality constituents by considering the source (water quality constituent), pathway (exposure/type of contact) and the receptor (potential effect/impact). However, while the guidelines are easy to use, certain shortcomings have been identified. Many of these are fundamental in nature (DWAF, 2008 and S Jooste, 2015, unpublished):

- They are generic and conservative in nature (one size fits all);
- The lack internal consistency (competing user groups);
- Their lack of alignment with important DWS policy, mandate and related initiatives (1998 NWA);
- The ease of misuse (e.g. guidelines verse standards); and
- They are not supported by current risk-based approaches;
- They do naturally facilitate informed use;

- They are limited in terms of local relevance, much of it has been based on international databases;
- They lack transparency in that the original data, algorithms and assumptions are not readily available;
- While simple, being generic they are over-simplified thereby compromising wide functionality;
- No procedures were indicated for incorporating new data; and
- The practical issues regarding the cumbersome nature of the hardcopy volumes in context of the digital age that we live in.

2.3.3 Need for a risk-based and context specific risk assessment approach

The decision support function of required for water resources management in terms of quality guidance has grown and become more complex due to the growing global scarcity of water resources and degradation of their quality and South Africa, is no exception. Increased scientific understanding of the complexity of water ecosystems and adaptive catchment management processes has led to new ways of managing water quality. Traditional scientific and management approaches may not deal well with contemporary water quality issues. In their place, holistic, best-practice approaches need to be taken to ensure that water resources are managed sustainably. Through the Phase 1 investigation undertaken on the review of the SAWQGs series (DWAF, 2008), the outcomes have recommended that the domestic user guidelines maybe improved in terms of the following:

- Confirmation of the definition of domestic use; what does it encompass specifically in light of the revised Water Services Act (Act 107 of 1997) Regulations and SANS 241 (2015);
- Expansion of the water quality range of constituents; to include relevant constituents including persistent organic pollutants and endocrine disrupting chemicals;
- Expansion where necessary the risk-based approach (explicitly risk based to include a tier of guideline criteria related to a risk for a particular water quality constituent);
- Alignment and reference to systems and frameworks within the South African regulatory domain.
- To account for site specificity (tiered system of guidelines);
- To produce a software base decision support tool for use by regulators, water managers, government officials, water resource practitioners and water users.

Addressing the gaps identified and making the improvements recommended will enhance and optimise the use and applicability of the guidelines specifically in terms of consistent guideline application and the decision support required in the current environment governing water resource management. The two fundamental enhancements viz. being explicitly risked based and the site specificity functionality are the components that distinguish the new envisaged guidelines from the 1996 guidelines. The Water Research Commission (WRC) has taken up the need to update and expand the current series of South African Water Quality Guidelines (SAWQGs) (DWAF, 1996) for fresh water by expanding its scope to facilitate improved decision making and applicability. This project was commissioned in 2015, is the third in the process, the focus of this project being the water quality guidelines for Domestic Water Use.

CHAPTER 3: DEVELOPING RISK BASED AND SITE-SPECIFIC WATER QUALITY GUIDELINES FOR DOMESTIC WATER USES

3.1 INTRODUCTION

The term “risk-based water quality guidelines” implies that the regulator and experts have accounted for all the factors that constitute a risk description (Jooste, 2015). It requires the quantification of the risk to yield measurable descriptors of the water quality constituent of concern associated with a fitness for use goal. This would need to done using either generic or context specific criteria that would quantify the acceptable risk associated with the constituent. In using risk-based guidelines expectation can be expressed mathematically on a continuous basis for example through probability or possibility. Risk-based guidelines are already used in many regulatory applications such as when undertaking environmental impact assessments, and with a suitable end-point risk-based guidelines will facilitate comparison. The intention is that the risk-based guidelines provide the risk estimation (risk assessment) to the user who is then able to apply the result to the risk management process (decision making context). It is important also to note that it is seldom possible to make a binary (good/ bad) decision in an environmental assessment. How a constituent presents itself in the uptake process can have a critical impact on what one would expect to happen: presence does not necessarily mean availability. At the same time one constituent may enter the target through various pathways so it is important to recognise the use scenarios.

Note

The water quality guidelines need to be an expression of science supporting a decision

If a water user is given a set of water analyses, what do they mean? What is the fitness for use of that water source and if there is a target fitness for use what are the ranges in values of the different parameters and at the same time what levels should give effect to that target? The outcome of the various discussions has indicated that while a set of water quality guidelines exist in those published in the 1996, and were developed with some degree of risk-assessment, they may be acceptable in certain cases however may be outdated in other cases and not reflect the current state of science. While not explicitly related to the concept of risk, a significant deficiency of the 1996 water quality guidelines is that there are generic and are uniformly applied. Fitness for use water is dependent on its composition in relation to its intended use. This therefore implies that site specificity is necessary so that decision making on water fitness for use can be assessed accurately based on its character and context of the intended use (see Figure 3-1). A further specification of the guidelines is that assessment should ensure that the experience of different users is the same, wherever they may be. The site-specific components of the risk-based guidelines relates primarily to the nature of the water resource (source water) and the nature of the water user. The nature of the water resource will relate to the composition of the water quality to be assessed (constituents and concentrations), while the nature of the water user will need to consider how the water is exposed to the domestic user. This considers the selected the conditions of the exposure (duration, volume, route, frequency) and the characteristics of the receptor (e.g. human – age, body weight).

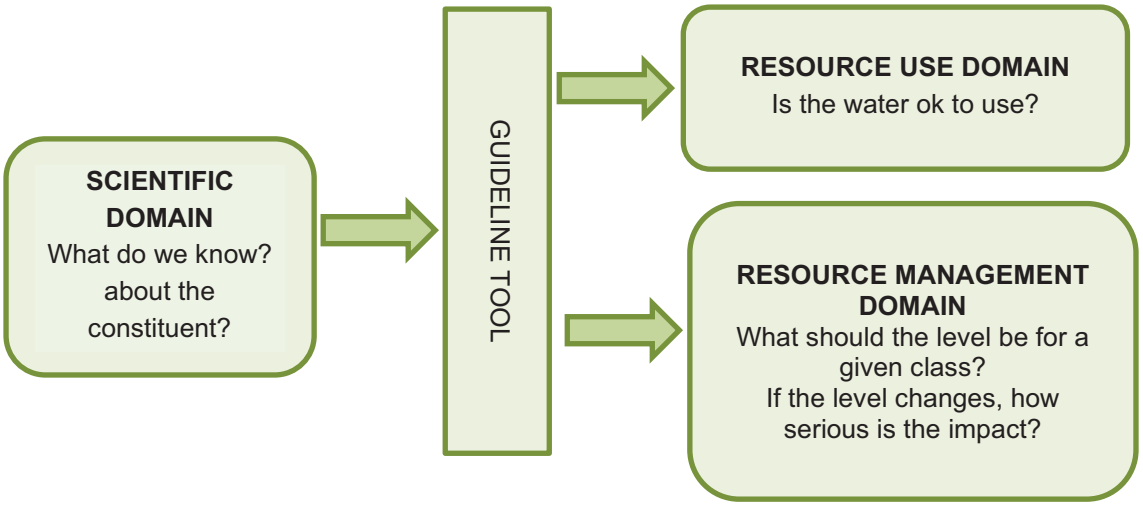


Figure 3-1: Decision context

It is important to note that guidelines reflect the scientific environment whereas standards reflect the regulatory environment. These risk-based water quality guidelines for domestic use reflect an expression of the science that would support a decision on the designated use of a particular water. In South Africa, drinking water quality (potable water) is governed by Section 9 of the Water Services Act (Act No. 108 of 1997), and regulated through the South African National Standards for Drinking Water (SANS 241, 2015, Parts 1 and 2). SANS 241 is a mandatory potable water standard and has the overall objective to protect public health. For bottled water SANS 1675 is applicable. SANS 241 is based on end-point analysis of treated drinking water supplies and is in line with international standards.

Most often standards are static while guidelines can be more flexible. The reason for this is that regardless of whether there are standards in place, a water user may want to know the risk of using a particular water source for a particular use because that may be the only water source available; which is where the guidelines come into play for water users. While there is a space for both standards and guidelines, they must not contradict each other and it must be clear that where a standard is legislated it obviously takes precedence over the guidelines. There are also instances where water supplied via treatment systems may be less than potable standards, and thus a domestic use guideline will help to assess the risks posed to the user, through use of these guidelines. However, it must be emphasised that this does not release the water services institution from their legal obligation to meet the SANS 241 standard. Thus, the need for the domestic use water quality guidelines does not stem from a legal obligation, but rather from the water resource management framework that demands decision support that accounts for all contexts of water use, in this case in the domestic environment.

The intention is that the guidelines will no longer represent a simple pass-fail number, which ignored spatial and temporal variability and with the promulgation of the NWA (Act No. 36 of 1998), lags behind in the evolution and advancement of water resource management in South Africa. The risk science and the approach to be adopted for the domestic user water quality guidelines would consider a combination of qualitative and quantitative risk assessment. At the core of the guidelines is a quantified risk, which is assessed in terms of threshold criteria that relates to categories of fitness for use or water quality requirements.

3.2 CONCEPTUAL FRAMEWORK FOR DEVELOPING THE GUIDELINES

3.2.1 The concept of acceptable risk

In the current context the risk refers to the probability of specific adverse/undesired effects to the domestic user of the water (human health and possibly physical effects associated with using less that target water quality, not necessarily as a frequency) (DWAF, 2008). In the update of the water quality guidelines, the adoption of the risk domain is that it can provide a common philosophical basis for decision-making in different contexts. A risk-based assessment for the purposes of deriving water quality guidelines provides an explicit and transparent process for the acceptance with the need to make management decisions for complex water resource systems and water user needs that may not be always fully understood. The risk science and the approach to be adopted for the update of the domestic user water quality guidelines considers a combination of qualitative and quantitative risk assessment. It is based on international practices, expert scientific knowledge and judgement, scientific data availability, applicability of mathematical models and tools, local conditions and context and practical considerations, which will need to ensure consistency and the explicit recognition of the uncertainties and the assumptions that apply.

Acceptable risk is a concept used in risk management to reflect the highest risk that can be tolerated for the specified adverse effect and target population. It depends on scientific data, social, economic, and political factors, and the perceived benefits arising from exposure to a contaminant (the hazard). Acceptable risk decisions are rarely easy. The subject of what constitutes an acceptable risk is an extremely complex issue and must be handled from a policy perspective. In determining acceptability, it is however largely the perceived risk that determines the basis of what can be tolerated. Acceptable risk is very location-specific, and in some cases culturally specific. For this reason, it plays an important role in adapting guidelines to suit local circumstances, where local stakeholder involvement and available data is vital. The subsection below provides some insights on some of the key international approaches currently applied in determining acceptable risk for the purposes of guideline development.

3.2.1.1 A predefined probability approach

'The lifetime exposure to a substance increases a person's chance of developing cancer by one chance in a million or less' (taken as essentially zero), has been a widely used environmental regulation using a probability approach. First derived in the United States in the 1960s, and later amended, the level of 10^{-6} has been something of a golden standard. A 10^{-5} risk of developing cancer represents 1 chance in 100,000 associated with environmental contaminants and has evolved into a target risk and is in line with WHO guidelines for drinking water quality. It is generally thought that where practical, an excess lifetime cancer risk of 10^{-5} for carcinogenic risks over a lifetime is acceptable (WHO, 2001). Another probability approach used by the USEPA, for microbial risk has been the use of *Giardia* as a reference organism – 'risk is less than 1 infection in 10 000 people per year'. The use of *Giardia* was that it was known to be more resistant to disinfection than other pathogens. Thus, protection to *Giardia* infection should provide protection to other organisms minimising all microbial illnesses. Putting the burden of chemical contamination into context, in South Africa, our current risk of developing cancer is approximately 1 in 4 (or 0.25) with international estimates of background levels of environmental contaminants contributing between 1 in 1 000 and 1 in 100 of this risk. Even with data that are not up-to-date, a perspective of relative risk contributions is provided (WRC, 2018)

3.2.1.2 Currently Tolerated approach

This approach adopts the principle that any risk that is tolerated is considered to be acceptable (i.e. it falls below a level that is already tolerated). It was initially used by the USEPA for recreational waters in setting allowable bacterial indicator densities. Based on studies of health effects of swimming associated gastroenteritis, illness rates (levels) per 1 000 bathers were identified. These levels were considered to be tolerated as people were still swimming and therefore considered to be acceptable. Further work has used epidemiological studies and dose response relationships to determine acceptable risk (WHO, 2001). A tolerated approach based on a combination of exposure distribution, dose-relationship and independent risk factors may be used for health-related acceptable risk. The premise of such an approach is that it should be based on an informed choice.

3.2.1.3 Disease burden approach

This approach considers health risks in terms of total disease burden of a community/population, and defines acceptability in terms of in falling below an arbitrary defined level (WHO, 2001). Descriptions of tolerable burdens of disease relating to water are typically expressed in terms of specific health outcomes such as maximum frequencies of diarrhoeal disease or cancer incidence. However, these descriptions do not consider the severity of the outcomes. The various hazards that may be present in water are associated with very diverse health outcomes with different impacts ranging from mild diarrhoea to potentially severe outcomes such as typhoid, paralysis or cancer. A common “metric” can be used to quantify and compare the burden of disease associated with different water-related hazards, taking into account varying probabilities, severities and duration of effects. The metric used by the WHO to evaluate public health priorities and to assess the disease burden associated with environmental exposures is the disability-adjusted life year, or DALY. The World Health Organization has used DALYs to aggregate different impacts on the quality and quantity of life and to focus on outcomes and not only potential risks. DALYs can be used to define tolerable burden of disease and the related reference level of risk, and therefore support public health priority setting.

3.2.1.4 Other Approaches:

Public acceptance of risk: This approach in determining acceptable risk is based on what is acceptable to the general public, *i.e*. a risk is acceptable if the public find it acceptable. While this is an ideal model in democratic societies this approach is faced with a number of practical and theoretical problems. This may include, varied perceptions of risk by the public, full access to information to the entire public, skills to interpret the information, people’s judgements being subject to bias, underestimation or overestimation of risk by the people, gaining consensus of the general population, media bias and influence by pressure group or activist groups. While public acceptance plays a key role in the decision-making process in determining acceptable risk, it is apparent that it cannot solely be relied upon to determine acceptable risk due to a number of serious difficulties (WHO, 2001).

An economic approach: An approach based on cost-benefit analysis. Acceptable risk is defined when the economic savings arising out of action to reduce a risk outweigh the cost of such action (e.g. new water treatment measures to compared to cost of illness over a lifetime). A simple cost-benefit model however poses many difficulties and it is not a simple exercise to compare financial costs to the exact amount of illness. Some key challenges encountered include difficulties in costing risk related to the element of probability (probability of an illness outbreak and linking this to long term financial cycles); the variance in risk reduction incurred by different groups to those that benefit from it (costs of new works are borne by the shareholders but who benefits would be varied – the swimmers, employers as employees are less likely to

fall ill, less burden on the health care system, etc.), costs not quantified in monetary terms (quality of life, pain, distress) and opportunity costs (best spending of scarce resources specifically in developing nations – water treatment for stricter microbiological standards versus health care systems). The science of economics does not provide society with tools for determining what risks may be acceptable. However, in determining acceptable risk, economic imperatives are critical and need to be considered (WHO, 2001).

3.2.2 The incorporation of site-specific considerations

While, site specificity is also not an explicit requirement in either the NWA or National Water Resource Strategy (2013), it is key component of sustainable water resource management. The sustainability of a water resource is influenced by a large number of factors and considerations, with site specific conditions, influences and characteristics being fundamental to how a resource is managed. Where a water resource is located, the source (surface or ground), its extent of use, its ecological health, sensitivity and importance, its water quality and flow condition, the degree and significance of land-based impacts, socio-economic reliance on it, public perceptions and aesthetic value amongst other factors thus influences not only the water resource but the decision-making processes applicable to the specific context. Fitness for use of a specific water resource will be thus dependent on its catchment characteristics and context. This therefore implies that site specificity is necessary so that decision making on water fitness for use can be assessed accurately. This does not imply that users in one area are treated better or worse that the same user in another geographic area. Two users in different geographical areas will, for example, experience the same water constitution differently depending on the abovementioned factors. The guidelines should ensure that the experience of different users is the same, wherever they may be. Fitness for use is a scientific judgement, involving objective evaluation of available evidence, of how suitable the quality of the water is for its intended use. Water quality can therefore only be expressed in terms of fitness for use.

The envisaged guidelines will address this by allowing the fitness for use assessments and water quality objective setting to be done for more specific scenarios (S Jooste, 2015, unpublished). While location is a major factor, the new guidelines would need to handle different scenarios or contexts at the same site (DWAF, 2008). The site / scenario specificity of the new guidelines will relate primarily to the nature of the water resource and the nature of the water user (DWAF, 2008). The inclusion of this functionality into the decision support system will overcome the shortcoming of the generic nature of the 1996 guidelines, as well as facilitate more informed decision making related to water resource use and management (DWAF, 2008).

3.2.3 Distinguishing features of the three tiers of risk assessment

The intention with the update of the guidelines is that the final product provides a series of tiered assessment levels that supports a greater diversity of guideline use and facilitates the decision making regarding the balance between the protection and use of the water resource (DWAF, 2008). A three-tiered set of guidelines is to be defined. The Tier 1 and Tier 2 facility must be as easy to use as possible. Each tier provides an output that has to comply with the concept of classification or categorization. The difference between the tiers lies primarily in the degree of site-specificity required to produce an output. It should not be equated to or confused with the tiers of risk assessment (S Jooste, 2015, unpublished). All tiers must be categorised in terms of risk objectives (classes). The three tiers proposed is as follows:

- **Tier 1**: Tier 1 requires no site-specific detail and it is intended to reflect the most conservative set of conditions, even if these do not occur together. Minimum user input required and simple output provided; the current guidelines updated as required.

- **Tier 2:** The second tier makes provisions for moderately site-specific risk assessments, requiring some skills, but largely uses pre-defined water use scenarios and limited site characterisation choices with common field observation and or measurement input required from the user for scenarios manipulation. Possibly rule-based output interpretation. This tier is aimed at a guideline user with reasonable insight into the water uses and who is able to select specific options presented in a pre-selected set of scenarios. This tier is seen as the most onerous part of the guidelines preparation work and comprises the biggest percentage of guideline impact.

- **Tier 3**: The third tier allow assessments and objective setting to be carried out in site-specific contexts not covered by tier 2. The third tier of the guidelines is intended for use in highly site-specific contexts. This comprises a description of what is expected of risk assessment or risk-based objective setting. It provides the most site-specific guidance – probably a risk assessment protocol, requiring highly skilled input- and output interpretation.

3.3 RISK ASSESSMENT APPROACH

3.3.1 Overview

The concept of risk used in the guidelines refers to the likelihood of an adverse effect, of an identified hazard causing harm. In this regard one can distinguish between the objective likelihood of effect or intrinsic risk (hazard) and the subjective or extrinsic risk (receptor). The intrinsic risk is determined by the interactions within the system while the extrinsic risk is determined by the subjective acceptability of a given level of intrinsic risk specified by the user. In the course of deriving the guidelines the risk refers to the probability of the adverse/undesired effects to the domestic user of using water containing a potential hazard, including the severity of the consequences. The hazard in this context refers to a range of water quality constituents that may be present in the water that renders it less fit for use, and its consequences based on the how the water is to be used within the domestic environment. Thus, risk is a function of hazard and exposure. Where *hazard* = biological, chemical or radiological agent that has the potential to cause harm, *hazard effect* = adverse impact on human health/appliances/household items that can result from exposure to a substance and *exposure* = contact between a substance and an individual or a population. The threat caused by a hazard depends not only on the severity of its effect but also on whether or not the effect is reversible (Leiss and Chiolco, 1994; NRMMC Australian Drinking WQGs, 2016).

Figure 3-2 presents the conceptual approach to the risk-based guideline development which includes the building blocks and components that comprise the reference and source data elements used to calculate the risk output. Each of the building blocks comprising the risk approach applied in the development of the domestic user water quality guidelines are detailed in the sub-sections below.

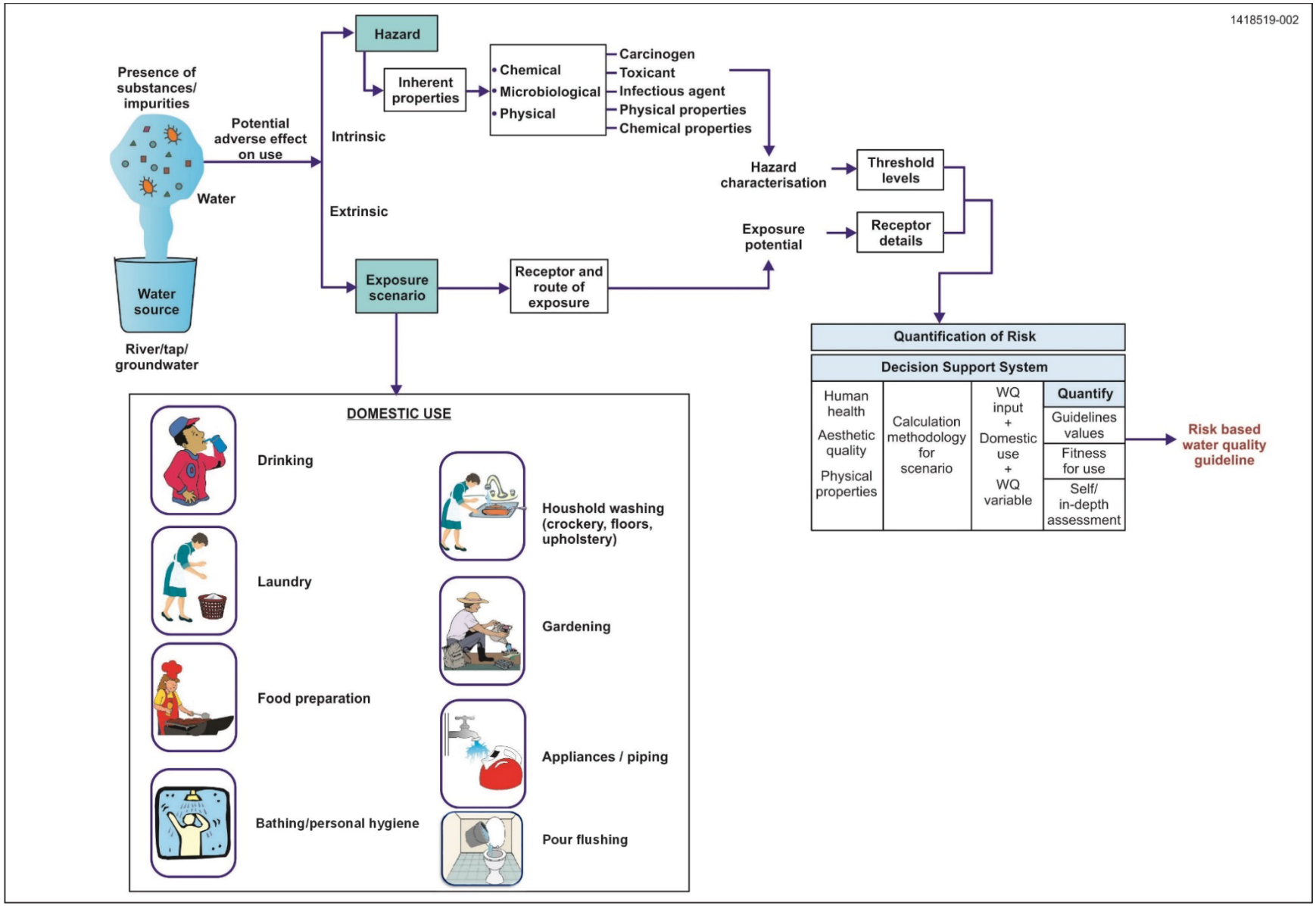


Figure 3-2: Approach to risk-based and site-specific water quality guideline development

3.3.2 Extrinsic risk and exposure route scenarios

For the purposes of this document, domestic water refers to any water that is used for domestic purposes, irrespective of its source and whether or not it has been treated. The domestic use water quality guidelines are to provide a given risk probability for a range of physical, chemical or microbiological contaminants in water, in relation to domestic use categories that define the similar water use characteristics and whose exposure profile is sufficiently similar. The domestic use categories comprise the 'scenario' (what is the water to be used for) that would be assessed to determine the risk based on the consequences identified, and the probability of occurrence. For the domestic user the following categories have been defined in terms of the types of intended uses of water within a household.

- Drinking;
- Food and beverage preparation;
- Bathing and personal hygiene use;
- Laundry;
- Household washing (washing of household items/ property, such as crockery, furniture, floors);
- Appliances and piping systems;
- Pour flushing; and
- Gardening (subsistence).

The domestic use categories dictate the exposure scenario and, on that basis, the route of exposure (how is the water being contacted – e.g. intake, uptake, contact) and the receptor details (the characteristics, the threshold tolerance, dose-response relationships, susceptibility of the contact point to the water quality constituent, *i.e*. the hazard) in which the potential risk presents itself. The potential exposure scenarios based on the various ways in which the domestic user may use water, are defined in Table 3-1. Based on the above, the three primary exposure scenarios that would present itself to the domestic user in the event that water with less than the target water quality is used, are to human health, aesthetic quality and physical effects. These exposure scenarios comprise the first building block to the risk assessment applied in the decision support system; in that it directs the criteria and considerations into the selection of the type of methodologies that should apply to determining the risk.

Table 3-1: Domestic user exposure scenarios per category of domestic use

Domestic Water Use category	Receptor details	Exposure Route	Exposure Scenario (related effect)
Drinking	Human attributes (e.g. age, body weight, sensitivities of population)	Ingestion (accounting for volume intake; frequency)	Human health
	Human palatability – threshold level	Ingestion (acceptability)	Aesthetic
Food and Beverage Preparation	Human attributes (e.g. age, body weight, sensitivities of population)	Minimal Ingestion (accounting for volume intake; frequency)	Human health
		Inhalation (accounting for volume intake; frequency)	Human health
	Human palatability – threshold level	Ingestion (acceptability)	Aesthetic

Domestic Water Use category	Receptor details	Exposure Route	Exposure Scenario (related effect)
Bathing and Personal Hygiene	Human palatability – threshold level	Minimal ingestion	Aesthetic
	Human attributes (e.g. age, body weight, sensitivities of population)	Inhalation (dose, duration)	Human health
	Human attributes (tolerance threshold)	Dermal contact (dose, duration)	
Pour flushing	Human attributes (e.g. age, body weight, sensitivities of population)	Inhalation (dose, duration)	Human health
	Human attributes (tolerance threshold)	Dermal contact (dose, duration)	
Laundry	Human attributes (tolerance threshold)	Dermal contact (dose, duration)	Human health
	Clothing, linen, similar (colour, fabric)	Physical immersion in water (frequency, concentration of hazard)	Aesthetic
			Physical Effect
Household washing	Human attributes (tolerance threshold)	Dermal contact (dose, duration)	Human health
	Floors, carpets, upholstery, crockery	Physical contact with water (frequency, concentration of hazard)	Aesthetic
			Physical Effect
Appliances, Piping	Household appliances, pipes	Continuous/regular, containment of water (concentration of hazard)	Aesthetic
			Physical Effect
Gardening	Vegetable/Fruit Crop roots and leaves (Type)	Root zone uptake; Leaf wetting *due to irrigating* (frequency, duration, sensitivity)	Physical damage to crop
	Human attributes (e.g. age, body weight, sensitivities of population)	Ingestion (raw vegetables; dose, frequency)	Human health

3.3.3 Intrinsic risk and hazard characterisation

The intrinsic risk in terms of the risk-based water quality guidelines represents the characteristics of the water quality constituent (the hazard) in question. This component of the development process has focused on undertaking research and a literature survey of international and local data sources and databases to draw primarily on empirical data generated from laboratory tests, statistical models or other functions and from qualitative expert judgements. The data derived from the survey and review of these risk assessments of the selected water quality constituents to be included in the technology demonstrator, is to comprise the input data components (reference data) into the calculation methodology.

3.3.3.1 Hazard identification

The physical characteristics/aesthetics of and chemical and microbiological contaminants in water determine its properties and thus its potential to cause an undesirable effect on the domestic use categories, *i.e.* the concentrations/presence of the contaminant or physical property of the water defines the hazard. As this

project is required to deliver on a technology demonstrator of the DSS, the focus was to identify and include a suite of representative water quality constituents that addresses the different types of hazards related to domestic use. The primary purpose of this endeavour is to develop the risk-based methodology for the different hazard groupings through the technology demonstrator that can then be expanded on through the next phase of the project, by the addition of further constituents.

As a departure point and based on the outcome of the workshop of the working committee held in May 2016, the identified water quality constituents to be included in the technology demonstrator include the suite of constituents comprising the 1996 Domestic Use Water Quality Guidelines (Volume 1), the incorporation of the relevant constituents included in SANS 241 (that are currently not included in the 1996, Volume 1 suite), and selected constituents of WHO Drinking Water quality guidelines. The final list of constituents forming part of the technology demonstrator are listed in Appendix A. The inclusion of the range of constituents in the DSS was dependant on the availability of toxicological assessment and empirical data, which was required to support the risk assessment. However, constituent lists in the DSS may be easily expanded in future and extended to include all recommended constituents. The functionality exists to include the new constituents to the database.

3.3.3.2 Hazard characterisation

The hazard characterisation outlined through this section is taken through to the decision support system as the input reference data, which is then run through a calculation methodology based on the pre-defined domestic user exposure scenarios that apply.

3.3.3.3 Categories of hazards

Based on the hazard characterisation and the user exposure scenarios described in the sections above five categories of hazards can be defined in terms of the type of resultant effect. These hazard categories dictate the calculation methodology to be applied in the DSS for the risk quantification of fitness for use per effect 'type'. The categories of hazards are indicated in Table 3-2.

Table 3-2: Categories of hazards as related to potential effect

Exposure route	Potential Effect	Hazard Category
Ingestion/partial ingestion/inhalation/dermal	Human health	• Toxicant • Carcinogen • Infectious agent
Ingestion/ partial ingestion	Aesthetic quality	• Physical properties of the water
Physical contact/immersion/containment in/of water	Physical Damage	
Root zone uptake; Leaf wetting	Physical Damage to the crop	• Chemical properties – related to gardening

3.3.4 Risk estimation and the concept of acceptable risk

Risk is generally taken to be the probability of injury, disease, or death under specific circumstances (WHO, 2001). Acceptable risk is used in risk management to reflect the highest risk that can be tolerated for the specified end-point and target population. It depends on scientific data, social, economic, and political factors,

and the perceived benefits arising from exposure to an agent. Acceptable risk decisions are rarely easy. The subject of what constitutes an acceptable risk is an extremely complex issue and must be handled from a policy perspective. In determining acceptability, it is however largely the perceived risk that determines the basis of what can be tolerated. A number of positions are used as a basis for determining when a risk is acceptable (WHO, 2001). These may amongst others include:

- Falling below a defined probability
- Falling below an arbitrary fraction of total disease burden;
- Falling below some level that is tolerated,
- Public health professional says it is acceptable;
- Costs of reducing the risk would exceed the costs saved, and
- The general public say it acceptable (or what is not).

For purposes of the risk-based guidelines acceptable risk applied includes internationally applied risk levels derived from the probability approach, the tolerated approach and disease burden approach.

3.4 DEVELOPMENT OF A DECISION SUPPORT SYSTEM

3.4.1 Risk assessment in a decision support system

The updated risk-based water quality guidelines for domestic use is to be presented as a software decision support system (DSS) allowing assessments and objective setting to be performed in generic and site-specific contexts. A DSS would offer the advantage of improve the way in which the guidelines are used because the focus will be directly on supporting decisions in specific contexts; by retaining the scientific rigour rather than producing simple numeric guidance. The scientific and technical context will be presented more accessible to the decision maker. The DSS provides a structured approach necessary for addressing the main decision contexts of the use of the guidelines. A software DSS provides many options on view selection, version control and access control that are useful in a dynamic product, without losing the risk assessment, mathematical calculations and risk-based guidance (S Jooste, 2015, unpublished). The term risk-based guidelines imply that all the factors that constitute a risk description have been considered, and involves a quantitative or semi-quantitative protocol for determining the water quality requirements and the degree of fitness for use of water for a specific domestic water use. Thus, the DSS has to incorporate the two primary functions (S. Jooste *et al.,* 2015, unpublished), i.e.:

- Deriving an expression of fitness for use expressed as a fitness for use category of water characterised by a given set of analyses or observations; and
- Setting water quality objectives corresponding to a required given level of fitness for use.

The potential risk is presented as the ‘guideline’ defining the risk that potentially exists for a domestic user by the use of water of a certain quality (fitness for use). This risk derived would be a function of specific domestic user scenarios and consequences expressed as effects. The core of the quantitative assessment is knowing something about the hazard, which is used to define a range of states of the water, characterised in terms of risk that would support decision making.

3.4.2 Water quality requirement assessment in the DSS

Water quality requirements of water users and the sustainability of our water resources are necessary considerations in the decision-making relating to fitness for use, thus a quantitative risk assessment approach would support risk-informed decision making for both regulators and water users. This risk approach generalises the basis for decision-making by incorporating as much of the relevant evidence as possible. In terms of the domestic user and for the purposes of the risk-based guidelines these categories are defined as follows:

- **Water quality required for drinking** is defined by the effect water quality constituents have in the event of ingestion of water or inhalation of volatiles released from the water during ingestion; and on the aesthetic quality of the water as it relates to taste, odour and colour.
- **Water quality required for food and beverage preparation** is defined by the effect water quality constituents have upon ingestion and on aesthetics, after food has undergone preparation using the water (cooking and boiling in water, washing of food and use in constitution of beverages).
- **Water quality required for bathing and personal hygiene** is defined by the effect water quality constituents have in the event of skin contact of water due to bathing and other personal hygiene applications. Small volumes ingestion of water or inhalation of volatiles released from the water and the effect of the aesthetic quality is also considered.
- **Water quality required for household washing** is defined based on the effects the water quality constituents will have on the washing application (dishes, floors). The consideration of water contact on skin is covered the human health aspect as related to the water use categories of drinking and bathing;
- **Water quality required for laundry** is defined by the effect water quality constituents have on clothing. The consideration of water contact on skin is covered by the human health aspect as related to the water use categories of drinking and bathing;
- **Water quality required for appliances and distribution systems** is defined by the effect water quality constituents will have on appliances and on general plumbing equipment.
- **Water quality required for gardening** is defined by the effect water quality constituents have as it relates to domestic gardening. This definition relates specifically to plants grown for subsistence purposes. The effects as related to crop reduction and microbial contamination are considered as part of domestic use. The water quality requirements (generic application) from the irrigation user water quality guidelines are relied upon and are adopted for domestic use. For more advanced assessments the user is directed to the risk-based irrigation water quality guidelines.
- **Pour flushing:** is defined by the use of greywater[1] for pour flushing (manual flushing) of toilets. The effect water quality constituents have in the event of skin contact of aerosols that arise from the water and small volumes of inhalation of volatiles released from the water during the pour flushing process is considered.

3.4.3 Fitness for use and site-specific considerations

The fitness for use assessment forms the core technical requirement of the guidelines. The focus of this endeavour is therefore to formulate a mathematical approach to the fitness for use assessment as the basis for derivation of the risk-based guidelines. The ability of the user to provide some input to the risk assessment process and contextualising the scenario, supports the proposal of presenting the guidelines as a software product rather than a static document. Based on this two-fold user application functionality the proposed decision support tool would have to include interfaces that cater for the fitness for use and for the objective setting. Fitness for use of a specific water resource will be thus dependent on its catchment characteristics and context. This therefore implies that site specificity is necessary so that decision making on water fitness for use can be assessed accurately. This does not imply that users in one area are treated better or worse that the same user in another geographic area. Two users in different geographical areas will, for example, experience the same water constitution differently depending on the abovementioned factors. The guidelines should ensure that the experience of different users is the same, wherever they may be. The site-specific components of the risk-based guidelines relates primarily to the nature of the water resource (source water) and the nature of the water user. The nature of the water resource will relate to the composition of the water quality to be assessed (constituents and concentrations), while the nature of the water user will need to consider how the water is exposed to the domestic user. This considers the selected the conditions of the

[1] Greywater: wastewater resulting from the use of water for domestic purposes but does not include human excreta. National Sanitation Policy, Department of Water and Sanitation, 2016.

exposure (duration, volume, route, frequency) and the characteristics of the receptor (e.g. human – age, body weight).

3.4.4 Functionality of the decision support system

The risk-based water quality guidelines for domestic use is presented as a software decision support tool and includes a tiered system of assessment which operates at two levels of functionality. The difference between the levels lies primarily in the degree of site-specificity required to produce an output. The DSS is done through a software demonstrator/ prototype system for the purposes of this project using MS Excel as the user platform. The assessment system accommodates for the needs of the novice, intermediate and expert user respectively includes three tiers. The following definition of the tiered assessment levels informs the basis of design for the DSS informatics. The DSS has been designed in terms of the project terms of reference and as aligned to the other user group water quality guidelines.

Table 3-3: Functionality of the DSS

Fitness for Use		**Water Quality requirement**
Site specific		**Generic**
Tier 3	**Tier 2**	**Tier 1**
The most site-specific guidance. A risk assessment protocol, requiring highly skilled input and output interpretation. Allows for the adjustment of the algorithm and reference data. Default site specific component options that can be changed to suit site specific circumstances (more specific models and parameters). Functionality/permissions to adjust the calculation methodologies, reference databases and algorithms to provide the detailed site-specific risk quantification for the scenario.	Moderately site-specific, requiring some skills, but largely uses pre-defined water use scenarios and limited site characterisation choices with common field observation and or measurement input required from the user for scenarios manipulation. Rule-based output interpretation. Calculations are specific to the domestic water use categories and are based on the detail of the site-specific information entered	Most generic (and by implication the most conservative) approach to risk guidance. Minimum user input required and simple output provided. Simplified generic conservative assumptions used and totally reliant on the default datasets (worst case exposure). Does not involve rigorous calculation methodology.

The DSS produces risk-based water quality guidance at two levels, either:

- as a water quality requirement, *i.e*. generic conservative threshold limits per constituent for a selected domestic use category, or
- as a quantified risk estimate of fitness for use expressed for a selected domestic use category based on an input and selected exposure conditions.

A simplified schematic representation of the DSS structure is shown in Figure 3-3.

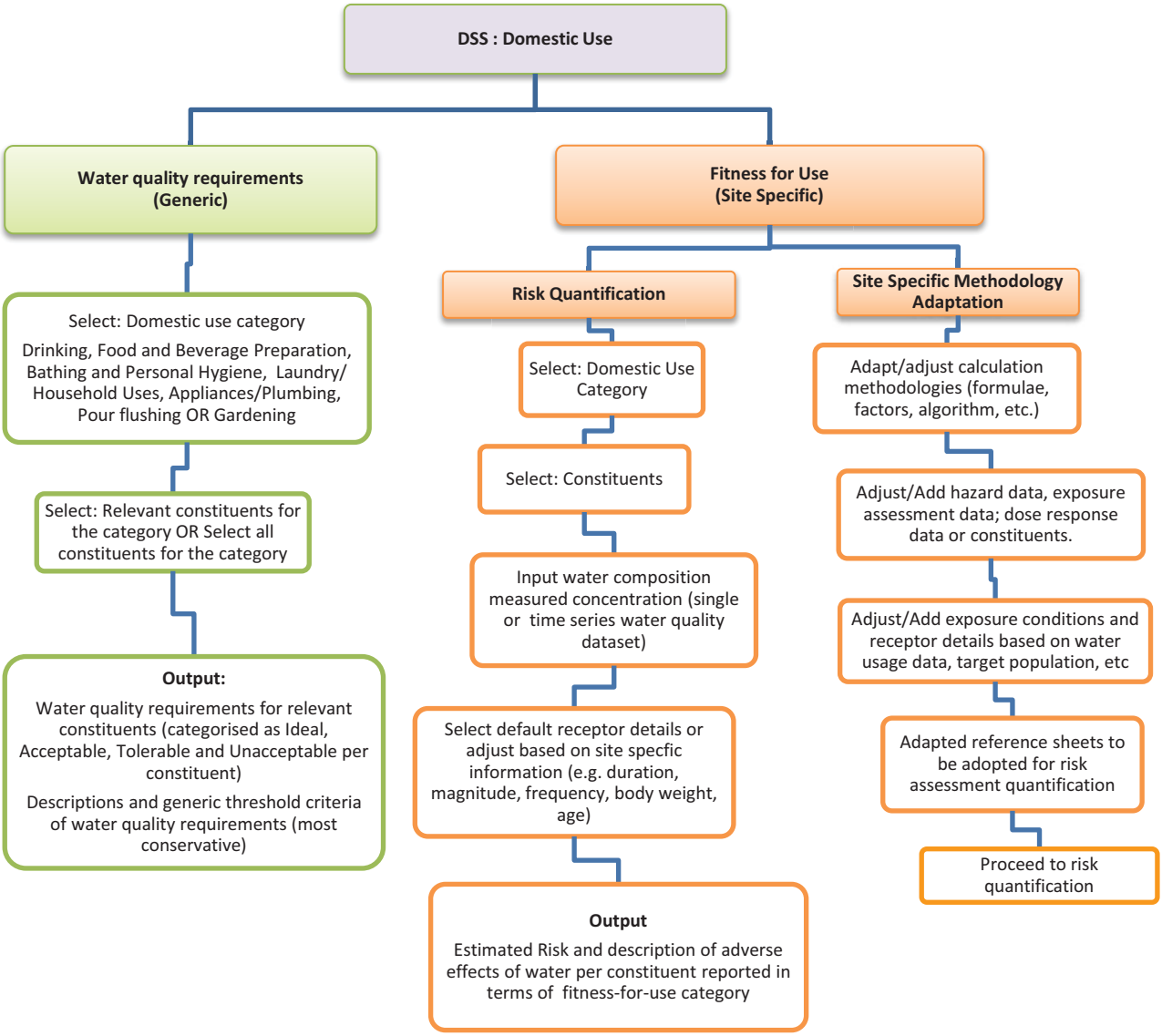


Figure 3-3: Simplified schematic representation of the DSS structure

3.4.5 Outputs of the decision support system

A 'risk-based guideline' (the probability of adverse effect occurring) is generated based on the computation of the following hazard and exposure input parameters through the algorithm for the assessment levels run in DSS:

For Generic (Water Quality Requirement):

- selected domestic use category;
- selected of water quality constituent(s) of interest;

For Site Specific (Fitness for Use):

- selected domestic use category;
- selected of water quality constituent(s) of interest;
- entry of water quality input (water composition either as a single entry or a time series);
- Selection of exposure scenarios (receptor, route, duration, magnitude, frequency).

Further for expert site specific

- functionality to revise the algorithms, methodology and reference data (and rerun as above).

The DSS includes the following as outputs:

- ***Water Quality Requirements (Tier 1)*****:** Use simplified conservative assumptions requiring no input for the assessment. *Output*: The water quality requirements per constituent are categorised as ideal, acceptable, tolerable or unacceptable based on the risk level and the associated adverse effect is reported for the domestic use type and routes of exposure (most conservative and generic). This information is reported for each constituent as selected by the user.

    - *Example:* a user wants to know what the water quality requirements are for domestic use for drinking purposes. The user selects 'Water Quality Requirements' tab on the homepage, the 'Drinking' use category; then water quality constituent of interest or 'All' constituents applicable to drinking. The risk-based threshold limit criteria for the water quality constituents relevant to drinking use are reported at the ideal, acceptable, tolerable or unacceptable levels as the DSS output, with a description of the adverse endpoint effects.

- ***Fitness for Use (Tier 2):*** Specific to the selected domestic water use categories and are based on the detail of the site-specific information entered or selected. It provides options and allows the user to define point concentrations and exposure details. The assessment can be utilised to obtain a conservative fitness for use output based on a specific domestic water composition (water quality) entered by the user. *Output:* A simplified risk estimate of the water quality specified by the user as compared to threshold risk criteria. The calculation of risk is based on a predefined (default) set of conditions with receptor and hazard characterisation remaining constant, allowing a single exposure input or input of a range of exposure concentrations (a record of historical water quality data); or the option to adjust receptor details to account for variabilities in the target population (magnitude, duration, frequency) as the components of site specificity.
    - *Example:* a domestic user who has a borehole and would like to know whether it is safe to drink takes a sample to a laboratory and gets a laboratory certificate of analysis. The

user selects the 'Fitness for Use' tab on the homepage. The 'Drinking' use category is then selected. The user selects water quality constituents of interest and inputs the values (single or time series) per water quality constituent into the DSS for the drinking use category. The user may change the details of who is primarily drinking this water, *i.e.* whether it is an adult, child or infant. The DSS provides a colour coded risk percentage output for each constituent for drinking that is linked to a probability of the adverse effects (endpoints) occurring that may be associated with that risk quantified.

- ***Fitness for Use – Site Specific Methodology Adaptation (Tier 3):*** User is able to make changes and tailor the fitness for use assessment in the DSS to suit more detailed site-specific scenarios/conditions. This functionality is targeted at the expert/experienced user. The user may change the risk calculation methodology and/or adjust the exposure assessment parameters (site specific exposure conditions), hazard reference data (dose-response assessments) or acceptable risk targets as required, based on new empirical scientific data or advancements, or more up to date literature, or based on site specific circumstances. New constituents of interest or of local relevance may also be included. The proposal is that accessibility to this functionality in the DSS is controlled and only password permitted.

    - *Example:* The user uses new toxicological study data to adjust the uncertainty factors and reference doses of the hazard (water quality constituent) in the reference data sheets or adjust the body weights of the receptors in the reference data sheets based on local knowledge and site-specific circumstances of target population. The user then accesses the 'Fitness for Use' functionality as described above to run the risk assessment on the selected scenario.

A separate user interface for water quality requirements and fitness for use guidance has been developed to support the level of functionality.

3.5 RISK REPORTING

"Risk based" guidelines simply allow the suitability of the water to be interpreted in terms of risk of specific adverse effects. The DSS reports on the risk of the likelihood of adverse effects that may be experienced when using the water for a domestic use in a given context. Water quality is therefore expressed in terms of the likelihood of the potential risk. A colour coded generic fitness for use categorization system is reported in the DSS as the potential risk. The DSS uses a two-type reporting system, either a four category or two category system which is dependent on the selected water quality constituent(s). The two-category system is applied to carcinogens and microbial infectious agents and the four-category system to toxicants and physical and aesthetic constituents. The two-category system reports a fitness for use either as (1) above or (2) below an acceptable risk target, and the four category systems reports the fitness for use as (1) ideal, (2) acceptable, (3) tolerable or (4) unacceptable.

This is aligned with the standard practice within DWS (and in line with the irrigation and recreational risk-based water quality guidelines recently developed). The four-category system is in harmony with a risk-based assessment of water quality in that the 'Ideal' category represents a no risk scenario (safe level), while the 'Unacceptable' category represents a high-risk scenario (likely presence of the adverse effects). The two-category system is aligned to the WHO (2017a) health-based target guidelines that is based on the health outcome type. The first outcome considers the burden of disease associated with different water-related hazards, taking into account varying probabilities, severities and duration of effects, and uses the disability-adjusted life years (DALY) tolerable burden of disease target as the

metric. The second outcome considers the incidence of cancer and includes an acceptable risk target level (no adverse effect or negligible risk). This fitness for use categorisation represented by the colour scheme is shown in Table 3-4 and 3-5. The same colour scheme is also used throughout the DSS to depict the fitness for use based on risk.

Table 3-4: A generic description of the fitness for use categories used for risk reporting

Reported Category	Description
Ideal	A water quality fit for a lifetime of use.
Acceptable	A water quality that would exhibit minimal impairment to the fitness of the water for its intended use. No observed adverse effects.
Tolerable	A water quality that would exhibit some impairment to the fitness of the water for its intended use. Minor risk of adverse effects presenting themselves.
Unacceptable	A water quality that would exhibit unacceptable impairment to the fitness of the water for its intended use. Significant risk of adverse effects, presenting themselves.

Table 3-5: A generic description of the of the fitness for use categories used for tolerable burden of disease or cancer risk reporting

Reported Category	Description
Below acceptable risk target	< the upper limit target DALY tolerable burden of disease < the acceptable risk for cancer
Above acceptable risk target	> the upper limit target DALY tolerable burden of disease > acceptable risk for cancer

The categorisation is based on threshold risk criteria as obtained from scientific literature and risk databases that include exposure assessment data for each constituent or on acceptable risk levels. The risk-based water quality guidelines is reported at two levels based on whether the user selects generic or site specific (input based) guidance, as follows:

- ***For Water Quality Requirements (generic)*:** The DSS report screen reports *all* risk threshold criteria and associated fitness for use levels (*i.e*. ideal, acceptable, tolerable or unacceptable) for the specific constituent(s) selected.

- ***For Fitness for Use (site specific)***: The DSS report screen reports only the fitness for use category within which the quantified risk estimates falls (ideal OR acceptable OR tolerable OR unacceptable OR >DALY OR <DALY) together with the risk estimate value, the exposure concentration of the specific constituent and the description of the associated adverse effects.

The threshold limit criteria that apply to the reported fitness for use categorisation differs for each domestic user scenario.

The DSS output reporting sheets of the risk-based threshold limit criteria of applicable water quality constituents per domestic water use category are included in Appendix C.

CHAPTER 4: HUMAN HEALTH EFFECTS

4.1 EXPOSURE TO CHEMICAL CONTAMINANTS

4.1.1 Overview

There are two primary sources of data that have been relied upon to assess health effects from exposure to chemical contaminants, *viz*. human population data and animal toxicological study data. However, the studies are frequently not available since there are significant ethical concerns associated with human testing of hazards. Epidemiological studies involve a statistical evaluation of human populations to examine whether there is an association between exposure to a stressor (the hazard) and a human effect. When data from humans are unavailable, data from animal studies (rats, mice, rabbits, dogs, etc.) are relied upon to draw inferences about the potential hazard to humans. Animal studies can be designed, controlled, and conducted to address specific gaps in knowledge, but there are uncertainties associated with extrapolating results from animal subjects to humans.

4.1.2 Hazard characterisation

A key component of hazard characterization involves evaluating the weight of evidence regarding a chemical's potential to cause adverse human health effects. The weight of evidence narrative may include some standard 'descriptors' that signify certain qualitative threshold levels of evidence or confidence have been met, such as 'Carcinogenic to humans' or 'Suggestive evidence of carcinogenic potential'. Characterization of the hazard as either a threshold (toxicant) or non-threshold (carcinogen) chemical is important as different approaches are used for the quantification of the risk estimate for threshold and non-threshold chemicals. Based on the hazards identified within each domestic use category, the hazard function related to each was characterised. In terms of the human health assessment, statistically controlled clinical studies on humans linking a stressor to a resulting effect provides the best evidence.

4.1.3 Determining the intrinsic toxicity of chemical hazards

The hazard formulation encapsulates what is known about the water quality constituent (hazard) and its interaction within the domestic use category (drinking, bathing, etc., i.e. based on the exposure route) at the level at which an adverse effect is described. It expresses what we know about the point at which we expect no adverse effect, and the point at which we fully expect an adverse effect. This assessment defines the dose response curves that will define the relationship of exposure and effect. Figures 4-1 to 4-3 depicts representative examples of dose response curves.

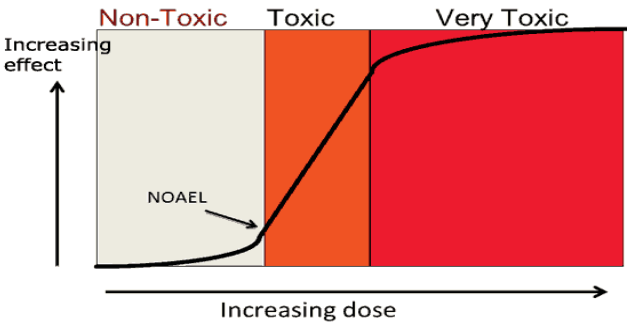


Figure 4-1: Illustrative representation of the relationship between exposure and effect. (http://www.stewardshipcommunity.com/stewardship-in-practice/human-health/hazard-risk-human-health-and-pesticides/hazard-profile-and-risk-assessment.html)

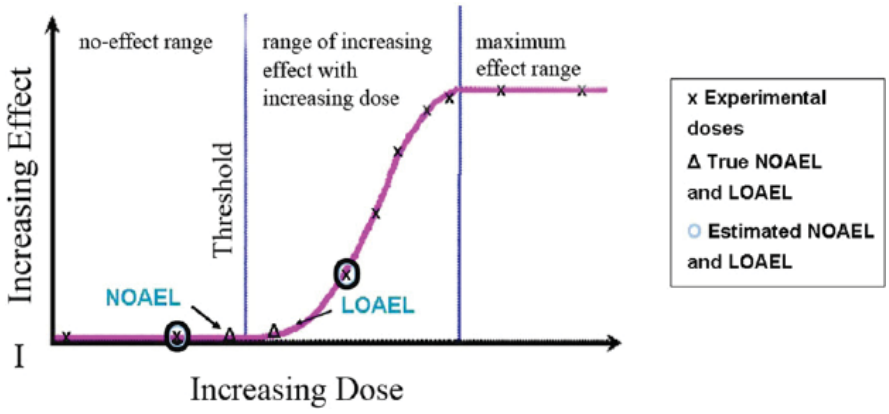


Figure 4-2: Illustrative representation of the lowest and no observable adverse effect curve to determine the relationship of exposure and effect. LOAEL = Lowest observable adverse effect level and NOAEL = No observable adverse effect level. (http://www.stewardshipcommunity.com/stewardship-in-practice/human-health/hazard-risk-human-health-and-pesticides/hazard-profile-and-risk-assessment.html)

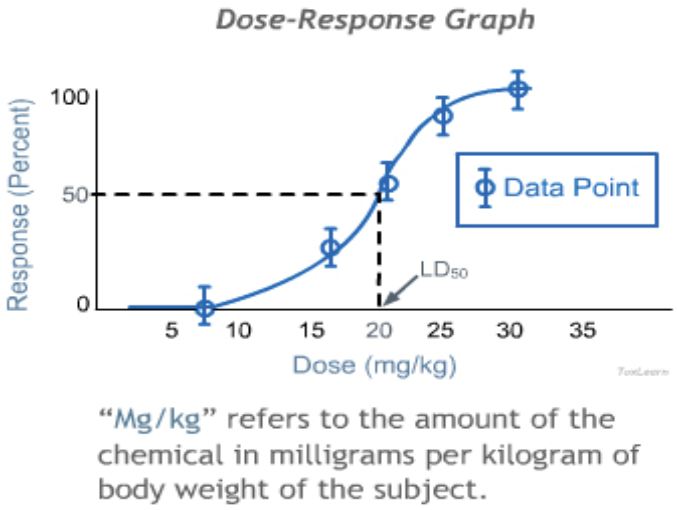


Figure 4-3: Illustrative representation of a dose response assessment curves to determine the relationship of exposure and effect. LD_{50}: Amount of substance required to kill 50% of the population (http://www.stewardshipcommunity.com/stewardship-in-practice/human-health/hazard-risk-human-health-and-pesticides/hazard-profile-and-risk-assessment.html)

4.1.3.1 Dose-response curves

A dose response relationship describes how the likelihood and severity of adverse health effects (the responses) are related to the amount and condition of exposure to an agent (dose provided). The same principles apply for studies where the dose is the exposure to a concentration of an agent (e.g. airborne concentrations applied to inhalation studies), referred to as a concentration-response relationship (https://www.epa.gov/risk/guidelines-developmental-toxicity-risk-assessment). The shape of the dose-response relationship depends on the agent, the kind of response (tumour, incidence of disease, death, etc.), and the experimental subject (human, animal) in question. For example, there may be one relationship for a response such as 'weight loss' and a different relationship for another response such as 'death'. Since it is impractical to study all possible relationships for all possible responses, toxicity research typically focuses on testing for a limited number of adverse effects. However, dose-response relationships observed from animal studies are often at much higher doses that would be anticipated for humans, so must be extrapolated to lower doses, and animal studies must also be extrapolated from that animal species to humans in order to predict the relationship for humans. These extrapolations, among others, introduce uncertainty into the dose-response analysis.

4.1.3.2 Tolerable daily intake

Non-linear dose response assessments relate to where the effects are observed only above a certain threshold dose, with no effects observed at doses below this threshold even with lifetime exposure. These threshold chemicals are classified as toxicants, but also include non-carcinogens. The threshold dose is derived/calculated based on tolerable daily intake (TDI) from which a guideline value is derived. The TDI is an estimate amount of substance in drinking water expressed on a body weight mass basis (milligram or microgram of body weight) that can be ingested over a lifetime without appreciable health risk, and with a margin of safety. The TDI signifies “permissibility, as the substance (hazard) has no intended purpose in the drinking water. (WHO, 2017a). Short term exposure levels exceeding the TDI is not a cause for concern, provided the individual’s intake averaged over the long term does not appreciably exceed the level set. Large uncertainty factors are generally involved in setting TDIs provide adequate level of protection against potential adverse effects of exceedances (WHO, 2017a).

4.1.3.3 No-observed-adverse-effect level

To determine the hazard or intrinsic toxicity of a chemical/contaminant, a comprehensive array of toxicity tests is performed, from which the critical effect and a “no-observed-adverse-effect level” (NOAEL) are derived. An uncertainty factor (sometimes called a safety factor), which is chosen in recognition of intra- and interspecies variability (maximum 10-fold for each) and the adequacy of the toxicological database, is applied to the NOAEL, to give a guidance value. Alternatively, a margin of safety of exposure can be calculated for a specific scenario by comparing the NOAEL with the actual exposure conditions (WHO, 2006). A NOAEL is the highest exposure level of a chemical in a study, found by experiment or observation, where statistically or biologically no significant increases are seen in the frequency or severity of the adverse effect between the exposed population and its appropriate control population. Wherever possible, the NOAEL, is based on long term studies, preferably of ingestion of drinking water. However, NOAELs obtained from short terms studies using other sources of exposure e.g. (food, air) may also be used. If a NOAEL is not available a Lowest-Observed-Adverse-Effect (LOAEL) may be used. The LOAEL refers to the lowest dose or concentration of a substance (hazard) tested at which a detectable adverse effect is noted. Should the LOAEL be used, an additional uncertainty factor is usually applied. (https://www.epa.gov/risk/guidelines-developmental-toxicity-risk-assessment; WHO, 2017). When the non-linear approach is applied, the LOAEL and NOAEL are used as a point of departure to lower doses.

4.1.3.4 Toxicity reference value

The objective of toxicity assessment is to identify potentially toxic effects of the hazard and determination of the amount of constituent that a receptor can be exposed to without experiencing unacceptable effects. This value is called the toxicity reference value (TRV) or toxicity benchmark. For humans the TRV is expressed as mg of a chemical per kg of body weight per day (mg/kg-d) for non-carcinogens, and as a slope factor $(mg/kg\text{-}d)^{-1}$ for carcinogenic chemicals (for human health only). The toxicity assessment provides the basis for evaluating what is an acceptable exposure and what level of exposure may adversely affect human health. The toxicity assessment is based on chronic exposure and not acute exposure.

4.1.3.5 Reference dose (RfD) and concentration (RfC)

The reference dose (RfD) is an oral or dermal dose derived from the LOAEL or NOAEL by application of order of magnitude uncertainty factors. The RfD is defined as an estimate of a daily oral exposure to the human population (including sensitive populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Uncertainty factors are applied to the NOAEL, LOAEL, Benchmark dose (BMD) or RfD in the derivation of guideline values, for the response considered to be the most biologically significant. These uncertainty factors take into account the variability and uncertainty that are reflected in the possible differences between test animals and humans (generally two 10-fold or 100x) and variability within the human population (another 10x) (https://www.epa.gov/risk/guidelines-developmental-toxicity-risk-assessment). Factors lower than 10 may be used for interspecies variation within humans known to be less sensitive than experimental animal species studied. Uncertainty factors greater than 1000, emphasize the higher level of uncertainty (WHO, 2017a). If a LOAEL is used, another uncertainty factor, generally 10x, is also used. In the absence of key toxicity data (duration or key effects), an extra uncertainty factor(s) may also be employed. Sometimes a partial uncertainty factor is applied instead of the default value of 10x, and this value can be less than or greater than the default. Thus, the RfD is determined by use of the following equation:

$$RfD = NOAEL \text{ (or LOAEL or BMD Level)} / UFs.$$

The RfD is generally expressed in units of milligrams per kilogram of bodyweight per day: mg/kg/day. A similar term, known as reference concentration (RfC) is used to assess inhalation risks, where concentration refers to levels in the air (generally expressed in the units of milligrams agent per cubic meter of air: mg/m^3). Risk assessment is intrinsically an uncertain process with uncertainty arising not only from hazard characterization but also from exposure factors and toxicity assessment. In addition to uncertainty arising from lack of (precise) knowledge, variability inherent to the environmental systems and from person to person (inter-individual variability) also contribute to uncertainties in the risk estimates. Inferences and assumptions are thus in most instances, conservative, and strive to overestimate risk.

4.1.4 Carcinogens

For hazards that do not appear to have a threshold, linear dose response assessment applies. These chemicals are capable of producing an adverse effect at any level of exposure, and include carcinogens. The extrapolation phase of this type of assessment does not use uncertainty factors; rather, a straight line is drawn from the point of departure for the observed data (typically the BMDL) to the origin (where there is zero dose and zero response). The slope of this straight line, called the slope factor or cancer slope factor, is use to estimate risk at exposure levels that fall along the line. When linear dose-response is used to assess cancer risk, an excess lifetime cancer risk is calculated (*i.e*. probability that an individual will

contract cancer over a lifetime) resulting from exposure to a contaminant by considering the degree to which individuals were exposed, as compared to the slope factor. Thus, the cancer risk is determined by use of the following equation:

$$\text{Cancer Risk} = \text{Exposure} \times \text{Slope Factor}$$

Carcinogens, in theory, do not exhibit threshold response behaviour. Rather, even at low doses, there is some risk of genetic damage. It is assumed that the carcinogenic effect may be induced at any level of exposure so there is no level below which is considered safe. The evaluation of the potential carcinogenicity of chemical substances is usually based on long term laboratory animal studies. Sometimes data are available from on carcinogenicity in humans, but mostly from occupational exposure. Health effects for known or suspected carcinogens are evaluated using the 'Incremental Lifetime Cancer Risk', ILCR. This is the increased risk attributed to chemical exposure, above and beyond background cancer risks caused by genetics, lifestyles and other non-chemical factors.

For the purpose of this project, the characterisation of the hazards has relied upon empirical data generated from laboratory tests, toxicological assessments and international reference databases. This process has relied on existing international literature and research for which the hazards have been characterised e.g. human health related hazards (chemical toxicants, carcinogens). The hazard characterisation data (the reference dose (RfD), reference concentration (RfC), oral slope factor, or inhalation unit risk) collated for the water quality constituents forming part of the technology demonstrator and brief literature review of each constituent included in Appendix B.

4.1.5 Risk estimation

The risk-based water quality guidelines for domestic use is presented as a software DSS allowing fitness for use assessments to be performed in site-specific contexts, as well reporting generic water quality requirements per category of use. The DSS has been designed to assess the quantitative risk to a domestic user. The acceptable risk is derived through a mathematical approach (calculation methodology) comprising the risk assessment that accounts for all the assumptions and exposure conditions. A total risk estimate is generated as the output in terms of the risk of adverse effects. The following sub-sections present calculation methodology applied that inform the informatics in the DSS. The calculation adopted in the DSS varies at three levels, *i.e.* in terms of the hazard categorisation/characterisation, the exposure route and the exposure scenario (domestic use and the receptor characteristics considering how the water is experienced). The hazard categorisation is as follows and is the primary determinant of the methodology used:

- Carcinogen (non-threshold – those that do not appear to have a threshold)
- Toxicant (effects are observed only above a certain threshold dose, with no effects observed at doses below this threshold even with lifetime exposure)

Each of calculation methodologies adopted in the DSS are described in the sections below.

4.1.5.1 Estimation of risks associated with ingestion of chemicals

This calculation method is relevant to calculate the exposure risk as a result of ingesting a certain quality of domestic water. For ingestion during bathing and personal hygiene this risk will be much lower due to lower volumes being ingested. In the bathing and personal hygiene case, the ingestion risk will be factorised

based on volume of use. The calculation methodology described below is based on a human health risk assessment that takes into consideration exposure and dosage effects of different constituents on human health. The risk assessment methodology considered is depicted in Figure 4-4. If a chemical has a threshold affect it is considered to have a 'safe' dose where no adverse effects will occur. For these chemicals, a reference dose is derived or calculated based on tolerable daily intakes from which a guideline value will be derived. Developing guidelines for chemicals without a threshold effect (carcinogens) it is assumed that the carcinogenic effect may be induced at any level of exposure and therefore no threshold exists below which it is considered 'safe'.

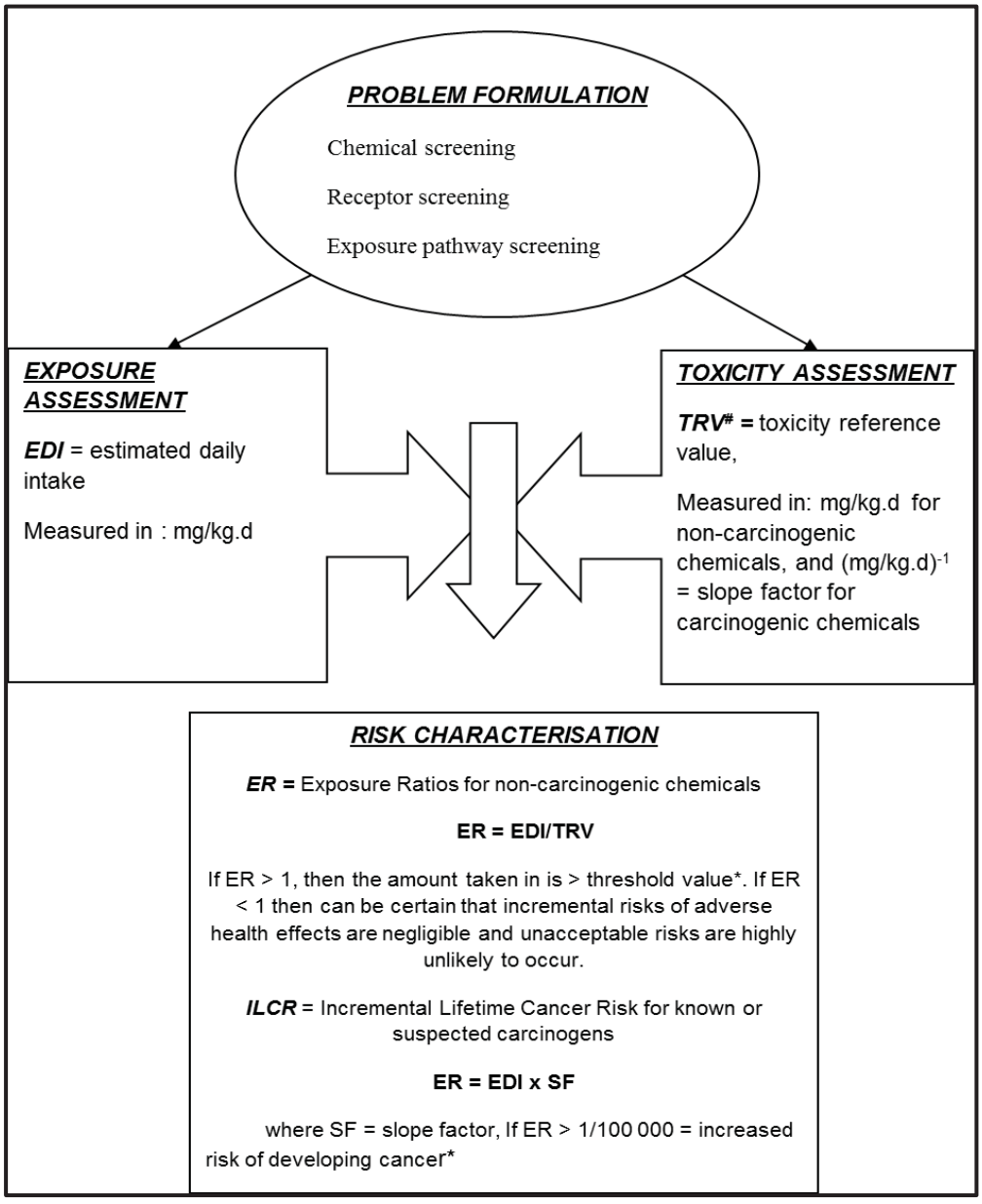


Figure 4-4: Risk assessment and calculation methodology for ingestion

The Canadian risk assessment methodology for Environmental Impact Assessment (EIA) purposes and US EPA guidelines for risk assessment has been adopted. The calculation model is essentially a risk matrix calculation based on:

- Constituent of concern
  - Inorganics;
  - Physical parameters'
  - Metals;
  - Organics;
  - Volatiles; and
  - Microbiological constituents.

- Humans – various age ranges
  - Infant;
  - Toddler;
  - Child;
  - Teen; and
  - Adult.

The methodology is described below:

Step 1

- Determine the water quality constituents (hazards) that require evaluation.
- Determine the receptor (infant, child, adolescent, adult)
- Identify the exposure pathway (e.g. ingestion, inhalation, dermal)

Step 2 – The expected daily intake (EDI) is calculated as follows in mg/kg.d:

$$EDI_{water} = [(C_W \times IR_W \times EF \times ED)/ (BW \times AT \times LE)] \times AF_{GIT}$$

Where: EDI_{water} = exposure due to ingestion of water
C_W = chemical concentration in water (mg/l)
IR_W = receptor water ingestion rate (l/d)
EF = Exposure frequency (d/yr)
AF_{GIT} = Absorption Factor Gastrointestinal tract
ED = Exposure Duration (years) (= 1 for non-carcinogens)
BW = receptor body weight (kg)
AT = 365 days (d)
LE = life expectancy (years) (for assessment of carcinogens only)
AF_{GIT} = 1

For carcinogens in water EDI is calculated as follows in mg/kg.d:

$$EDI_{water} = [(C_W \times IR_W \times AF_{GIT} \times D_2 \times D_3 \times ED)/ (BW \times AT \times LE)]$$

Where D_2 = days/7 days
D_3 = weeks/52 weeks

Step 3 – The exposure ratio is then calculated as follows:

ER (non-carcinogens) = EDI/TRV; where TRV is either the RfD, NOAEL or LOAEL (as discussed in 2.2.2.3)
Acceptable risk: If ER < 1 then the risk is negligible and the water is safe for use. If ER>1 then Step 4 needs to be conducted

ILCR (carcinogens) = (EDI*TRV) X ADAF where TRV = slope factor

Where: ER = Exposure Ratio
TRV = Toxicity reference value
RfD = Reference dose
ADAF = Age dependent adjustment factor

Acceptable risk: If ILCR <10E-05, risk is negligible and the water is safe for use. If ILCR> 10E-05, the risk level for carcinogens is exceeded.

Step 4 – Comparing threshold values for fitness for use

The TRV's can be presented as oral reference doses (RfDs) for non-carcinogenic chemicals. These reference doses are defined as the amount of constituent per unit body weight that can be taken into the body each day, with negligible risk of adverse health effects.

Evaluation of Carcinogens

For carcinogenic chemicals the slope factor is derived from dose-response relationships from epidemiological or animal toxicity studies that have measured the relationship between exposure to substances and incidence of cancer. The evaluation of human risk exposure relevant to carcinogens must be taken into consideration as well. The above calculation methodology takes into consideration the carcinogenic impact. In the absence of adequate data on humans, it is reasonable, for practical purposes [it is biologically plausible and prudent (IARC, 1987)], to regard chemicals for which there is sufficient evidence of carcinogenicity in animals, as if they presented a carcinogenic risk to humans (IARC, 1983; IARC, 1987). Carcinogenic and/or mutagenic effects are seen as non-threshold processes. The equation as per step 3 above, is used to calculate the EDI for carcinogens in a similar manner as for non-carcinogens. The resultant will be an EDI of the contaminant in mg/kg/day, which is then adjusted by the relevant slope factor to derive a carcinogenic risk, reported as the disability-adjusted life years (DALY) in the DSS, (WHO, 2017a) based on a health-based target guideline (acceptable risk level).

The WHO (2017) defines the acceptable carcinogen risk level as "an estimated upper-bound excess lifetime cancer risk of one additional case of cancer per 100 000 of the population ingesting drinking water containing the substance at the set guideline value for 70 years (life expectancy)." This level of risk is much lower than developing cancer from other sources such as genetics, family history and diet (risk level of 25 000 in 100 000; Health Canada, 2006) and some voluntary practices such as smoking (incremental risk of 5 000 in 100 000). An increased risk of 1 in 100 000 ensures that exposure to environmental chemicals does not significantly increase the risk of people developing cancer. The WHO and various countries worldwide have set their acceptable risk level at 10^{-5}. In cases where an excess lifetime cancer risk of 10^{-5} is not feasible or practical because of inadequate analytical or treatment technology, or local circumstances dictate otherwise, the WHO recommends that a provisional guideline value is set at a practical level and the estimated associated cancer risk presented. The acceptable risk of 1 in 100 000 cancer risk has been adopted in the DSS as the acceptable target.

Ingestion: Consumption volume

In terms of the calculation methodology associated with drinking (ingestion) of the domestic water, a consumption volume of 2 L is adopted for adults and elderly; 1 L for children (1-12 years) and 750 ml for infants. This is based on international best practice for the recommended ingestion rate values for use in exposure assessments, and that used for the guideline derivation in the WHO Drinking Water Quality guidelines (2017, 4th edition). This rate includes water consumed in the form of juices and other beverages containing domestic water. The chemical risk calculation for other domestic water uses other than drinking requires an adjustment of the ingestion rate in the methodology. Minimal ingestion is considered in instances such as bathing and personal hygiene, food preparation and household washing. In these uses the default ingestion volume rate of 2 L/day would need to be adjusted to a lower dose to cater for these specific use scenarios. The risk is lower and therefore an appropriate adjustment either by lowering the water ingestion rate and/or the exposure frequency is required to account for the lower risk. For minimal ingestion from bathing and personal hygiene and food preparation, the drinking ingestion volume rate of 2 L/day is reduced in the calculation methodology to estimated ingestion volumes associated with the different water uses as shown in Table 4-1. No empirical data or international literature could be sourced to confirm a specific daily intake volume associated with these activities. The volumes are based estimations relative to the 2 L/day drinking volume, and are conservative.

Table 4-1: Uses of water and associated exposures to water for calculation of risk (modified from WHO, 2016)

Domestic Use Activity	Route of Exposure	Ingestion Volume (ml)/per person
Bathing and personal hygiene	Ingestion	15
Food preparation	Ingestion	500

Age and Body weight:

Life stages are defined in the risk assessment as periods of life with distinct anatomical, physiological, and behavioural or functional characteristics that contribute to potential differences in vulnerability to environmental exposures. Infancy is the period from birth through the first birthday; child encompasses all early postnatal life stages from birth until adolescence, which occurs approximately between 12 and 21 years of age. The continuum between the reproductive-age adult and aged adult begins at approximately 21 years of age and reaches aged adulthood at approximately 65 years. The calculation methodology adopted is based on these life stages in line with international best practice (EPA and WHO) for exposure assessments. The default body weights adopted by the calculation methodology in the DSS is as follows:

- Infant (0-1 years): 5 kg
- Child (1-12 years): 35 kg
- Adolescent (12-21 years): 45 kg
- Adult: 60 kg.

4.1.5.2 Estimation of risks associated with inhalation

Volatile substances in water may be released into the atmosphere during showering and through household washing and flushing of toilets with grey water. For the inhalation risk as a result of bathing, personal hygiene and flushing, Exposure Ratio (ER) is calculated using RfC and not RfD, and the same methodology as for the ingestion risk is followed (see Section 4.1.5.1). The chemical concentration that enters the nostrils (at the boundary of the body) during inhalation of the volatile substances present in the water is higher than

the amount of the constituent that actually enters the body through the upper respiratory tract and lung, (US EPA, 2011). However, the conservative case air volume ingested at the *boundary* will be used in the DSS calculations. Reference concentrations (RfCs) is used to compare against the constituent concentration in the inspired air. US EPA, 2011 also recommends that the concentration of the constituent in the air in mg/m^3 (mg/l) be used instead of the intake of a contaminant in air based on inhalation rate and body weight (mg/kg-day). It should also be noted that based on their size, physiology, behaviour and activity level, the inhalation rates of children differ from those of adults. Infants and children have a higher resting metabolic rate and oxygen consumption rate per unit of body weight than adults because of their rapid growth and relatively larger lung surface area (SA) per unit of body weight, (US EPA, 2011). A distinction is made between long- and short-term inhalation rates in US EPA, 2011. Long-term inhalation rates for both adult and children are presented as daily rates in m^3/day and is defined as repeated exposure for more than 30 days up to approximately 10% of the life span in humans. Short-term exposure is repeated exposure for more than 24 hours up to 30 days. For exposure from domestic water use it is expected and assumed that the "contaminated" water will be used repeatedly for at least up to a year period, hence the long-term exposure rates will be considered. These volumes will be adjusted to account for the limited exposure to the domestic water use as compared to constantly breathing in contaminated air. Therefore the m^3/day value as reported in Table 4-2 column 2, will be divided by 48 (as shown in column 3 of Table 4-2) since it is assumed that the adult or child will only be exposed to the water vapour for a 30 minute period per day.

Table 4-2: Recommended long-term exposure values for inhalation (males and females combined) (Source: Exposure Factor Handbook, US EPA, 2011)

Age group	Mean Inhalation volume (m^3/day)	Adjusted inhalation volume for domestic use (m^3/30 min)
Birth to <1 month	3.6	0.075
1 to <3 months	3.5	0.073
3 to <6 months	4.1	0.085
6 to <12 months	5.4	0.113
Birth to <1year	5.4	0.113
1 to <2 years	8.0	0.167
2 to <3 years	8.9	0.185
3 to <4 years	10.1	0.210
6 to <11 years	12.0	0.250
11 to <16 years	15.2	0.317
16 to < 21years	16.3	0.340
21 to < 31years	15.7	0.327
31 to < 41years	16.0	0.333
41 to < 51years	16.0	0.333
51 to < 61years	15.7	0.327
61 to < 71years	14.2	0.296
71 to < 81years	12.9	0.269
≥ 81 years	12.2	0.254

Calculation procedures:

Step 1 is calculated as for ingestion (see Section 4.1.5.1).

Step 2 in the calculation for EDI, the ingestion rate of water of 2 L/day is replaced with the following inhalation volume for the calculation. These values are taken from Table 4-2 and average inhalation volumes for the age groups below:

- Infant (birth to 1 year) – 0.092 m^3/day
- Child (1 to 12 years) – 0.226 m^3/day
- Adolescent (12 to 21 years) – 0.329 m^3/day
- Adult (>21years) – 0.306 m^3/day

Step 3 – The exposure ratio is then calculated as follows:

ER (non-carcinogens) = EDI/TRV; where TRV is the RfC or NOAEL or LOAEL (as discussed in section 2.2.2.3).
Acceptable risk: If ER < 1 then the risk is negligible and the water is safe for use. If ER>1 then Step 4 needs to be conducted.

ILCR (carcinogens) = (∑EDI*TRV) x ADAF where TRV = slope factor
Acceptable risk: If ILCR <10E-05, risk is negligible and the water is safe for use. If ILCR> 10E-05, the risk level for carcinogens is exceeded.

Step 4 – Comparing threshold values for fitness for use

The TRVs can be presented as inhalation reference concentrations (RfCs) for non-carcinogenic chemicals. These reference doses are defined as the amount of constituent per unit body weight that can be taken into the body each day, with negligible risk of adverse health effects.

4.1.5.3 Estimation of risks associated with dermal exposure

Constituents that may be hazardous as a result of dermal contact will be addressed as per the ingestion calculation (see Section 4.1.5.1). Some substances may be absorbed through the skin during bathing and during the use of grey water for flushing, but this is not usually a major source of uptake. Dermal exposure presents similar risk to ingestion as once the water is absorbed through the dermal layer the risk is the same as for ingestion of the constituent of concern (same pathway of exposure). The consequence is however smaller and takes a longer period to manifest. The use of the ingestion calculation is considered adequate for the purposes of the risk-based guidelines quantification in the DSS. The risk as discussed is lower and therefore an appropriate adjustment by lowering the water ingestion rate is adopted to account for the lower risk. According to US EPA, 2007, exposure is described as the amount of an agent that contacts the outer boundary of the body (dose) and is capable of being distributed to one or more organs to exert a toxic effect (target dose). The amount of exposure will depend on the concentration of the chemical contacting a given area of skin – the dermal loading or skin adherence, the ability of the chemical to penetrate and pass through the skin – dermal dose and the duration and frequency of contact in terms of the intervals of contact and the number of intervals per day, weeks, months or even a lifetime. For the case of the domestic use guidelines, the duration and frequency of contact can be summarised as:

- Intervals of contact – 0.5 hour
- Number of intervals per day – 1

In dermal exposure assessment, the contaminant concentration is the amount of chemical contaminant in the water that is available for contact that can be deposited on the skin during a given activity (domestic use scenario). The Office of Water (OW) as stated in US, EPA, 2007, has not established assessment criteria as yet for contaminants present in drinking water. US EPA, 2007, however, provides assessment from the Office of Solid Waste and Emergency Response (OSWER), Office of Superfund Remediation and Technology Innovation (OSRTI)/ OSWER has developed some guidance to address the dermal exposures of toxic chemicals that result from contact with contaminated water. Only those chemicals that contribute more than 10% of the dose may occur from water ingestion are considered sufficiently important for the dermal exposure risk assessment. For dermal water pathways, the dermal absorbed dose that results from the contact of chemicals in contaminated water is calculated as:

$$DAD = (DA_{event} \times ED \times EV \times EF \times SA) / (BW \times AT)$$

Where DAD = dermal absorbed dose (mg/kg-d)
DA_{event} = absorbed dose per event (mg/cm^2 – event)
SA = skin surface area available for contact (cm^2)
EV – event frequency (events/d)
EF – exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)
AT = averaging time (d)

Where $DA_{event} = K_p \times C_w \times t_{event}$
K_p = dermal permeability coefficient (cm/h)
C_w = concentration in water (mg/l)
t_{event} = event duration (h/event)

For carcinogens in contaminated water dermal risk is calculated as follows:

DALY = DAD X slope factor

Where DAD = dermal absorbed dose (mg/kg-d)
DALY = Daily adjusted life years

The OSWER: Superfund uses adults (>18 years) of body weight 70 kg and children (1-6 years) of body weight 15 kg. The body weight adopted in the DSS is 60 kg for adults and elderly, 45 kg for adolescents (12-21years), 35 kg for children (1-12 years) and 5 kg for an infant (< 1year).

Skin is exposed during bathing for adults and children is estimated at 6600 cm^2 and for other activities at 2 800 cm^2 which includes head, hands, forearms, lower legs and feet exposed (US EPA, 2007). For the domestic use activities, the following dermal surface area exposed is adopted in the DSS to calculate the exposure dose;

- Bathing – 6 600 cm^2
- Laundry – 2 800 cm^2
- Household use – 2 800 cm^2

The value of K_p for inorganics ranges from 0.0006 to 0.002 cm/h for metals, except mercury vapour which is 0.24 cm/h. For all other inorganics, K_p is given as 0.001 cm/h. For organics it was assumed that K_p is 0.001 cm/h.

4.2 EXPOSURE TO BIOLOGICAL CONTAMINANTS

4.2.1 Overview

Generally, water-transmitted pathogens include viral, bacterial and protozoan pathogens. These microorganisms may result from sewage effluents; the recreational population using the water (which can be directly from faecal material or shedding as a result of immersion in water); livestock (cattle, sheep, etc.); industrial processes; farming activities; domestic animals and wildlife. These pathogens are represented by Norovirus (virus), Campylobacter (bacterial) and Cryptosporidium (protozoa) (Mara and Bos, 2010). These groups of pathogens differ in their ability to survive in the environment. Viruses are the smallest of the waterborne pathogens and are unable to multiply outside the host cell. They are more resistant to environmental inactivation than most pathogenic bacteria and have a lower infective dose. The number of microorganisms (dose) that may cause infection or disease depends on the specific pathogen, the conditions of exposure and the host's susceptibility and immune status. For viral and parasitic protozoan illness, this dose might be very few viable infectious units (Fewtrell *et al*., 1994; Teunis, 1996; Haas *et al*., 1999; Okhuysen *et al*., 1999; Teunis *et al*., 1999). The types and numbers of pathogens in the environment will differ depending on the incidence of disease and carrier states in the contributing human and animal populations and the seasonality of infections. As a result, numbers will vary greatly in different communities and times of year. A general indication of pathogen numbers in raw sewage is given in Table 4-3 to provide an indication of sewage as a contribution to surface water quality (Modified from WHO Guidelines for Safe Recreational Water Environments, 2006 and WHO Drinking Water Quality Guidelines 2017).

Table 4-3: Quantities of organisms present in wastewater as an indication of contribution to surface water quality (Modified from WHO Guidelines for Safe recreational Water Environments, 2006 and WHO Drinking Water Quality Guidelines 2017)

Organism / Pathogen	Numbers in sewage / 100 mL	Numbers in Surface Water /100 mL
Bacteria		
Campylobacter	10^4-10^5	10-1 000
Clostridium perfringens spores	6 x 10^6-8 x 10^8	
Escherichia coli	10^6-10^7	10-10 000
Faecal streptococci	5 x 10^3-4 x ^{105}	
Salmonella spp	0.2-8.0 x 10^3	
Shigella spp	0.1-1 x 10^3	
Viruses		
Poliovirus	180-5 x 10^5	0.001-1
Rotavirus	4.0 x 10^2-8.5 x 10^4	0.001-10
Adenovirus	1.15 x 10^5 (gene copies)	
Norovirus	4 x 10^3 (gene copies)	
Hepatitis viruses	5.1 x 10^1 (gene copies)	
Parasites		
Cryptosporidium parvum oocysts	0.1-39	0-100
Giardia lamblia cysts	12.5-3.0 x 10^4	0-100

Pathogens might be at concentrations too low to be detected and still pose a risk to public health (Signor & Ashbolt, 2006; Smeets *et al*., 2007). Microbial risks are therefore often assessed by modelling within a QMRA process. For example, pathogen monitoring data from surface water sources may have a large number of non-detects even when the water sources are known to be influenced by faecal sources. This is often due to the event-driven nature of microbial loading and the limitations of small monitoring data sets to capture these events. Modelling the pathogen concentration in faecal sources, followed by hydrologic modelling of contamination events, may therefore provide more useful information for QMRA than relying on monitoring data alone (Ferguson *et al*., 2007; Ashbolt *et al.*, 2010; Sokolova *et al*., 2015).

4.2.2 Quantitative microbial risk assessment (QMRA)

The quantitative microbial risk assessment (QMRA) is adopted for calculation of the microbiological hazards. The QMRA is an approach that combines scientific knowledge about the presence and nature of pathogens, their routes of exposure to humans and the possible health effects into a single assessment that allows evidence-based, proportionate and transparent management of the risk of waterborne infectious disease transmission. QMRA has developed as a scientific discipline over the last two decades and has been embedded in the WHO water-related guidelines (WHO, 2001, 2006a&b, 2017). The Quantitative Microbial Risk Assessment (QMRA) is adopted as the basis of the calculation methodology adopted in the DSS. The approach used in these risk-based guidelines is the Quantitative Microbial Risk Assessment approach or QMRA used by the World Health Organisation in their Drinking Water Quality Guidelines (WHO, 2017a); Potable Reuse-Guidance for Producing Safe Drinking-Water (WHO, 2017b) and Quantitative Microbial Risk Assessment: Application for Water Safety Management (WHO, 2016). The process of QMRA is derived from the chemical risk assessment paradigm that encompasses the four steps of risk assessment, namely: hazard identification and characterisation; exposure assessment; dose-response assessment and lastly risk characterisation (WHO, 2016, 2017a, 2017b, US-EPA, 1993). The four steps of the harmonized QMRA framework comprise:

- problem formulation; where the overall context such as reference pathogens, exposure pathways, hazardous events and health outcomes is defined,
- exposure assessment; where the magnitude and frequency of exposure to each reference pathogen are quantified (dose calculated based on water quality and consumption volume)
- health effects assessment; where dose is linked to probability of infection or illness and probability of illness are identified for each organism; and
- risk characterization where a quantitative measure of risk is generated.

The QMRA approach provides an estimate of the probability of infection based on the number of pathogens ingested (dose) and pathogen specific dose-response models based on data from human volunteer or outbreak studies to provide an estimate of the probability of infection associated with that exposure. Including some simplifying assumptions to the relationship between numbers of organisms ingested and infection, simple dose-response relationships were derived and are given in Table 4-4.

The WHO (2016, 2017b) suggests using low-dose approximations of the QMRA formulae with outcomes of the two models reported to be similar. The dose dependent models adopted by WHO (2017) were used in these Water Quality Guidelines (Table 4-5). At very low concentrations, this simplification is valid; however, at higher pathogen concentrations the full single-hit (exponential or Beta-Poisson) model should be used to account for the risk of infection associated with exposure to more than one organism (For more specific data refer to WHO, 2016).

Table 4-4: Probability of Infection Models

Daily risk (probability) of infection	
Beta-Poisson Model (WHO, 2001) $$\boldsymbol{Pi = 1 - [1 + \frac{dose}{\beta}]^{-\alpha}}$$ and $$\boldsymbol{N_{50} = \beta * [2^{1/\alpha} - 1]}$$ therefore $$\boldsymbol{P_i = 1 - [1 + \frac{d}{N_{50}}](2^{1/\alpha} - 1)^{-\alpha}}$$	Exponential model (Haas, 1996) $$P_i = 1 - e^{-rN}$$
Pi = probability (risk) of infection ***d*** = dose or exposure (number of organisms ingested based on consumption of water (l) per day β = parameter characterised by dose-response relationship α = parameter characterised by dose-response relationship N_{50} = median infectious dose **r** = parameter characterised by dose-response relationship	

Table 4-5: Low-dose approximation formulae

Reference pathogen	**Campylobacter**	**Norovirus**	**Cryptosporidium**
Low-dose extrapolation formula	$Pinf = \frac{\alpha}{\beta} \times dose$	$Pinf = \frac{\alpha}{(\alpha + \beta)} \times dose$	$Pinf = r \times dose$
Dose-response parameters	α = 0.145 β = 7.58 Approximate beta Poisson	α = 0.0044 β = 0.002 Hypergeometric	r = 0.2 Exponential

4.2.3 Probability of infection based on multiple exposures:

Multiple or long-term exposures result in a probability of infection calculated based on the number of exposures (events) expected to occur over a year (WHO, 2006). The method of calculating the annual probability of infection based on multiple exposures is presented in Table 4-6. This method of calculating annual risk of infection assumes a constant water quality (and therefore a constant daily dose) which is not a true reflection of annual risk. Additional methods of calculating annual risk are available that take variability of water quality and dose into account (and therefore daily risk of infection). The alternate annual risk formula is based on the product of independent daily infection probabilities and allows for variation in daily risk of infection (Benke and Hamilton, 2008). Adjustments to this formula have been proposed by other researchers to account for variations (Karavarsamis and Hamilton, 2010).

Table 4-6: Probability of infection based on multiple exposures

$P\ multiple = 1 - \ (1 - P\ inf)^n$,
where *n* is the number of times exposure occurs. For example, monthly exposure, *n* = 12; daily exposure, *n* = 365 and for weekly exposure, *n* = 52.

The numerous dose estimates in the calculations can be either direct measurements or generated through simulation of an exposure model. As it is not practical to have daily samples analysed and simulation tools are not always accessible for conducting a risk-based assessment, uncertainty and variability are ignored in this calculation of annual risk (Karavarsamis and Hamilton, 2010), but are acknowledged. The probability of infection involves the multiplication of microorganisms such as bacteria, viruses, and parasites. An infection may cause no symptoms and be sub-clinical, or it may cause symptoms and be clinically apparent. The different outcomes resulting from exposure to microbial pathogens is illustrated in the following diagram. Various models exist for the dose-response relationship for infection, and the dose-response relationship for illness when infected, is depicted in Figure 4-5.

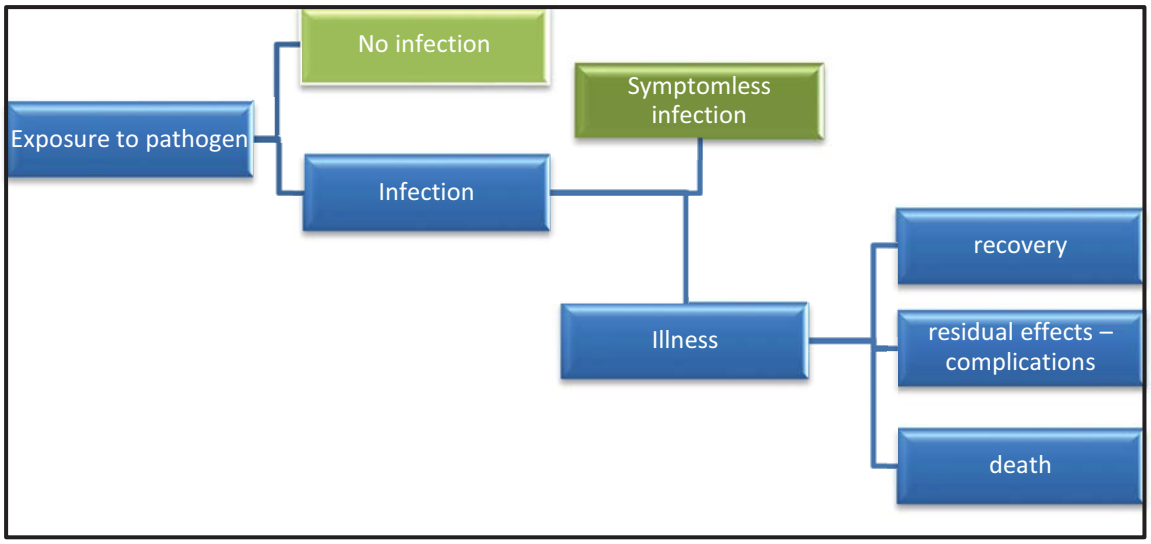


Figure 4-5: Disease Progression Model (Adapted from Pruss and Havelaar, 2003)

Depending on the type of pathogen of interest, different surrogate organisms can be used in assessing probabilities of infection. The risk of virus infection is usually higher than for bacteria and parasites. Viruses can persist for long periods in water and have low infective doses. Rotaviruses, enteroviruses and noroviruses have been identified as potential reference pathogens in QMRA. Rotaviruses and Noroviruses are the most important cause of gastrointestinal infection in children and can have severe consequences, including hospitalization and death, with fatality rates being more frequent in low-income regions. Typically, viruses are excreted in very large numbers by infected patients, and waters contaminated by human waste could contain high concentrations. QMRA can be used to characterise risks associated with a particular pathogen to calculate a concentration of a specific pathogen that would correspond to a pre-specified level of risk, or to evaluate the relative ranking of pathogen/exposure combinations.

Many studies have been undertaken in which dose-response models have been fitted to experimental data. The most common pathogens used as reference pathogens in QMRAs for managing water quality include:

- Campylobacter
- *E. coli*
- Enteroviruses
- Adenovirus
- Rotavirus
- Norovirus
- *Giardia lamblia*
- *Cryptosporidium*
- *Campylobacter*

The reference pathogens used as examples in the Drinking Water Quality Guidelines (WHO, 2017a) are *Campylobacter*, rotavirus and *Cryptosporidium*. The introduction of a rotavirus vaccine however is changing the incidence and severity of disease outcomes from this pathogen complicates the use of it as a reference pathogen (Gibney et al., 2014). Norovirus, which fulfils the requirement of a reference pathogen, is therefore a suitable alternative to use as a reference pathogen. Norovirus causes about 18% of acute diarrhoeal disease globally with similar proportions in high- and low-income settings (Lopman et al., 2015) and is a common cause of waterborne outbreaks (Guzman-Herrador et al., 2015; Moreira *et al.,* 2016). Norovirus is used in these domestic guidelines as a reference organism.

4.2.4 Microbiological (Infectious agent)

Because it is not feasible to test water for all potential waterborne pathogens, including bacteria, viruses, protozoa and helminths a more practical approach is needed which identifies reference pathogens to represent groups of pathogens. Variations in characteristics, behaviours and susceptibilities of each group must be taken into account to represent different pathogenicity and survival characteristics. Depending on the type of pathogen of interest, different surrogate organisms can be used in assessing probabilities of infection for organisms other than viruses, although the risk of virus infection is much higher than for bacteria and parasites. Viruses can persist for long periods in water and have low infective doses. Rotaviruses, enteroviruses and noroviruses have been identified as potential reference pathogens in QMRA. Rotaviruses and Noroviruses are the most important cause of gastrointestinal infection in children and can have severe consequences, including hospitalization and death, with fatality rates being more frequent in low-income regions. Typically, viruses are excreted in very large numbers by infected patients, and waters contaminated by human waste could contain high concentrations. In addition to the epidemiological studies providing the correlation to faecal indicators, QMRA can be used to characterise risks associated with a particular pathogen to calculate a concentration of a specific pathogen that would correspond to a pre-specified level of risk, or to evaluate the relative ranking of pathogen/exposure.

The disability-adjusted life year (DALY) measure is often used to develop health-based guidelines in the food industry and more recently for water quality guidelines. The DALY is a common “metric” that can be used to quantify and compare the burden of disease associated with different water-related hazards, taking into account varying probabilities, severities and duration of effects. The DALY is used by the WHO to evaluate public health priorities and to assess the disease burden associated with environmental exposures. A tolerable burden of disease (or acceptable risk) must be defined to calculate allowable levels of microbial contamination. The concept of tolerable disease burden (equated to acceptable risk) was set out in the fourth edition of the Guidelines for Drinking Water Quality or GDWQ (WHO, 2011).

The guidelines defined the tolerable burden of disease as an upper limit of 10^{-6} disability-adjusted life year (DALY) per person per year. This measure takes into account illness, premature death and life lived with a disability. The DALY is calculated by adding the years of life lost (YLL) as a result of premature death and time spent with an illness (years lost due to disability or YLD). YLD takes into account the number of cases, how long the cases typically last and how severe the symptoms are, ranging from 1, the most severe for death and 0, for good health. The DALY allows for quantification and comparison of the burden of diseases between countries, areas, population groups and different diseases. The DALY = YLL+YLD. The burden of a single case of disease is calculated to determine the tolerable number of disease cases per year. The WHO DWQGs define safe drinking water as not representing any risk to health over a lifetime of consumption, setting the tolerable disease burden at 10^{-6} DALYs per person year (WHO, 2004, 2011 and 2017). One DALY per million people per year approximately compares to one cancer death per 100 000 in a 70-year lifetime – which is the benchmark often used in chemical risk assessments (WHO, 2004) as the acceptable risk level. This level of health burden is equivalent to a mild illness such as watery diarrhoea with a low fatality at an approximately 1 in 1000 annual risk of disease to an individual (WHO, 1996; Havelaar & Melse, 2003).

A "tolerable" risk of 10^{-6} DALY per person per year allows for the loss of 365 healthy days in a population of one million over the course of one year. The 10^{-6} DALY tolerable burden of disease target may not be achievable or realistic in some locations and circumstances in the near term. Where the overall burden of disease by multiple exposure routes (water, food, air, direct personal contact, etc.) is very high, setting a 10^{-6} DALY per person per year level of disease burden from waterborne exposure alone will have little impact on the overall disease burden. Setting a less stringent level of acceptable risk, such as 10^{-5} or 10^{-4} DALY per person per year from waterborne exposure may be more realistic, yet still consistent with the goals of protecting public health. Concentrations of pathogens equivalent to a health outcome target of 10^{-6} DALY per person per year are typically less than 1 organism per 10 000-00 000 litres making it more feasible and cost-effective to monitor for indicator organisms such as *E. coli*. QMRA is a sensitive tool that can estimate the probability of infection that could not be measured through epidemiological studies and is a complement to epidemiological studies. QMRA predicts infection or illness rates based on the measured or predicted densities of a specific pathogen combined with ingestion rates of water associated with different activities. The estimated ingestion volumes associated with different water uses for microbiological risk are shown in Table 4-7.

Table 4-7: Uses of Water and associated exposures to water (microbiological risk) (modified from WHO, 2016)

Activity	Route of exposure	Volume (ml)	Frequency per person per year	Comments
Garden irrigation	Ingestion via sprays	1	90	Indirect ingestion through contact with plants
Food crop consumption of home-grown food	Ingestion	11	50	
Toilet flushing	Ingestion via sprays	0.01	1100	Based on 3 flushes per day
Laundry	Ingestion via sprays	15		

The concentrations of three groups of reference pathogens (representing bacterial, viral and parasite pathogens) equivalent to 10^{-6} DALYs per person per year (DALY) are calculated as depicted in Figure 4-6 with formulae and parameter values shown in Table 4-8. Concentrations of pathogens equivalent to a health outcome target of 10^{-6} DALY per person per year are typically less than 1 organism per 10^4-10^5 litres. Therefore, it is more feasible and cost-effective to monitor for indicator organisms such as *E. coli*. Because QMRA is a sensitive tool that can estimate the probability of infection that cannot be measured through epidemiological studies, it complements epidemiological studies. QMRA predicts infection or illness rates based on the densities of a specific pathogen and predicted or measured ingestion rates of water associated with different activities.

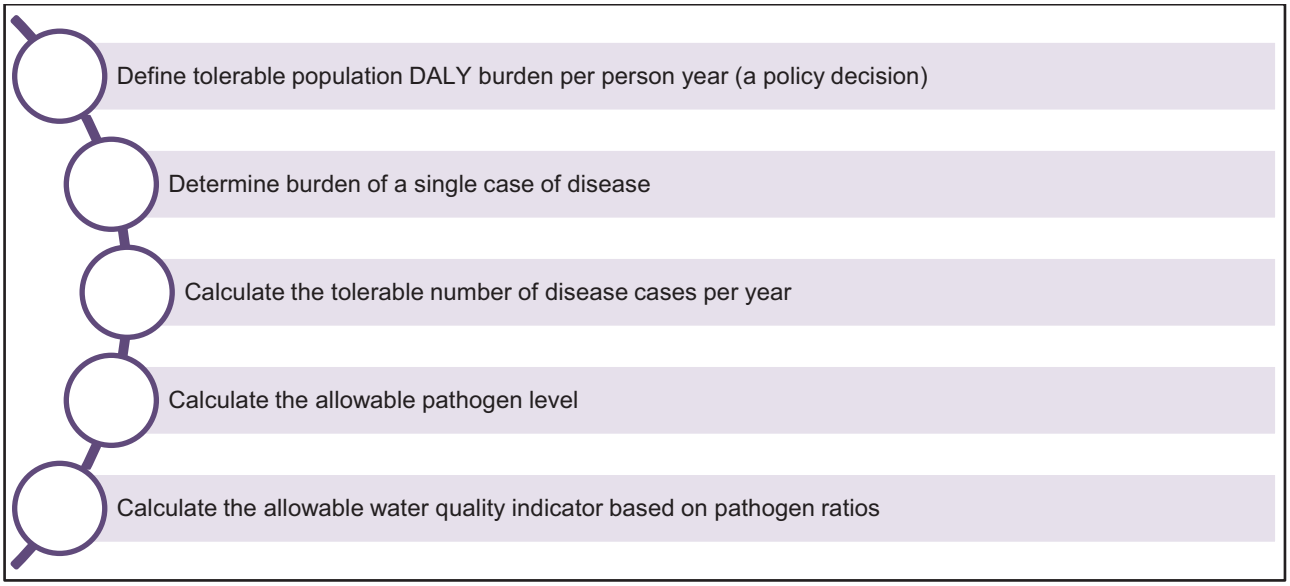


Figure 4-6: Steps in calculating target guidelines

Table 4-8: Reference pathogen parameters used in DALY and probability of infection calculations (Source FAO /WHO, 2003; WHO, 2017b)

Reference pathogen	Campylobacter	Norovirus	Cryptosporidium
Low-dose extrapolation formula	$Pinf = \frac{\alpha}{\beta} \times dose$	$Pinf = \frac{\alpha}{(\alpha+\beta)} \times dose$	$Pinf = r \times dose$
Dose-response parameters and distribution model	α = 0.145 β = 7.58 Approx. Beta Poisson	α = 0.0044 β = 0.002 Hypergeometric	r = 0.2 Exponential
Probability of infection from a single organism	0.019	0.69	0.2
Likelihood of becoming ill if infected	0.3	0.7 (0.8)	0.7
Disease Burden per case	$2.4X10^{-2}$	$5 X 10^{-4}$	$1.7X10^{-3}$

Health based targets can be set in different ways. They can be determined using:

- epidemiological studies, QMRA (and DALY approach);
- water quality (*E. coli*) monitoring approach;
- performance targets for treatment requirements, and
- specific treatment requirements.

These risk-based water quality guidelines for Domestic Use, makes use of *E. coli* levels in the DSS to calculate protection from Norovirus infection as this protects against bacterial and parasite infections. Norovirus is recognised as one of the most common agents of viral diarrhoea. Although the risk of infection by norovirus would usually be modelled based on measured or modelled norovirus particles, here the risk of norovirus infection per person per year is determined using *E. coli* counts per 100 ml. This is used to estimate a norovirus concentration to predict the probability of illness established using Norovirus dose-response parameters (Teunis *et al.*, 2008) (Figure 4-7 and Table 4-9).

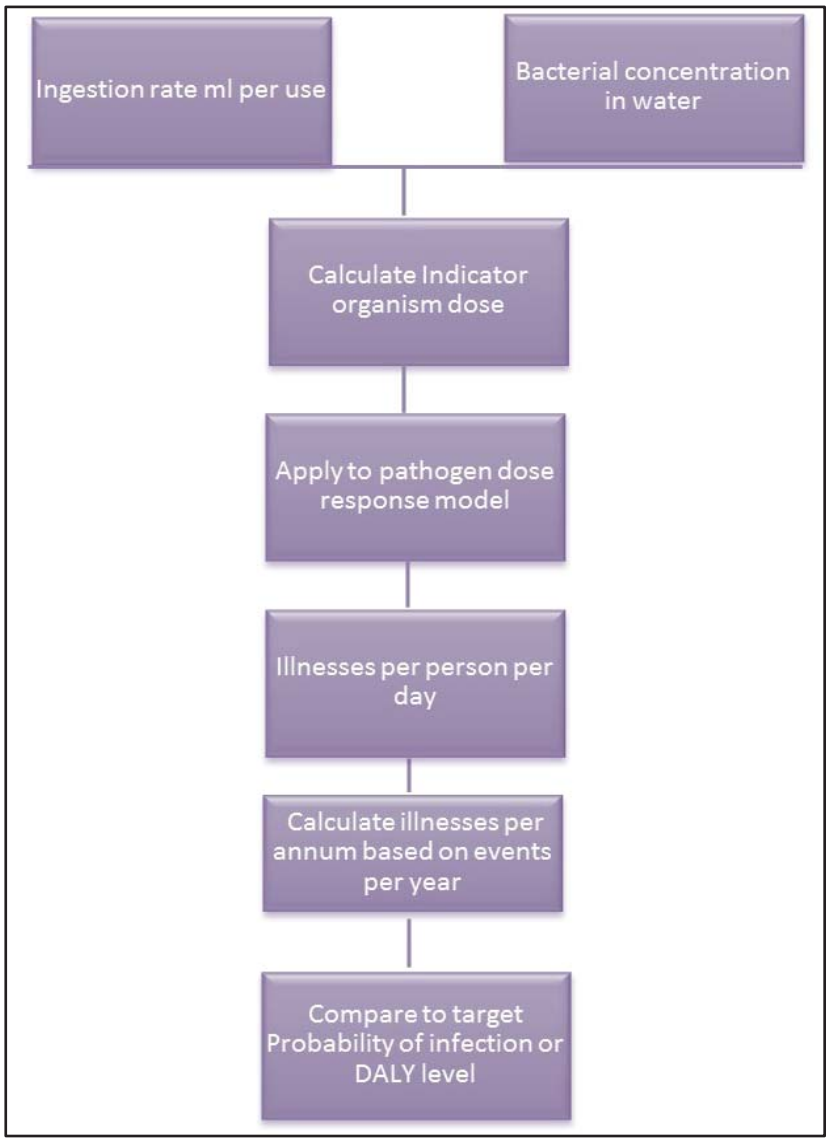


Figure 4-7: Flow chart illustrating steps to calculate the probability of infection to meet a health-based target

Table 4-9 shows that a norovirus dose of 1.14 X10^{-5} noroviruses per L (or 0.00001142/L) is the target concentration to satisfy the 10^{-6} DALY. If the ratio of norovirus to *E. coli* is 1 to 100 000, less than 1 *E coli* /100 ml of water is the target value. Table 4-10 illustrates *E. coli* levels and associated probability of infections and DALYs. If water used for drinking water contains 1 *E. coli* /100 ml the DALY is 1.25 X10^{-5} which is more than 10 times higher than the "target" 10^{-6} DALY. The WHO Drinking Water Quality guidelines (WHO, 2017a) do not make use of indicator organism concentrations in the QMRA approach, but instead make use of treatment targets based on QMRA and the quality of untreated water. Calculating the probability of infection on a population level using raw (untreated source) water quality is used together with the target DALY or risk level to determine the level of treatment needed to ensure water safety. For example, if 20 000 / L noroviruses are present in the raw water, a 9.5 log reduction is required to meet the 10^{-6} – DALY pppy target. This calculation methodology based on the tolerable burden of disease has been adopted in the DSS. The WHO health-based guideline defined as an acceptable risk a tolerable burden of disease of 10^{-6} DALY per person per year) is used as the target guideline in the DSS to determine microbiological risk. The concentration of *E. coli* in the sample is used to calculate protection from Norovirus infection as this protects against bacterial and parasite infections. Norovirus is recognised as one of the most common agents of viral diarrhoea. Although the risk of infection by norovirus would usually be modelled based on measured or modelled norovirus particles, here the risk of norovirus infection per person per year is determined using *E. coli* counts per 100 ml. The *E. coli* count is converted to a dosage per litre of predicted Norovirus dose based on a dose response function, and the individual risk and annual risk of infection is then determined per person per year, represented as the DALY.

Table 4-9: *E. coli* target value calculation based on Norovirus DALY dose and probability of infection / illness targets

Calculation of Norovirus equivalent to 10^{-6} DALYs pppy	
Probability of *infection* per 1 organism	0.69
Illness rate per infection	0.7
Therefore, probability of *illness* per 1 organism	0.483
Disease Burden per case	0.0005
Number of exposure events assumed per year	365
Target Norovirus dose for 10^{-6} DALY target	1.14 X 10^{-5}
Target *E coli* dose/100 ml based on 1:10^5 ratio norovirus to *E coli* (ingested volume dependent)	<1
Where Target dose for specific DALY $= \frac{(Target\ DALY)}{Prob\ illness\ 1\ org \times DB \times 365}$ **= number of organisms permitted per volume ingested**	

Table 4-10: *E. coli* levels and associated probability of infections and DALYs

***E coli* /100 ml**	**Volume (ml) ingested based on water use**	**Calculated *E. coli* dose**	**Predicted Norovirus Dose (no. of organisms)**	**Probability infection**	**No. of events per annum**	**Annual probability of infection**	**DALY**
0.1	1000	1	0.00001	6.88E-06	365	2.51E-03	1.25E-06
1	1000	10	0.0001	6.88E-05	365	2.48E-02	1.25E-05
5	1000	50	0.0005	3.44E-04	365	1.18E-01	6.27E-05
10	1000	100	0.001	6.88E-04	365	2.22E-01	1.25E-04

An ingestion volume of 1 L per day (unboiled) is assumed for calculation methodology for microbial risk, based on the default ingestion rate of 2 L per day for adults. It is assumed that the additional 1 L of water per day that is consumed has been boiled, and thus the microbial risk is negligible. According to the WHO (2017) a tolerable burden of waterborne disease from drinking water is suggested as 10^{-6} DALY per person per year. This has been adopted in the DSS as the acceptable risk target.

4.3 RISK REPORTING RELATED TO HUMAN HEALTH

For human health related adverse effects experienced through ingestion, inhalation and dermal contact exposure routes; human health toxicological exposure assessment threshold limits apply. For the risk-based guidelines pertaining to toxicants a four-level categorisation of the threshold limit criteria apply. The threshold risk criteria adopted in the DSS are defined in Table 4-11 with each marking a distinction in the fitness for use category. The threshold limit criteria represent how the adverse effects and likelihood of occurrence of the risk would be linked to the fitness for use each category. A risk estimate (as a percentage) has been defined based on these threshold limit criteria and represents the probability of occurrence and severity of the end point effect as shown in Table 4-12. Note that endpoints and adverse effects are available for the NOAEL and LOAEL fitness for use ranges. For the endpoint of the tolerable range, no literature-based values are available as this threshold limit is not commonly determined for exposure assessments. For the purposes of the risk reporting and the four-level categorisation required in the DSS an arbitrary threshold limit between NOAEL (acceptable limit) and LOAEL (unacceptable limit) has been interpolated. Risks will therefore be reported relative to these effects and levels.

A **No-Observed-Adverse-Effect Level (NOAEL)** is the highest exposure level at which there are no biologically significant increases in the frequency or severity of adverse effect between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered adverse or precursors of adverse effects.

The **Lowest-Observed-Adverse-Effect (LOAEL)** refers to the lowest exposure level at which there are biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group.

The **reference dose (RfD)** is an oral or dermal dose, and the RfC, an inhalation reference concentration, are derived from the LOAEL or NOAEL by application of order of magnitude uncertainty factors. The RfD is defined as an estimate of a daily oral exposure to the human population (include sensitive populations) that is likely to be without an appreciable risk of harmful effects during a lifetime. These uncertainty factors take into account the variability and uncertainty that are reflected in the possible differences between test animals and humans (generally 10-fold or 10x) and variability within the human population (another 10x) (US EPA). For the human health risk-based guidelines pertaining the carcinogens and microbial infectious agents a two-level categorisation based on acceptable risk applies. The WHO acceptable risk targets adopted in the DSS are shown in Table 4-13.

Table 4-11: Risk rating, probability and severity of effect for toxicants

Risk estimate (Percentage)	Probability of Occurrence	Severity of the effect
<1	None	None
1-5	Rare	Negligible
>5-15	Possible	Minor
>15-100	Certain	Significant

Table 4-12: Threshold limit criteria defining the fitness for use categories for the toxicant human health risk reporting

Fitness for use Category	Threshold limit criteria	Risk estimate (%)
Ideal	< RfD/RfC	<1
Acceptable	> RfD/RfC; <NOAEL	1-5
Tolerable	>NOAEL; < LOAEL	>5-15
Unacceptable	> LOAEL	>15-100

Table 4-13: Acceptable risk and DALY risk targets defining the fitness for use categories for the carcinogens and microbial agents risk reporting

Fitness for use	Threshold limit criteria (Acceptable risk level)
Below acceptable risk target	$< 10^{-6}$ DALY target per person per year (microbial) $< 10^{-5}$ lifetime risk of cancer
Above acceptable risk target	$> 10^{-6}$ DALY target per person per year (microbial) $> 10^{-5}$ excess lifetime risk of cancer

The DALY (disability-adjusted life years) is a common metric that is used to quantify the burden of disease associated with water related hazards, which takes account of probabilities, severities and duration of effects. The DALY accounts for the impact on the quality and quantity of life and focuses on the health outcome. The DALY is adopted by the WHO in setting health-based targets where health outcomes must be quantified. DALYs can be used to define tolerable burden of disease and related reference level of risk (WHO, 2017a). The 'Tolerable burden of disease' (or reference level of risk) represents the upper limit of the burden of the health effects associated with the disease. The WHO tolerable burden of disease target applicable to the risk of cancer is 10^{-5} lifetime risk and microbial risk is 10^{-6} DALY per person per year (WHO, 2017a). These targets have been adopted in the DSS as the threshold criteria to quantify the risk and related fitness for use.

CHAPTER 5: PHYSICAL AND AESTHETIC EFFECTS

5.1 INTRODUCTION

For water quality constituents of a 'physical' nature and for the non-human health related effects of constituents, guideline values or thresholds are not absolute and are considered to be value judgments determined from a wide range of values that may be broadly classed as acceptable. Quantification of the risk has considered a number of factors including:

- Taste and odour thresholds;
- The concentration that would produce noticeable stains on laundry or corrosion and encrustation of pipes or fittings; and
- The concentration that would be just noticeable in a glass of water and lead to a perception that the water was not of good quality.

5.2 AESTHETIC/PHYSICAL EFFECTS AND ASSOCIATED HAZARDS

Review of local and international drinking water guidelines and international data sources for aesthetic thresholds and physical characteristics was undertaken to characterise the non-human health related (physical/aesthetic) hazard effects. The hazard effect information collated for this aesthetic and physical characterisation and the brief literature review of each constituent included in Appendix B. For the constituents, associated with the aesthetic and physical water quality effects on domestic use, *i.e.* household washing (washing of dishes, floors), laundry use, household appliances and equipment, and for aesthetic acceptability, the use of indices and threshold limits have been applied to quantifying each of the exposure end point levels (with respect to the associated effect such as scaling, corrosion, staining and discolouration) in the DSS to determine the related risk, based on an increasing concentration of the hazard. Table 5-1 lists the aesthetic or physical effect that maybe encountered with the associated water quality presenting the hazard.

Table 5-1: Aesthetic/physical Effects and associated hazards

Potential aesthetic/physical effect	Water quality constituent (Hazard)
• Impaired Taste:	Zinc, magnesium, calcium, sodium, copper, iron, sulphate, ammonia, organic substances
• Impaired Colour:	Iron, manganese, copper
• Odour:	Organic, inorganic substances
• Dental Enamel Discolouration:	Iron, sulphide, manganese, copper, nickel
• Soap lathering (laundry, household washing)	Hardness (calcium, magnesium), alkalinity, pH
• Staining/Discolouration (laundry, household fixtures)	Iron, manganese, copper, sulphur, suspended solids
• Impaired piping	Suspended solids, turbidity, iron
• Scaling and Corrosion of appliances/equipment	Hardness, alkalinity, pH

With respect to gardening as a domestic water use, the associated adverse effects that may be encountered by using water with less than target water quality include human health and non-human health (physical damage) associated consequences.

- Human health related consequences potentially encountered include microbial infection due to irrigation with contaminated water and then raw vegetable consumption (mild infection/toxic effect);
- Physical damage may include crop reduction/a poorer quality crop due to foliar damage or root zone effects associated with increased salinity because of irrigation with saline water.

This water uses although on a small scale, does however impact on the domestic user who relies on the crops for his/her livelihood and subsistence. Irrigation with impaired water quality would have negative consequences on a user.

The direct contact of irrigation water with a crop mostly affects crop quality, while indirect impacts mostly affect crop yield. Indirect impacts are a consequence of the accumulation and redistribution of irrigation water constituents within the root zone of soil that is irrigated (Du Plessis *et al.*, 2017). The risk-based approach adopted for irrigation water use guidelines (Du Plessis *et al.*, 2017) will also apply to gardening on a domestic scale. The risk-based irrigation guidelines identify five indicators to describe the effects irrigation water constituents have on crop yield and quality. Due to small scale use and the potential domestic use adverse effects indicated above, three of these would apply specifically to gardening; *viz*:

- Root zone effects;
- Foliar damage (leaf scorching when wetted); and
- Microbial contamination.

The identified key hazards (water quality constituents) that apply to the above indicators include (Du Plessis *et al.*, 2017):

- Root zone effects: Electrical Conductivity (EC of a saturated soil extract), Boron (B) and chloride (Cl) and sodium (Na) – the accumulation of salts, B, Cl and Na in the root zone of crops has an indirect effect on crop yield as when these constituents are absorbed by plant roots, they are transferred to the above ground parts where they affect several plant physiological processes which affect crop growth and yield.
- Leaf scorching when wetted: Na and Cl – some crops are sensitive to the absorption of excessive quantities of Cl and Na through their leaves when wetted by sprinkler irrigation, giving rise to leaf scorching, which affects crop quality and yield due to foliar damage.
- Microbial contamination: *E. coli* – poor microbial quality of irrigation water increases the microbial contamination of irrigated crops that could cause human health impacts should the crop be consumed raw or with minimal processing.

5.3 RISK ESTIMATION

5.3.1 Estimation of risks associated with physical effects

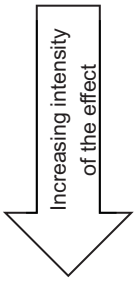
5.3.1.1 Household washing, Laundry use

The use of indices has been applied to quantifying risk of the physical effects of the chemical constituents based on exposure and threshold tolerance levels for each. An index range of 5 data points (0-4) with increasing concentrations has been selected to define the exposure curve. This is categorised in terms of the increasing intensity of the adverse effect, *i.e.* the severity of the consequence increases linearly with increasing concentration of the constituent of concern. For the different water uses the data points will differ

per constituent based on the exposure route and tolerance level related to the water use. The risk in the DSS is determined by assuming that for one range, the values increase linearly, relative to the risk probability which is also linear. The risk probability is then calculated from where the sample concentration sits on the range. For example, Table 5-2 shows how staining of laundry, manganese would be categorised as follows in terms of the exposure effect.

Table 5-2: Relationship between manganese concentration and associated effects of exposure

Range	Manganese concentration	Effect of the exposure
0	0.0-0.05 mg/L	No staining
1	0.05-.1 mg/L	Slight staining of white clothes
2	0.1-0.4 mg/L)	Moderate staining of clothes and fixtures
3	0.4-5 mg/L)	Severe staining of clothes and fixtures
4	5-10 mg/L)	Extreme staining of clothes and fixtures



5.3.1.2 Appliances and equipment

The Langelier saturation index (a formula) is an approximate indicator of the degree of saturation of calcium carbonate in water. It is calculated using the pH, alkalinity, calcium concentration, total dissolved solids and water temperature and is based on a study of carbonate equilibrium in water. The Langelier Saturation Index is used to determine how well water is balanced between corrosive and scale-forming.

- Langelier Index is negative, then the water is under saturated with calcium carbonate and will tend to be corrosive.
- Langelier Index is positive, then the water is over saturated with calcium carbonate and will tend to deposit calcium carbonate forming scales in appliances and equipment.
- If Langelier Index is close to zero, then the water is just saturated with calcium carbonate and will neither be strongly corrosive or scale forming.

The formula for the calculation of the Langelier saturation index (LSI) is:

$$LSI = pH + \frac{\log\left(K_a\, \gamma_{Ca^{2+}}[Ca^{2+}]\gamma_{HCO_3^-}[HCO_3^-]\right.}{\gamma_{H^+}\, K_{sp}}$$

The Langelier Index is defined as the difference between the actual pH (measured) and modelled pHs. (from the chemical analysis of water quality constituents). The pHs represents the theoretical equilibrium.

The Saturation Index (SI) = pH – pHs.

The magnitude and sign of the Langelier Index value shows water's tendency to dissolve scale, and thus to inhibit or encourage corrosion. If the pHs is lower than the actual pH (negative SI), the water is corrosive. Vice versa, a positive SI is indicative of a scale forming water. A Langelier Index in the range of -1 to +1 has a relatively low corrosion impact on metallic components of the appliances and equipment. Langelier Index values outside this range may result in laundry stains or leaks. The calculation methodology adopted in the DSS to quantify the risk of corrosion and scaling on appliance equipment makes use of the Langelier Index calculation. The calculation requires the water composition of TDS, water temperature, pH and Calcium Carbonate and alkalinity to be inputted by the user. The calculation adopted is as follows:

$$LI = pH_a - pH_s \text{ where}$$

pH_a is the measured pH of the water sample, and
pHs the calculated pH of a water of the given analysis when in chemical equilibrium with solid $CaCO_3$.

pHs is calculated as follows:

$$pH_s = (9.3 + A + B) – (C + D)$$

Where:
$A = (Log_{10}(TDS) – 1) / 10$
$B = -13.12 \times Log_{10}(°C + 273) + 34.55$
$C = Log_{10}(Ca \text{ as } CaCO_3) – 0.4$
$D = Log_{10}(\text{Alkalinity as } CaCO_3)$
(Concentrations are measured as mg/L).

The following threshold ranges of the LI, are used to define the acceptability of corrosion and scaling (tolerable risk) (Table 5-3:). The calculated LI is assessed against the threshold criteria to determine risk-level of corrosion and scaling.

Table 5-3: Langelier index thresholds for corrosion and scaling

	Langelier Index	
Range	**Corrosion**	**Scaling**
Ideal	> -0.5	<+0.5
Acceptable	-0.5 to -1.0	+0.5 to +1.0
Tolerable	-1.0 to -2.0	+1.0 to +2.0
Unacceptable	< -2.0	>+2.0

5.3.2 Estimation of risks associated with aesthetic acceptability

Water for domestic use should not only be safe but acceptable in colour, appearance and taste. The acceptability of drinking-water to users is subjective and can be influenced by many different constituents. The concentration at which constituents are objectionable to users is variable and dependent on individual and local factors (WHO, 2017). Guideline values have not been established in the WHO Guidelines for Drinking Water Quality for constituents influencing water quality that have no direct link to adverse health impacts. However, guideline values have been established for some substances that may cause taste or odour in drinking-water at much lower concentrations than the guideline value because there is such a wide range in the ability of users to detect them by taste or odour (WHO, 2017a). The calculation methodology in the DSS includes the use of indices (as above for physical effects) to quantify acceptable aesthetic aspects of odour and colour derived from literature-based exposure and threshold criteria levels determined for each. The risk in the DSS is determined by assuming that for one range, the values increase linearly, relative to the risk probability which is also linear. The risk probability is then calculated from where the sample concentration sits on the range.

5.3.2.1 Taste and odour

Taste and odour are two of the primary criteria domestic users use to judge the quality and acceptability of drinking water. People's sense of taste and smell tends to vary, and so the acceptability of the same water can vary from person to person and from day to day for the same person. Whilst taste and odour present in water does not generally have a health impact, the presence of tastes and odours may raise consumer concern with regard to water quality (NRMMC Australian Drinking WQGs, 2016). Taste and odour can originate from natural inorganic and organic chemical contaminants and biological sources or processes (e.g. aquatic microorganisms), from contamination by synthetic chemicals, from corrosion or as a result of problems with water treatment (e.g. chlorination). Taste and odour may also develop during storage and distribution as a result of microbial activity (WHO, 2017a). In the assessment of drinking water quality, the sense of taste is more useful in detecting inorganic constituents, while the sense of smell detects organic constituents more effectively (DWA, 1996).

Inorganic compounds are generally present in water in substantially higher concentrations than organic compounds. Taste thresholds for some commonly occurring inorganic ions are about 0.1 mg/L for manganese, 0.3 mg/L for iron, 3 mg/L for copper, 3 mg/L for zinc, 250 mg/L for chloride, and 250‑500 mg/L for sulphate. Most of these ions have health guidelines at concentrations higher than their taste thresholds (except copper at 2 mg/L). In most cases the domestic user would reject the water for aesthetic reasons before it would be of health concern (NRMMC Australian Drinking WQGs, 2016). Most common odours in water can be described as musty, earthy or woody. Odours can be linked primarily to cyanobacterial (*viz.* geosmin and 2 methyl isoborneol) and algal compounds. Disinfection compounds can also contribute to taste and odour in water. Odour levels in water can be described either in terms of a quantitative measure such as Threshold Odour Number (TON) or qualitatively as absence of "objectionable" or offensive odours. The TON is defined as the greatest dilution of sample with odour free water that yields a final water with an odour which is just detectable under carefully controlled test conditions (SAWQGs, 1996). TON is calculated as follows:

$$TON = (A + B)/ A$$

Where:
A – Volume of Sample with odour
B – Volume of Pure Water with no odour Added (to 200 ml)

Table 5-4 shows the odour threshold criteria (acceptable risk) adopted in the DSS. Example: If A was a 100 ml sample and 100 ml of water had to be added to not detect the odour – the TON would be 2 (Table 5-5). TON = (100 + 100)/ 100.

Table 5-4: Odour threshold criteria (acceptable risk) (adapted from SAWQGs, 1996):

Range	TON	Effect of the exposure
Ideal	1	Odourless
Acceptable	1-2	Noticeable odour
Tolerable	2-5	Strong odour which is likely to be objectionable to a large percentage of users
Unacceptable	5-10	Stronger odour, increasingly objectionable

Table 5-5: Calculation of TON based on sample dilution

Sample dilution to 200 mL (B)	Threshold Odour number (TON)
200	1
100	2
70	3
50	4
35	6
25	8
17	12
8.3	24
5.7	35
4	50
2.8	70
2	100
1.4	140
1	200

5.3.2.2 Colour

Colour, cloudiness, particulate matter and visible organisms may also be noticed by consumers and may create concerns about the quality and acceptability of a domestic water. Drinking-water should ideally have no visible colour. Colour in drinking-water is usually due to the presence of coloured organic matter (primarily humic and fulvic acids) associated with the humus fraction of soil. Colour is also strongly influenced by the presence of iron and other metals, either as natural impurities or as corrosion products or by waste discharges, for example from dyeing operations in the textile industry, and paper manufacture. Most users can detect colour above 15 true colour units (TCU) in a glass of water. A threshold level of 15 TCU is often acceptable to users, and is generally accepted as a guideline value. No health-based guideline value is currently proposed for colour in drinking-water internationally.

5.4 RISK REPORTING RELATED TO AESTHETIC QUALITY AND PHYSICAL EFFECTS

For the reporting of the physical effect and aesthetic risk probabilities, threshold limits are used as the criteria for the fitness fir use categorisation. The threshold limits are defined by the endpoints that apply for each level of the physical or aesthetic effect (e.g. hardness, turbidity, colour, odour) which is categorised in terms of the increasing intensity of the adverse effect, *i.e*. the severity of the consequence increases linearly with increasing concentration of the constituent of concern. A risk estimate (as a percentage) has been has been defined based on these threshold limit criteria and represents the risk probability of occurrence and the severity associated with the end point effect (e.g. scaling or staining) (Table 5-6).

Table 5-6: A risk estimate categories for aesthetic and physical effects

Risk estimate (Percentage)	Probability of Occurrence	Severity of the effect
<1	None	None
1-5	Rare	Negligible
>5-15	Possible	Minor
>15-100	Certain	Significant

For the physical effect risks associated with corrosion and scaling, the Langelier Index threshold limits are used as the criteria to define the four-level fitness for use risk categorisation. In terms of aesthetic water quality risk (colour and odour) the literature-based threshold limits have been applied. The threshold criteria adopted for the risk descriptors for physical effects and aesthetic quality are described in Tables 5-8 to 5-9.

Table 5-7: Threshold limit criteria defining the fitness for use categories for physical effect risk reporting

Physical Effect				
Fitness-for-Use Category	Threshold limit criteria			
	Hardness (mg/l)	Total Dissolved Salts (mg/l)	Turbidity (NTU)	Risk estimate (%)
Ideal	0-100	0-450	0-0.1	<1
Acceptable	100-150	450-1000	0.1-1.0	1-5
Tolerable	150-200	1000-2000	1-5	>5-15
Unacceptable	>200	>2000	>5	>15-100

Table 5-8: Threshold limit criteria defining the fitness for use categories for the Physical Effects: Scaling and Corrosion risk reporting

Corrosion or Scaling			
Fitness-for-Use Category	Threshold limit criteria		
	Corrosion (Langelier Index)	Scaling (Langelier Index)	Risk estimate (%)
Ideal	> -0.5	< 0.5	<1
Acceptable	-0.5 to -1.0	+0.5 to +1.0	1-5
Tolerable	-1.0 to -2.0	+1.0 to 2.0	>5-15
Unacceptable	< -2.0	> +2.0	>15-100

Table 5-9: Threshold limit criteria defining the fitness for use categories for Aesthetic Quality (Odour and Colour) risk reporting

Colour and Odour			
Fitness-for-Use Category	Threshold limit criteria		
	Colour (Total Colour units)	Odour (Threshold Odour numbers) (linked to taste)	Risk estimate (%)
Ideal	< 5	1	<1
Acceptable	5 to -10	1-2	1-5
Tolerable	10 to 15	2-5	>5-15
Unacceptable	> 15	5-10	>15-100

CHAPTER 6: DOMESTIC WATER USE FOR GARDENING

6.1 INTRODUCTION

Gardening as a use is considered as a component in both the domestic use and the irrigation use risk-based water quality guidelines. For the purposes of the domestic use water quality guidelines, the risk associated with the impact on the domestic user who relies on the crops for his/her livelihood and subsistence is included at a reference level in tier 1, which specifies water quality requirements. Based on availability of the calculation methodology and philosophy already devised as part of the risk-based Irrigation water quality guidelines, the DSS for domestic use has adopted the generic Tier 1 fitness for use criteria of the irrigation risk-based water quality guidelines, as its Tier 1 (conservative limits). For further analysis, *i.e*. fitness for use assessments and water quality requirements the user is directed to the irrigation risk-based water quality guidelines (Du Plessis *et al.*, 2017). On guidance by the study reference group no further development has been undertaken in the domestic use guidelines for gardening, as it was considered a duplication of the methodology already available. Irrigation water quality impacts associated with crop yield and quality is an identified component of specific relevance to domestic use. The water quality indicators for domestic gardening use include root zone effects, leaf scorching and microbial contamination. The criteria per water quality suitability indicator as specified in the irrigation risk-based water quality guidelines are used.

6.2 RISK CALCULATION METHOD

6.2.1 Overview

The acceptable risk is derived through a mathematical approach (calculation methodology) comprising the risk assessment that accounts for all the assumptions and exposure conditions. A total risk estimate is generated as the output in terms of the risk of adverse effects. The following sub-sections present calculation methodology applied that inform the informatics in the DSS. The calculation adopted in the DSS varies at three levels, *i.e.* in terms of the hazard categorisation/characterisation, the exposure route and the exposure scenario (domestic use and the receptor characteristics considering how the water is experienced). The hazard categorisation is as follows and is the primary determinant of the methodology used:

- Carcinogen (non-threshold – those that do not appear to have a threshold)
- Toxicant (effects are observed only above a certain threshold dose, with no effects observed at doses below this threshold even with lifetime exposure)
- Infectious agent (microbiological – disease burden quantification)
- Physical properties (aesthetic acceptability and physical damage); and
- Chemical properties (damage to subsistence garden crops)

The following have been used in the risk-based irrigation water use guidelines (Du Plessis *et al.*, 2017) in terms of assessing the impacts of the hazards of the gardening related effects:

6.2.2 Root zone effects

The tolerance of the crops to EC, B, Cl and Na in the root zone assessed in terms of the yield response, i.e. sensitivity based on either a maximum threshold concentration or on a range dependant concentration, is defined. The approach to deduce the yield response of the crops uses the concentration of salts (EC), B, Cl and Na concentration in the root zone which is then linked to the crop yield response data of the concentration of the individual constituents in the root zone, in order to estimate how the crop yield is affected. The criteria used in the DSS to determine the fitness for use category based on the relative yield are indicated in Table 6-1 (Du Plessis *et al.*, 2017).

Table 6-1: Irrigation water concentration related to corresponding crop yield

	Relative crop yield (%)	Irrigation water concentration that will give rise to the corresponding relative crop yield			
		Salinity (EC) mS/m	Boron (B) mg/L	Chloride (Cl) mg/l	Sodium (Na) (SAR)
Root Zone effects	90-100	<57	<0.40	<208	<2.99
	80-90	57-75	0.40-0.67	208-269	2.99-3.27
	70-80	75-92	0.67-0.93	269-331	3.27-3.54
	<70	>92	>0.93	>331	>3.54

6.2.3 Leaf scorching

Crops susceptible to foliar damage caused by salts absorbed directly through their leaves exhibit great yield reductions than when only exposed to rot zone effects (Du Plessis *et al.*, 2017). Limited quantitative data is however available to assess the susceptible of crops to foliar damage. In the DSS, the degree of leaf scorching is thus evaluated only in qualitative terms of leaves sprinkled with saline water (sodium and chloride concentration ranges associated with the indicated qualitative degree of leaf scorching). The criteria used in the DSS are indicated in Table 6-2 (Du Plessis *et al.*, 2017).

Table 6-2: Irrigation water concentration related to related degree of leaf scorching

	Degree of leaf scorching	Irrigation water concentration that may cause the corresponding degree of leaf scorching under sprinkler irrigation	
		Chloride (Cl) mg/l	Sodium (Na) (mg/l)
Leaf Scorching when wetted	None	<70	<50
	Slight	70-135	50-83
	Moderate	135-180	83-115
	Severe	> 180	>115

6.2.4 Microbial contamination

The main concern is the health risk posed by crops destined for human consumption that have been contaminated during irrigation (*i.e.* crops consumed raw or with minimal processing). Microbial risk for irrigation it is determined by a quantitative microbial risk assessment, using *E. coli* as an indicator of microbial pathogens, and is based on an annual intake which is calculated from the volume of irrigation water retained by the crop and how much is consumed on an annual basis. The risk of norovirus infection is then determined based on the *E. coli* count based on a dose response function, and the individual risk and annual risk of infection is then determined per person per year. The risk is expressed as the number of excess infections per 1000 persons per annum. The criteria used in the DSS to determine the fitness for use category based on the calculated number of excess infections per thousand persons per annum are indicated in Table 6-3. For this Tier 1 calculation the excess infections are reported assuming lettuce to be the most sensitive crop (retaining the largest volume of irrigated water consumed for crops assessed, *viz.* 11 ml (Du Plessis *et al.*, 2017).

Table 6-3: Fitness for use criteria as related to the number of excess infections per one thousand persons

	Excess infections per 1000 persons p.a.	Irrigation water concentration predicted to give rise to the indicated excess infections per 1000 persons p.a. (*E. coli* counts per 100 ml)
Microbial contamination	**<1**	**<351**
	1-3	**351-1052**
	3-10	**1052-3506**
	>10	**>3506**

6.3 WATER QUALITY RISK REPORTING RELATED TO GARDENING

The water quality indicators for domestic gardening use include root zone effects, leaf scorching and microbial contamination. The criteria per water quality suitability indicator as specified in the irrigation risk-based water quality guidelines are used as the four-level categorisation. The threshold limits applicable to the risk descriptions and associated fitness for use categorisation for root zone effects, leaf scorching and microbial contamination are indicated in Tables 6-4 to 6-6, respectively. The DSS for domestic use has adopted the generic fitness for use criteria of the irrigation risk-based water quality guidelines, as its water quality requirement (conservative) guidelines.

Table 6-4: Threshold criteria defining the fitness for use categories for crop yield (subsistence gardening) risk reporting

	Fitness-for-Use	Relative crop yield (%)	Irrigation water concentration that will give rise to the corresponding relative crop yield			
			Salinity (EC) mS/m	Boron (B) mg/L	Chloride (Cl) mg/l	Sodium (Na) (SAR)
Root Zone effects	Ideal	90-100	<57	<0.40	<208	<2.99
	Acceptable	80-90	57-75	0.40-0.67	208-269	2.99-3.27
	Tolerable	70-80	75-92	0.67-0.93	269-331	3.27-3.54
	Unacceptable	<70	>92	>0.93	>331	>3.54

Table 6-5: Threshold criteria defining the fitness for use categories for leaf scorching (subsistence gardening) risk reporting

	Fitness-for-Use	Degree of leaf scorching	Irrigation water concentration that may cause the corresponding degree of leaf scorching under sprinkler irrigation	
			Chloride (Cl) mg/l	Sodium (Na) (mg/l)
Leaf Scorching when wetted	Ideal	None	<70	<50
	Acceptable	Slight	70-135	50-83
	Tolerable	Moderate	135-180	83-115
	Unacceptable	Severe	> 180	>115

Table 6-6: Threshold criteria defining the fitness for use categories for microbial contamination (subsistence gardening) risk reporting

	Fitness-for-Use	Excess infections per 1000 persons p.a.	Irrigation water concentration that may cause the corresponding degree of leaf scorching under sprinkler irrigation
Microbial contamination	Ideal	<1	<351
	Acceptable	1-3	351-1052
	Tolerable	3-10	1052-3506
	Unacceptable	>10	>3506

CHAPTER 7: CONCLUSIONS & RECOMMENDATIONS

7.1 CONCLUSIONS

The project objective was to develop a risk-based methodology for determining Water Quality Guidelines for Domestic Use enabled through a user-friendly and practical Decision Support System (DSS). The key components comprised firstly, the development of the approach and methodology for the risk calculations based on supporting science to be included in the technology demonstrator; and secondly the development of the informatics for a demonstrator decision support system that addresses the main decision contexts for the use of the guidelines. The stated objectives have been achieved, with the following defining the key elements comprising the basis and outputs of the risk based domestic water quality guidelines development process:

- Definition of Extrinsic Risk component – The description of the exposure scenarios for the domestic user. This comprised the types of domestic water use typically encountered in the domestic environment catered for in the DSS. This was guided by the 1996 SAWQGs Volume 1, working committee and the reference group.

- A suitable common end-point for the stressors and target combinations were selected. An end-point that was a quantifiable, but not necessarily unique to a stressor was selected. The three primary end points that would apply to the domestic use exposure scenarios are human health, aesthetic quality and physical effects. These exposure scenarios direct the criteria and considerations into the selection of the type of methodologies that apply to determining the risk.

- Characterisation of the Intrinsic Risk component – Selection of stressors (hazard identification) and hazard characterisation and hazard categorisation (as related to exposure route) was undertaken. The characterisation of the hazard risk comprised largely of the review and interrogation of the available literature of risk, exposure and toxicological assessment data to determine the individual endpoints. The definition of the stressor-endpoint combinations that described the relevant target processes were adopted. This was a fundamental component to the risk-based guideline development process.

- For the purposes of the development of a technology demonstrator the range of water quality constituents (stressors/hazards) addressed were limited to 50 constituents, comprising the different types of hazards – toxicants, carcinogens, infectious agents, physical and aesthetic aspects.

- Quantification of the risk in the DSS – Definition of the calculation methodologies based on the scenario, exposure routes and receptor details followed. The state of knowledge was assessed included the uncertainties, variabilities and the quantification of relationships. A fundamental component to the development of the risk-based water quality guidelines was to determine applicable methodologies to quantify the risk within the decision context framework, which required a formulation of and hazard expression for each stressor.

- The calculation methodologies sourced from international literature and risk assessment best practices formed the definition of the quantification relationships and the basis of the informatics for the DSS. These were adapted and applied to the risk assessment quantification based on the exposure scenarios to best represent the expression of risk. A risk assessment protocol for each stressor-target combination was formulated. Key exposure and hazard variables were selected for the risk calculation. A key element to the risk quantification component was the input of the water quality composition which is the defining characteristic of the risk-based water quality guideline.

- Risk reporting – description of the system and criteria on how the risk is reported as the water quality guidance output. It is the quantitative and/or qualitative output that guides the user to a sensible decision. This comprises the presentation of risk-based water quality guideline in the DSS, and what the user is presented with. A reporting system which is aligned with the standard practice within Department of Water and Sanitation (DWS) and to the World Health Organisation (WHO) health-based target guidelines that is based on the health outcome has been defined. The categorisation system is colour coded for ease of reference to the risk level quantified, with a description of adverse effect (endpoint) if applicable.

- Technology demonstrator DSS – an engineered computational software system presented as a demonstrator/ prototype that provides a structured approach necessary for addressing the main decision contexts for the use of the guidelines. It incorporates the key features of Tier 1 and 2, based on exposure assessment data, risk assessment methodologies and mathematical calculations to provide risk-based guidance on water quality used for domestic purposes, using MS Excel as the user platform. The DSS incorporates the elements described above to produce risk-based outputs supporting decisions in specific contexts.

The domestic risk-based water quality guidelines represent a paradigm shift in the decision-making context to water quality management and in how water quality guidelines are used and applied. The development methodology of the decision support tool presents a fundamental change from the use of simple numeric values to providing both regulators and water users with a quantifiable assessment of the risk. In doing so the user would need to make a judgement call based on the available information, context and influencing factors. This risk approach generalises the basis for decision-making by incorporating as much of the relevant evidence as possible.

7.2 RECOMMENDATIONS

The project aim was successfully achieved, with the DSS as a product fulfilling the requirements of the technology demonstrator for risk based domestic use water quality guidelines. However, the following is required and recommended to develop the product further to a fully functional system to be utilised within the water resource management sector in South Africa:

- The further development of the domestic user DSS methodology in the next phases would need to address:
  - The functionality of the water quality objective setting at Tier 2;
  - Expansion of the water quality constituent database to include all constituents relevant to domestic use, specifically in the South African context;
  - The consideration of synergistic and antagonistic effects of constituents and expansion of the calculation methodology to address this;
  - The update of the methodology to include the assessment of multiple constituents simultaneously;
  - Endpoint confirmation of all hazards;
  - The incorporation of local domestic water use pattern information where applicable to improve site specificity, calculation methodology and receptor information;
  - Processes and procedures for the updating of the methodologies and exposure assessment data, based on the best available science information as it becomes available;
  - Functionality that allows export of water quality monitoring data from national and local monitoring programmes directly into the DSS;
  - A structured procedure applicable to Tier 3 users should be developed to control and maintain the original product while providing the user with a clear method of detailed analysis; and

- Currently the DSS tool has been demonstrated using MS Excel, however in going forward to full scale application, it is recommended that available on-line databases be tested to select a software suitable for the DSS for the guideline series.

- Wider stakeholder buy-in and guidance is required to gain acceptance of the risk-based approach to the assessment of water quality. Users may be hesitant to want to take decisions on the basis of a risk quantification that the DSS provides, without requisite understanding of the support it is meant to provide. More engagement is required to get users to accept the philosophy and approach.

- Further testing with the wider stakeholder user groups is required to refine the product and to update the DSS to improve user-friendliness and utility, based on feedback from users.

- A DSS tool that is available through an on-line platform is recommended.

- Next phases of the project require the integration with the user guidelines that needs to consider the selection of coding platform, intellectual property issues, controlled access to software system, version controls as well as processes and procedures on the updating of the methodologies and functionality of the DSS for the water user groups.

- Such a system places stringent demands on the custodianship of the product. An owner and champion within the DWS is required to spearhead the next phases of the DSS, its integration, its promotion and maintenance.

The following key challenges were experienced during life of project:

- The innovative and progressive nature of the project brief involved breaking new ground which resulted in much discussion and time in the definition of the envisaged product. Deliberation and discussion over much of the project was needed to adjust and confirm the scope of work and to manage the expectations of the reference group and users. This took longer than anticipated and resulted in adjustments over the course of the project from the original project scope, which proved to challenging from a time and budget point of view.
- The lack of understanding and total buy-in of the reference group on a quantifiable risk-based approach concept to provide water quality guidance proved to be challenging. The idea that the user of the DSS is required to make a judgement call on the fitness for use of the water has proven to be a challenge, that has highlighted the fact that more engagement is required with the water resources sector and then on to the general public. The understanding that the DSS is not providing a ‘line in the sand’ in terms of a static guideline value needs to be sufficiently and adequately communicated. For the DSS to be fully utilised to its potential and achieve the purpose for which it has been developed, a fundamental mind set change is required among users. It is no longer a situation of a simple ‘pass-fail’ number, the DSS provides common philosophical basis for decision-making in different contexts.
- The technical assessments and deliberations proved to be complex and thus time-consuming, which presented a challenge from the project delivery point of view. The two-year period time was not adequate do to justice to all the aspects that continually emerged on the approach and product development.
- The process required intensive literature review and assessments which proved to be data intensive. While international scientific databases and algorithms were adopted and adapted for the DSS development as these were easily accessible and tested, limited time and budget prevented investigations to make adjustment for local circumstances.
- The availability of toxicological data and exposure assessment studies and time constraints, limited the range of the constituents included at this phase of the project.

- Lack of risk-based assessment data of physical and aesthetic constituents, limited the extent of the risk quantification as compared to the human health related constituents. Internationally most countries focus on drinking water and not in the domestic use context as South Africa does.
- The lack of documented available domestic water uses pattern data for the South African context prevented the adjustment of the risk calculation methodologies to reflect local circumstances. While the risk calculations are scientifically sound, they are based much on the USEPA and WHO water use data.
- The inclusion of the functionality to determine a water quality objective (for the source water) based on an accepted risk level and risk management scenario, (reverse functionality of the fitness for use) was not feasible during this process due to project time and budget constraints, however is recommended for the next phase.

In conclusion it can be said that the development of risk-based approach and a technology demonstrator DSS for domestic water quality guidelines was a challenging undertaking requiring a shift in thinking and approach and innovation in conceptualisation and development. It however proved to be exciting and forward thinking, with the resultant DSS product presenting a novel and revolutionary manner of how domestic water quality may be expressed in supporting the multifaceted dimensions and complexities to water quality management in South Africa.

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APPENDIX A: LIST OF WATER QUALITY CONSTITUENTS

Constituents	Guideline type	DWS 1996 Water Quality Guidelines	SANS 241 – 2015	WHO Drinking Water Guidelines (2017)	In Decision support system demonstrator	Comment
Acrylamide	health			✓		No toxicity study data available
Algae (Chlorophyll a)	health	✓				Microcystin as representative constituent
Algae (Blue Green)	health	✓				Microcystin as representative constituent
Aluminium	operational	✓	✓		✓	
Ammonia	aesthetic	✓	✓		✓	
Antimony as Sb	health		✓	✓	✓	
Arsenic	health	✓	✓	✓	✓	
Asbestos	health	✓				Inhalation is the primary risk. Not considered a serious health threat from water
Atrazine	health	✓			✓	
Barium as Ba	health		✓	✓	✓	
Benzene	health			✓	✓	
Benzo(a)pyrene	health			✓	✓	
Boron as B	health		✓	✓	✓	
Bromide	health				✓	
Cadmium	health	✓		✓	✓	
Calcium	aesthetic	✓			✓	
Carbon tetrachloride	health			✓	✓	
Chloride	aesthetic	✓	✓		✓	

Constituents	Guideline type	DWS 1996 Water Quality Guidelines	SANS 241 – 2015	WHO Drinking Water Guidelines (2017)	In Decision support system demonstrator	Comment
Chlorine	health		✓		✓	
Chloroform	health				✓	
Chromium (VI)	health	✓		✓	✓	
Coliphages	health	✓				*E. coli* is used as the indicator
Colour	aesthetic	✓	✓	✓	✓	
Copper	health	✓	✓	✓	✓	
Cyanide (as CN^-)	health				✓	
Dissolved Organic Carbon	aesthetic	✓				no toxicity study data available
DDT	health			✓	✓	
Electrical Conductivity	aesthetic		✓		✓	
Enteric Viruses	health	✓				*E. coli* is used as the indicator
Escherichia coli	health		✓	✓	✓	
Ethylbenzene	health			✓	✓	
Faecal Coliforms	health	✓	✓			*E. coli* is used as the indicator
Fluoride	health	✓	✓	✓	✓	
Gross α and β particles	health	✓				no toxicity study data available
Glyphosate and AMPA	health				✓	Recommended for inclusion
Heterotrophic plate count	operational	✓	✓			Recommended for removal
Iron	aesthetic	✓	✓		✓	
Iron	health	✓	✓		✓	
Lead	health	✓	✓	✓		No adequate toxicity study data available
Magnesium	aesthetic	✓			✓	
Manganese	aesthetic	✓	✓		✓	

Constituents	Guideline type	DWS 1996 Water Quality Guidelines	SANS 241 – 2015	WHO Drinking Water Guidelines (2017)	In Decision support system demonstrator	Comment
Manganese	health		✓		✓	
Monochloramine	health		✓	✓	✓	
Mercury	health	✓	✓	✓	✓	
Nickel	health		✓	✓	✓	
Nitrate as N	health	✓	✓	✓	✓	
Nitrite as N	health		✓	✓		No adequate toxicity study data available
Odour	aesthetic	✓	✓		✓	
pH	aesthetic	✓	✓		✓	
Phenols	aesthetic	✓	✓		✓	Not of health concern in terms of WHO guidelines.
Potassium	health	✓				Not of health concern in terms of WHO guidelines
Protozoan Parasites	health	✓	✓		✓	*E. coli* is used as the indicator
Radioactivity	health	✓				Recommended for removal
Selenium	health	✓	✓	✓	✓	
Settleable matter	aesthetic	✓				
Sodium	aesthetic	✓	✓		✓	
Somatic coliphages	operational	✓	✓			*E. coli* is used as the indicator
Sulphate	aesthetic	✓	✓		✓	
Sulphate	health	✓				No toxicity study data available
Taste	aesthetic		✓		✓	
Toluene	health			✓	✓	
Total Coliforms	operational	✓	✓			*E. coli* is used as the indicator
Total Dissolved salts	aesthetic	✓	✓			Recommended for removal
Total Hardness	aesthetic	✓			✓	
Microcystin-LR	health			✓	✓	

Constituents	Guideline type	DWS 1996 Water Quality Guidelines	SANS 241 – 2015	WHO Drinking Water Guidelines (2017)	In Decision support system demonstrator	Comment
Trihalomethanes	health	✓	✓	✓		Chloroform is included as a representative of Trihalomethanes
Turbidity	aesthetic	✓	✓		✓	
Uranium (238)	health		✓	✓	✓	
Vanadium	health	✓				no toxicity study data available
Xylene	health			✓	✓	
Zinc	aesthetic	✓	✓		✓	

APPENDIX B: HAZARD CHARACTERISATION AND EXPOSURE ASSESSMENT DATA

1. ACRYLAMIDE

Acrylamide was initially produced for commercial purposes by reaction of acrylonitrile with hydrated sulfuric acid and separation of the product from its sulphate salt. Direct uses of acrylamide include photopolymerization systems, adhesives and grouts, and polymer cross-linking. The primary use of acrylamide is in the production of polyacrylamides, which are used for enhanced oil recovery in water flooding, in oil well drilling fluids, in fracturing aids, in sewage treatment flocculants, in soil conditioning and stabilization, in papermaking aids and thickeners, in adhesion-promoting polymers, in dye acceptors, in textile additives, and in paint softeners [https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0286tr.pdf]. Release of acrylamide to the environment may occur during its production and use or in the production of polyacrylamide.

Products and compounds containing polyacrylamide may serve as sources of exposure to residues of acrylamide. Examples include polyacrylamide compounds used in oil well drilling operations (well drilling muds), as flocculants in water treatment, coagulants in food processing, sealing grouts and some coatings, and as foam builders, lubricants, and emollients in some personal care and grooming products. Localized contamination may arise from the use of acrylamide in grouting operations [https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0286tr.pdf].

Residual acrylamide monomer occurs in polyacrylamide coagulants used in the treatment of drinking-water. At a monomer content of 0.05%, this corresponds to a maximum theoretical concentration of 0.5 µg/l of the monomer in water (WHO, 2017a). Drinking water authorities need to certify that, for polyacrylamides used as coagulants or flocculants in drinking water treatment, the level of acrylamide monomer in the polymer does not exceed 0.05% and the application rate for the polymer does not exceed 1 mg/L. Human exposure is much greater from food than from drinking-water, owing to the formation of acrylamide in foods (e.g. breads, fried and roasted foods) cooked at high temperatures (WHO, 2017a). Following ingestion, acrylamide is readily absorbed from the gastrointestinal tract and widely distributed in body fluids.

Acrylamide can cross the placenta. It is neurotoxic, affects germ cells and impairs reproductive function [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1]. Neurological impairment is a well-established human health hazard associated with acute and repeated occupational exposure involving inhalation of airborne acrylamide and dermal contact with acrylamide-containing materials [https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0286tr.pdf].

Acrylamide (Ingestion)
RfD UF: 30
RfD Rats: 0.002 mg/kg/d (Johnson *et al*., 1986)
NOAEL Rats: 0.5 mg/kg-day (Friedman *et al*., 1995)
LOAEL Rats: 2 mg/kg-day (Friedman *et al*., 1995)
BMDL Rats: 0.053 mg/kg-day (Johnson *et al*., 1986)
Oral Slope Factor: 5×10^{-1} per mg/kg-day

Acrylamide (Inhalation)
RfC UF: 30
RfC Rats: 0.006 mg/m^3 (Johnson *et al*., 1986)
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: 0.18 mg/m^3 (Johnson *et al*., 1986)
Slope Factor: No data

2. ALUMINIUM

Aluminium is the most abundant metallic element and constitutes about 8% of the Earth's crust. It occurs naturally in the environment as silicates, oxides, and hydroxides, combined with other elements, such as sodium and fluoride, and as complexes with organic matter (World Health Organization, 2003). [http://apps.who.int/iris/bitstream/10665/75362/1/WHO_SDE_WSH_03.04_53_eng.pdf]. Aluminium metal is used as a structural material in the construction, automotive, and aircraft industries, in the production of metal alloys, in the electric industry, in cooking utensils, and in food packaging. Aluminium compounds are used as antacids, antiperspirants, and food additives (ATSDR, 1992). Aluminium salts are also widely used in water treatment as coagulants to reduce organic matter, colour, turbidity, and microorganism levels. The process usually consists of addition of an aluminium salt (often sulphate) at optimum pH and dosage, followed by flocculation, sedimentation, and filtration (Health Canada, 1993).

Aluminium levels in drinking-water vary according to the levels found in the source water and whether aluminium coagulants are used during water treatment. At an average adult intake of aluminium from food of 5 mg/day and a drinking-water aluminium concentration of 0.1 mg/litre, the contribution of drinking-water to the total oral exposure to aluminium will be about 4%. The contribution of air to the total exposure is generally negligible. In humans, aluminium and its compounds appear to be poorly absorbed, although the rate and extent of absorption have not been adequately studied (World Health Organization, 1998). [http://apps.who.int/iris/bitstream/10665/75362/1/WHO_SDE_WSH_03.04_53_eng.pdf]. There is little indication that aluminium is acutely toxic by oral exposure despite its widespread occurrence in foods, drinking-water, and many antacid preparations (WHO, 1997). There is no indication that aluminium is carcinogenic (WHO, 1998). [http://apps.who.int/iris/bitstream/10665/75362/1/WHO_SDE_WSH_03.04_53_eng.pdf].

Workers who breathe large amounts of aluminium dusts can have lung problems, such as coughing or changes that show up in chest X-rays. The use of breathing masks and controls on the levels of dust in factories have largely eliminated this problem. Some workers who breathe aluminium-containing dusts or aluminium fumes have decreased performance in some tests that measure functions of the nervous system (ATSDR, 2008). [https://www.atsdr.cdc.gov/ToxProfiles/tp22-c1-b.pdf]. Oral exposure to aluminium is usually not harmful. Some studies show that people exposed to high levels of aluminium may develop Alzheimer's disease, but other studies have not found this to be true. We do not know for certain that aluminium causes Alzheimer's disease. Some people who have kidney disease store a lot of aluminium in their bodies. The kidney disease causes less aluminium to be removed from the body in the urine. Sometimes, these people developed bone or brain diseases that doctors think were caused by the excess aluminium. Although aluminium-containing over the counter oral products are considered safe in healthy individuals at recommended doses, some adverse effects have been observed following long-term use in some individuals (ATSDR, 2008). [https://www.atsdr.cdc.gov/ToxProfiles/tp22-c1-b.pdf]. Brain and bone disease caused by high levels of aluminium in the body have been seen in children with kidney disease. Bone disease has also been seen in children taking some medicines containing aluminium. In these children, the bone damage is caused by aluminium in the stomach preventing the absorption of phosphate, a chemical compound required for healthy bones (ATSDR, 2008). [https://www.atsdr.cdc.gov/ToxProfiles/tp22-c1-b.pdf]

Aluminium (Ingestion)
RfD UF: 100
Rfd Rats: No data
NOAEL Rats: 52 mg/kg/day (Gomez et al., 1986) (Short-term exposure)
[http://apps.who.int/iris/bitstream/10665/75362/1/WHO_SDE_WSH_03.04_53_eng.pdf]

LOAEL Rats: No Data

Aluminium (Inhalation)
RfC UF: No data
RfC Rats: No Data
NOAEL Rats: No Data
LOAEL Rats: No Data

3. AMMONIA

Ammonia is a chemical that is made both by humans and by nature. Ammonia is a colourless gas with a very sharp odour. Ammonia in this form is also known as ammonia gas or anhydrous ("without water") ammonia. Ammonia gas can also be compressed and becomes a liquid under pressure. The odour of ammonia is familiar to most people because ammonia is used in smelling salts, household cleaners, and window cleaning products (ATSDR, 2004). Ammonia easily dissolves in water. In this form, it is also known as liquid ammonia, aqueous ammonia, or ammonia solution. In water, most of the ammonia changes to the ionic form of ammonia, known as ammonium ions, which are represented by the formula $NH4^+$. Ammonia can also be combined with other substances to form ammonium compounds, including salts such as ammonium chloride, ammonium sulphate, ammonium nitrate, and others (ATSDR, 2004). Most of the ammonia in the environment comes from the natural breakdown of manure and dead plants and animals. Since ammonia occurs naturally in the environment, we are regularly exposed to low levels of ammonia in air, soil, and water (ATSDR, 2004). Ammonia is found naturally in the environment. You may be exposed to ammonia by breathing air, eating food, or drinking water that contains it, or through skin contact with ammonia or ammonium compounds. Exposure to ammonia in the environment is most likely to occur by breathing in ammonia that has been released into the air (ATSDR, 2004). Ammonia is a corrosive substance and the main toxic effects are restricted to the sites of direct contact with ammonia (i.e. skin, eyes, respiratory tract, mouth, and digestive tract). If you walked into a dense cloud of ammonia or if your skin comes in contact with concentrated ammonia, your skin, eyes, throat, or lungs may be severely burned. These burns might be serious enough to cause permanent blindness, lung disease, or death. Likewise, if you accidentally ate or drank concentrated ammonia, you might experience burns in your mouth, throat, and stomach. There is no evidence that ammonia causes cancer. Ammonia has not been classified for carcinogenic effects by EPA, the Department of Health and Human Services (DHHS), or the International Agency for Research on Cancer (IARC) (ATSDR, 2004).

Some restrictions have been placed on levels of ammonium salts allowable in processed foods. FDA states that the levels of ammonia and ammonium compounds normally found in food do not pose a health risk. Maximum allowable levels in processed foods are as follows: 0.04-3.2% ammonium bicarbonate in baked goods, grain, snack foods, and reconstituted vegetables; 2.0% ammonium carbonate in baked goods, gelatins, and puddings; 0.001% ammonium chloride in baked goods and 0.8% in condiments and relishes; 0.6-0.8% ammonium hydroxide in baked goods, cheeses, gelatins, and puddings; 0.01% monobasic ammonium phosphate in baked goods; and 1.1% dibasic ammonium phosphate in baked goods, 0.003% in non-alcoholic beverages, and 0.012% in condiments and relishes (ATSDR, 2004). OSHA has set an 8-hour exposure limit of 25 ppm and a short-term (15-minute) exposure limit of 35 ppm for ammonia in the workplace. NIOSH recommends that the level in workroom air be limited to 50 ppm for 5 minutes of exposure (ATSDR, 2004).

Ammonia (Ingestion)
RfD UF: No data
RfD Humans: An RfD was not derived for ammonia (IRIS, 2012)
[https://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=506581]
NOAEL Humans: No Data
LOAEL Humans: No Data
BMDL Humans: No Data
Oral Slope Factor: N/A

Ammonia (Inhalation)
RfC UF:10
RfC Rats: No Data
RfC Human: 0.5 mg/kg/d (Holness et al., 1989), (Rahman et al., 2007), (Ballal et al., 1998), (Ali et al. 2001)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0422_summary.pdf#nameddest=rfd]
0.07 mg/m^3 (ATSDR, 2004)
[http://www.michigan.gov/documents/deq/deq-rrd-chem-AmmoniaDatasheet_527725_7.pdf]
NOAEL Human: 4.9 mg/m^3 (Holness et al., 1989), (Rahman et al., 2007), (Ballal et al., 1998), (Ali et al. 2001)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0422_summary.pdf#nameddest=rfd]
2.3 mg/m^3 (ATSDR, 2004) [http://www.michigan.gov/documents/deq/deq-rrd-chem-AmmoniaDatasheet_527725_7.pdf]

4. ANTIMONY AS SB

Elemental antimony forms very hard alloys with copper, lead and tin. Daily oral uptake of antimony appears to be significantly higher than exposure by inhalation, although total exposure from environmental sources, food and drinking-water is very low compared with occupational exposure. The form of antimony in drinking-water is a key determinant of the toxicity, and it would appear that antimony leached from antimony-containing materials would be in the form of the antimony(V) oxo-anion, which is the less toxic form. The subchronic toxicity of antimony trioxide is lower than that of potassium antimony tartrate, which is the most soluble form [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1]. Concentrations of antimony in air are considered to be lower today because industrial emissions have been significantly reduced by the introduction of dust filters. At present, abrasion of antimony (and other metals) from brakes, tyres and street surfaces as well as emission of aerosolic antimony in vehicle exhaust are the main sources of antimony in urban fine dust.
[http://www.who.int/water_sanitation_health/water-quality/guidelines/chemicals/antimony.pdf?ua=1].

The toxicity of antimony is a function of the water solubility and the oxidation state of the antimony species under consideration. Soluble antimony salts, after oral uptake, exert a strong irritating effect on the gastrointestinal mucosa and trigger sustained vomiting. Other effects include abdominal cramps, diarrhoea and cardiac toxicity.

[http://www.who.int/water_sanitation_health/water-quality/guidelines/chemicals/antimony.pdf?ua=1].
Multimedia antimony exposures are essentially negligible by comparison to occupational exposures at which discrete clinical health effects have been observed. Myocardial effects, chronic respiratory uptake of antimony-containing dusts leads to irritation of the respiratory tract and myocardial and liver damage are among the best-characterized human health effects associated with antimony exposure [https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=6].

Antimony (Ingestion)
RfD UF: 1000
RfD Rats: 4E-04 mg/kg/d (Schroeder *et al*., 1970)
NOAEL Rats: No data
LOAEL Rats: 0.35 mg/kg-day (Schroeder *et al*., 1970)
BMDL Rats: No data
Oral Slope Factor: No data

Antimony (Inhalation)
RfC UF: No data
RfC Rats: 0.006 mg/m^3 (Johnson *et al*., 1986)
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data
Slope Factor: No data

5. ARSENIC

Arsenic is naturally present at high levels in the groundwater of a number of countries. Arsenic is highly toxic in its inorganic form. Release of arsenic in the environment is a result of both manmade and natural activity. Arsenic enters the environment naturally through: ground water, mineral ore, and geothermal processes. Arsenic is released into the air by volcanoes, through weathering of arsenic-containing minerals and ores, and by commercial or industrial processes. Arsenic occurs naturally in the earth's crust, and much of its dispersion in the environment stems from mining and commercial uses. In industry, arsenic is a by-product of the smelting process (separation of metal from rock) (ATSDR, 2013). [https://www.atsdr.cdc.gov/csem/arsenic/docs/arsenic.pdf] Arsenic may be found in seafood (especially bivalves [clams, oysters, scallops, mussels], crustaceans [crabs, lobsters], and certain cold water and bottom feeding finfish, and seaweed/kelp, but it exists in the organic forms, which have not been shown to produce adverse effects in humans consuming these seafoods. This type of organic arsenic is also rapidly excreted (ASDR, 2013). [https://www.atsdr.cdc.gov/csem/arsenic/docs/arsenic.pdf].

Contaminated water used for drinking, food preparation and irrigation of food crops poses the greatest threat to public health from arsenic (WHO, 2016). [http://www.who.int/mediacentre/factsheets/fs372/en/]. The immediate symptoms of acute arsenic poisoning include vomiting, abdominal pain and diarrhoea. These are

followed by numbness and tingling of the extremities, muscle cramping and death, in extreme cases. The first symptoms of long-term exposure to high levels of inorganic arsenic (e.g. through drinking-water and food) are usually observed in the skin, and include pigmentation changes, skin lesions and hard patches on the palms and soles of the feet (hyperkeratosis). These occur after a minimum exposure of approximately five years and may be a precursor to skin cancer. In addition to skin cancer, long-term exposure to arsenic may also cause cancers of the bladder and lungs.

The International Agency for Research on Cancer (IARC) has classified arsenic and arsenic compounds as carcinogenic to humans, and has also stated that arsenic in drinking-water is carcinogenic to humans. Other adverse health effects that may be associated with long-term ingestion of inorganic arsenic include developmental effects, neurotoxicity, diabetes, pulmonary disease and cardiovascular disease. Arsenic-induced myocardial infarction, in particular, can be a significant cause of excess mortality. Arsenic is also associated with adverse pregnancy outcomes and infant mortality, with impacts on child health, and there is some evidence of negative impacts on cognitive development (WHO, 2016). [http://www.who.int/mediacentre/factsheets/fs372/en/]. There is no ambient air standard (i.e. no general air pollution limit) for arsenic [EPA 2007]. EPA has set 10 ppb as the allowable level for arsenic in drinking water (maximum contaminant level) [EPA 2006]. The World Health Organization recommends a provisional drinking water guideline of 10 ppb.

Arsenic (Ingestion)
RfD UF: 3
RfD Human: 3E-4 mg/kg-day (Tseng, 1977; Tseng et al., 1968)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0278_summary.pdf]
NOAEL Human: 0.0008 mg/kg-day (Tseng, 1977; Tseng et al., 1968)
LOAEL Human: 0.014 mg/kg-day (Tseng, 1977; Tseng et al., 1968)
BMDL Rats: No data
Oral Slope Factor: 1.5+0 mg/kg/day (IRIS, 1995)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0278_summary.pdf]

Arsenic (Inhalation)
RfC UF: No data
RfC Human: No Data
NOAEL Human: No Data
LOAEL Human: No Data
BMCL Human: No Data
Slope Factor: 4.3E-3 ug/cu.m (IRIS, 1995)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0278_summary.pdf]

6. ASBESTOS

Asbestos is a group of naturally occurring fibrous minerals with current or historical commercial usefulness due to their extraordinary tensile strength, poor heat conduction, and relative resistance to chemical attack. For these reasons, asbestos is used for insulation in buildings and as an ingredient in a number of products, such as roofing shingles, water supply lines, and fire blankets, as well as clutches and brake linings, gaskets, and pads for automobiles. The main forms of asbestos are chrysotile (white asbestos) and crocidolite (blue asbestos). Other forms include amosite, anthophyllite, tremolite and actinolite [http://www.who.int/news-room/fact-sheets/detail/asbestos-elimination-of-asbestos-related-diseases]. Asbestos is introduced into water by the dissolution of asbestos-containing minerals and ores as well as from industrial effluents, atmospheric pollution and asbestos cement pipes in the distribution system. Exfoliation of asbestos fibres from asbestos cement pipes is related to the aggressiveness of the water supply.

Asbestos is a known human carcinogen by the inhalation route. Although well studied, there has been little convincing evidence of the carcinogenicity of ingested asbestos in epidemiological studies of populations with drinking-water supplies containing high concentrations of asbestos [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1]. Limited data indicate that exposure to airborne asbestos released from tap water during showers or humidification is negligible (WHO, 2017a). A large number of studies of occupationally-exposed workers have conclusively demonstrated the relationship between asbestos exposure and lung cancer or mesothelioma. There is some evidence which suggests that the different types of asbestos fibres vary in carcinogenic potency relative to one another and site specificity. It appears, for example, that the risk of mesothelioma is greater with exposure to crocidolite than with amosite or chrysotile exposure alone [https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=6].

Asbestos (Ingestion)
RfD UF: No data
RfD Rats: No data
NOAEL Rats: No data
LOAEL Rats: No data
BMDL Rats: No data
Oral Slope Factor: No data

Asbestos (Inhalation)
RfC UF: No data
RfC Rats: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data
Slope Factor: No data

7. ATRAZINE

Atrazine is the common name for an herbicide that is widely used to kill weeds. It is used mostly on farms. Pure atrazine-an odourless, white powder-is not very volatile, reactive, or flammable. It will dissolve in water. Atrazine is made in the laboratory and does not occur naturally (ATSDR, 2003). [https://www.atsdr.cdc.gov/toxprofiles/tp153-c1-b.pdf]. Certified herbicide workers may spread atrazine on crops or croplands as a powder, liquid, or in a granular form. Atrazine is usually used in the spring and summer months. For it to be active, atrazine needs to dissolve in water and enter the plants through their roots. It then acts in the shoots and leaves of the weed to stop photosynthesis. Atrazine is taken up by all plants, but in plants not affected by atrazine, it is broken down before it can have an effect on photosynthesis. The application of atrazine to crops as an herbicide accounts for almost all of the atrazine that enters the environment, but some may be released from manufacture, formulation, transport, and disposal (ATSDR, 2003). [https://www.atsdr.cdc.gov/toxprofiles/tp153-c1-b.pdf]

One of the primary ways that atrazine can affect your health is by altering the way that the reproductive system works. Studies of couples living on farms that use atrazine for weed control found an increase in the risk of pre-term delivery. These studies are difficult to interpret because most of the farmers were men who may have been exposed to several types of pesticides. Atrazine has been shown to cause changes in blood hormone levels in animals that affected the ability to reproduce. Some of the specific effects observed in animals are not likely to occur in occur in humans because of biological differences between humans and these types of animals. However, atrazine may affect the reproductive system in humans by a different mechanism. Atrazine also caused liver, kidney, and heart damage in animals; it is possible that atrazine could cause these effects in humans, although this has not been examined (ATSDR, 2003). [https://www.atsdr.cdc.gov/toxprofiles/tp153-c1-b.pdf]

Not enough information is available to definitely state whether atrazine causes cancer in humans. Studies of human populations indicate that there may be a link between atrazine use and some types of cancer, but the information was not specific enough to make a definitive connection between atrazine and cancer (ATSDR, 2003). [https://www.atsdr.cdc.gov/toxprofiles/tp153-c1-b.pdf]. The EPA has set a maximum amount of atrazine allowable in drinking water of 3 µg/L. In addition, atrazine is designated as a Restricted Use Pesticide, which means that only certified pesticide applicators can use atrazine (ATSDR, 2003). [https://www.atsdr.cdc.gov/toxprofiles/tp153-c1-b.pdf].

Atrazine (Ingestion)
RfD UF: 100
RfD Rats: 3.5E-2 mg/kg/d (Ciba-Geigy Corp., 1986)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0209_summary.pdf]
NOAEL Rats: 3.5 mg/kg/d (Ciba-Geigy Corp., 1986)
LOAEL Rats: 25 mg/kg/d (Ciba-Geigy Corp., 1986)
BMDL Rats: No data available (IRIS, 1993)
Oral Slope Factor: No data

Atrazine (Inhalation)
RfC UF: No data
RfC Human: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data
Slope Factor: No data

8. BARIUM

Barium is present as a trace element in both igneous and sedimentary rocks, and barium compounds are used in a variety of industrial applications; however, barium in water comes primarily from natural sources. Food is the primary source of intake for the non-occupationally exposed population [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1]. However, where barium concentrations in water are high, drinking-water may contribute significantly to total intake (WHO, 2017a).

Acute hypertension has been observed in humans following accidental or intentional ingestion of soluble barium salts. The human and animal inhalation and intra-tracheal studies suggest that the respiratory system is a target of barium toxicity. Systemic effects, such as hypertension, may occur following inhalation exposure. Exposure to insoluble forms of barium such as barium sulphate and barite ore results in baritosis. The available human data on baritosis suggest that the accumulation of barium in the lungs does not result in medical disability or symptomatology. A decline in the profusion and opacity density, suggesting a decrease in the amount of accumulated barium in the lung, has been observed several years after termination of barium exposure. There is no evidence that barium is carcinogenic or genotoxic.

Barium (Ingestion)
RfD UF: 300
RfD Rats: 0.2 mg/kg-day (NTP, 1994)
NOAEL Rats: No data
LOAEL Rats: No data
BMDL Rats: 63 mg/kg-day
Oral Slope Factor: No data

Barium (Inhalation)
RfC UF: No data
RfC Rats: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data
Slope Factor: No data

9. BENZENE

Benzene or benzol is a colourless liquid with a sweet odour. Benzene is known to be a very flammable substance, which evaporates quickly into air and can dissolve slightly in water (ATSDR, 2007). Benzene can be found in water, air and soil. Benzene is widely used as an industrial solvent, as an intermediate in chemical syntheses, and as a component of gasoline. Natural sources of benzene includes gas emissions from volcanoes and forest fires (ATSDR, 2007). Inhalation exposure is the major route of exposure to benzene, although oral and dermal routes are also important. Several factors determine whether harmful health effects will occur after being exposed to benzene. These factors include the amount of benzene being exposed to and the length of exposure (ATSDR, 2007).

Benzene is toxic by all routes of administration. Hematotoxicity and immunotoxicity have been consistently reported to be the most sensitive indicators of noncancerous toxicity in both humans and experimental animals, and these effects have been the subject of several reviews (Aksoy, 1989; Goldstein, 1988, Snyder et al., 1993; Ross, 1996; U.S. EPA, 2002). Brief exposure of 5 to 10 minutes to very high levels (10 000-20 000 ppm) of benzene in the air can result in death. Where lower levels of 700 to 3000 ppm can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion and unconsciousness (ATSDR, 2007). In majority of such cases the effects will subside once the individuals are no longer exposed to the benzene. Chronic exposure to benzene results in progressive deterioration in hematopoietic function. Anaemia, leukopenia, lymphocytopenia, thrombocytopenia, pancytopenia, and aplastic anaemia have been reported after chronic benzene exposure (Aksoy, 1989; Goldstein, 1988).

Chronic Health Hazard Assessments for Noncarcinogenic Effects

Benzene (Ingestion)

RfD UF: 300

RfD Human Studies: 1 x 10^{-3}-4 x 10^{-3} mg/kg/day (Rothman et al., 1996)

LOAEL Human Studies: 7.6 ppm (Rothman et al., 1996)

BMDL: 1.2 mg/kg/day (Rothman et al., 1996)

BMCL: 23 mg/m^3 (Rothman et al., 1996)

Oral Slope Factor: 0.055 1/mg/kg/day (United States Environmental Protection Agency, 2003) [http://www.popstoolkit.com/tools/HHRA/SF_USEPA]

Benzene (Inhalation)

RfC UF: 300

RfC Human Studies: 3.0 x 10^{-2}-6 X 10^{-2} mg/m^3 (Human occupational inhalation study; Rothman et al., 1996) (Ward et al., 1985)

LOAEL Human Studies: 7.6 ppm (Rothman et al., 1996)

LOAEL Rat Studies: 300 ppm (Ward et al., 1985)

NOAEL Human Studies: 7.6 ppm (Rothman et al., 1996)

NOAEL Rat Studies: 30 ppm (Ward et al., 1985)

BMCL Rats: 8.2 mg/m^3 (U.S. EPA, 2000)

Inhalation Slope Factor: 0.027 1/mg/kg/day (United States Environmental Protection Agency, 2003) [http://www.popstoolkit.com/tools/HHRA/SF_USEPA]

Benzo[a]pyrene

Benzo[a]pyrene is a liquid and a polycyclic aromatic hydrocarbons (PAHs) which is a widespread environmental contaminant formed during incomplete combustion or pyrolysis of organic material. These substances are found in air, water, soils and sediments, generally at trace levels except near their sources. PAHs are present in some foods and in a few pharmaceutical products based on coal tar that are applied to the skin. Tobacco smoke contains high concentrations of PAHs (IARC, 1973, 1983, 2010). [https://monographs.iarc.fr/ENG/Monographs/vol100F/mono100F-14.pdf]. The general population can be exposed to benzo[a]pyrene through tobacco smoke, ambient air, water, soils, food and pharmaceutical products (IARC, 1973, 1983, 2010). Occupational exposure to PAHs occurs primarily through inhalation and via skin contact. Benzo[a]pyrene produces tumours in all animal species tested (mouse, rat, hamster, guinea-pig, rabbit, duck, newt, monkey) for which data were reported following exposure by many different routes (oral, dermal, inhalation, intratracheal, intrabronchial, subcutaneous, intraperitoneal, intravenous). Benzo[a] pyrene had both a local and a systemic carcinogenic effect, was an initiator of skin carcinogenesis in mice, and was carcinogenic in single-dose studies and following prenatal and transplacental exposures (IARC, 1973) (IARC, 1983) (IARC, 2010).
[https://monographs.iarc.fr/ENG/Monographs/vol100F/mono100F-14.pdf]

In several studies in which benzo[a]pyrene was applied to the skin of different strains of mice, benign (squamous cell papillomas and keratoacanthomas) and malignant (mainly squamouscell carcinomas) skin tumours were observed (Van Duuren et al., 1973; Cavalieri et al., 1977, 1988a; Levin et al., 1977; Habs *et al*., 1980, 1984; Warshawsky & Barkley, 1987; Albert et al., 1991; Andrews et al., 1991; Warshawsky et al., 1993). In a lifetime inhalation study (Thyssen *et al*., 1981) in male hamsters, benzo[a]pyrene induced dose-related increases in the incidence of papillomas and squamous-cell carcinomas in both the upper respiratory tract (nose, larynx and trachea) and the upper digestive tract (pharynx, oesophagus and forestomach).
[https://monographs.iarc.fr/ENG/Monographs/vol100F/mono100F-14.pdf]

Benzo[a]pyrene (Ingestion)
RfD UF: 300
RfD Rats: 3 x 10^{-4} mg/kg/d (Chen et al., 2012)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0136_summary.pdf#nameddest=rfd]
NOAEL Mice: No Data
LOAEL Mice: No Data
BMDL Rats: No Data
Oral Slope Factor: 1:100 000

Benzo[a]pyrene (Inhalation)
RfC UF: 3
RfC Rats: 2 x 10^{-6} mg/kg/d (Archibong et al., 2002)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0136_summary.pdf#nameddest=rfd]
NOAEL Human: NO Data
LOAEL Human: 25 µg/m^3 (Archibong et al., 2002)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0136_summary.pdf#nameddest=rfd]
BMCL Hamsters: 0.16 mg/m^3 (Thyssen et al., 1981)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0136_summary.pdf#nameddest=rfd]
Slope Factor: 1 per mg/kg-day (Beland and Culp, 1998)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0136_summary.pdf#nameddest=rfd]

10. BORON AS B

Boron compounds are used in the manufacture of glass, soaps and detergents and as flame retardants. The general population obtains the greatest amount of boron through food intake, as it is naturally found in many edible plants. Boron is found naturally in groundwater, but its presence in surface water is frequently a consequence of the discharge of treated sewage effluent, in which it arises from use in some detergents, to surface waters. Boron is actually a mixture of two stable isotopes, 10B (19.8%) and 11B (80.2%). Boron is a naturally-occurring element that is widespread in nature at relatively low concentrations Boron concentrations in rocks and soils are typically less than 10 ppm, although concentrations as high as 100 ppm have been reported in shales and some soils. Boron is not transformed or degraded in the environment, but depending

on environmental conditions (e.g. pH, moisture level), changes in the specific form of boron and its transport can occur [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1].

The most important source of exposure for human populations is ingestion of boron from food (primarily fruits and vegetables). Occupational exposure to borate dust and exposure to borates in consumer products (e.g. cosmetics, medicines, insecticides) are other potentially significant sources. Boron is well absorbed from the gastrointestinal tract in humans. Boron is absorbed during inhalation exposure. Seizures and other milder effects were reported in seven infants who consumed boron in a honey-borax mixture applied to pacifiers. The most frequent symptoms of boron poisoning are vomiting, abdominal pain, and diarrhoea. Other common symptoms include lethargy, headache, light-headedness, and rash. For boric acid, the minimum lethal dose by oral exposure is approximately 15-20 g in adults, 5-6 g in children, and 2-3 g in infants. The literature regarding toxicity of boron by inhalation exposure is sparse. A report from the Russian literature of reduced sperm count and sperm motility from semen analysis of six workers who were a part of a group of male workers exposed to very high concentrations of boron aerosols (22-80 mg/m^3) for over 10 years. These effects are consistent with the testicular effects reported in oral studies. [https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0410tr.pdf].

Boron (Ingestion)
RfD UF: 66 (Price *et al*., 1996a; Heindel *et al.*, 1992)
RfD Dogs: 2E-01 mg/kg-day (Price et al., 1996a; Heindel *et al*., 1992)
NOAEL Dogs: No data
LOAEL Dogs: No data
BMDL Dogs: 10.3 mg/kg-day (Price et al., 1996a; Heindel *et al*., 1992)
Oral Slope Factor: No data

Boron (Inhalation)
RfC UF: No data
RfC Rats: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data
Slope Factor: No data

11. BROMIDE

Bromide is commonly found in nature along with sodium chloride, owing to their similar physical and chemical properties, but in smaller quantities. Bromide concentrations in seawater range from 65 mg/l to well over 80 mg/l, in fresh water from trace amounts to about 0.5 mg/l and in desalinated waters up to 1 mg/l (WHO 2017a).

The results of human studies suggest a conservative no-observed-effect level (NOEL) (for marginal effect within normal limits of electroencephalograms in females) of 4 mg/kg body weight per day, giving an average daily intake of 0-0.4 mg/kg body weight, including a safety factor of 10 for population diversity. The upper limit of the average daily intake of 0-0.4 mg/kg body weight yields an acceptable total daily intake of 24 mg/person for a 60 kg person. Assuming a relative source contribution of 50%, the drinking-water value for a 60 kg adult consuming 2 litres/day would be up to 6 mg/l; for a 10 kg child consuming 1 litre/day, the value would be up to 2 mg/l. However, the dietary bromide contribution for a 10 kg child would probably be less than that for an adult. These are reasonably conservative values, and they are unlikely to be encountered in drinking-water supplies average daily intake (WHO 2017a).

Bromide can be involved in the reaction between chlorine and naturally occurring organic matter in drinking-water, forming brominated and mixed chloro-bromo by-products, such as trihalomethanes (THMs) and halogenated acetic acids (HAAs), or it can react with ozone to form bromate. The levels of bromide that can result in the formation of these substances are well below the health-based values suggested above. This guidance applies specifically to inorganic bromide ion and not to bromate or
organohalogen compounds, for which individual health-based guideline values have been developed (WHO 2017a).

Bromine disproportionates in water and physiological systems to bromide (stable) and hypobromite (unstable) ions; consequently exposure in living organisms is principally to the bromide ion. The only bromine residue of toxicological concern is bromate which has a maximum contaminant level of 0.01 mg/L (10 µg/L). Concentrated bromine is a potent irritant and systemic exposure is therefore limited. There is extensive clinical experience with various bromide salts based on their use as sedative-hypnotics and in treatment of seizures disorders. Repeated oral exposure in various mammalian species is associated with central nervous system effects expressed as behavioural and EEG changes. Repeated oral dosing also causes a hypothyroid effect that is specific to rats and not observed clinically or when assessed in volunteers [http://www.techstreet.com/direct/nsf/bromine_bromide_es.pdf].

Bromide/Bromine (Ingestion)
RfD UF: 10
RfD Human: 0.7 mg/kg/day (Van Gelderen et al., 1993) (Sangster et al., 1983)
[http://www.techstreet.com/direct/nsf/bromine_bromide_es.pdf]
NOAEL Human: 7 mg/kg/day (Van Gelderen et al., 1993) (Sangster et al., 1983)
[http://www.techstreet.com/direct/nsf/bromine_bromide_es.pdf]
LOAEL Human: No data
BMDL Human:
Oral Slope Factor: N/A
Bromide (Inhalation)
RfC UF: No data
RfC Human: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data
Slope Factor: No data

12. CADMIUM

Low levels of cadmium exposure occur through diet. Currently, these background exposures through diet are not believed to cause adverse health effects. Higher than average exposures to cadmium because of occupation, hobby, or personal habits such as smoking. The types of workers potentially exposed include: alloy makers, aluminum solder makers, ammunition makers, auto mechanics, battery makers, bearing makers, braziers and solderers, cable and trolley wire makers, cadmium alloy and cadmium-plate welders, cadmium platers, cadmium vapour lamp makers, ceramic and pottery makers, copper-cadmium alloy makers, dental amalgam makers, electric instrument makers, electrical condenser makers, electroplaters, engravers, glass makers, incandescent lamp makers, jewellers, lithographers, lithopane makers, metal sculptors, mining and refinery workers, municipal solid waste recovery workers, paint makers, paint sprayers, pesticide makers, pharmaceutical workers, photoelectric cell makers, pigment makers, plastic products makers, smelterers, solder makers, and textile printers (CSEM, 2008).
[https://www.atsdr.cdc.gov/csem/cadmium/docs/cadmium.pdf]

Once in the lungs, from 10% to 50% of an inhaled dose is absorbed, depending on particle size, solubility of the specific cadmium compound inhaled, and duration of exposure (Jarup, 2002). Most orally ingested cadmium passes through the gastrointestinal tract unchanged as normal individuals absorb only about 6% of ingested cadmium, but up to 9% may be absorbed in those with iron deficiency (ATSDR, 1999). Also, cadmium in water is more easily absorbed than cadmium in food (5% in water versus 2.5% in food) (IRIS, 2006). [https://www.atsdr.cdc.gov/csem/cadmium/docs/cadmium.pdf].

Depending on the route of exposure, cadmium has differing rates of absorption and varying health effects. Cadmium is a cumulative toxin. Its levels in the body increase over time because of its slow elimination (CSEM,

2008). It accumulates chiefly in the liver and kidneys. However, it also accumulates in muscle and bone. The principal organs affected by cadmium's toxicity, both acutely and chronically, are the: kidneys, bone, and lungs. The lungs can be damaged by acute inhalation exposures as well as suffering effects from more chronic occupational exposures. The kidneys can be damaged with both acute high-dose but more commonly, long-term chronic exposures. The bone disease that occurs with above average chronic exposures is thought to be secondary to cadmium's effects on the kidney. Cadmium's carcinogenic effects have been demonstrated in experimental animals; evidence in humans is somewhat less conclusive (CSEM, 2008). [https://www.atsdr.cdc.gov/csem/cadmium/docs/cadmium.pdf]

Cadmium (Ingestion)

RfD UF: 10

RfD Human: 5E-4 mg/kg/day (water), 1E-3 mg/kg/day (food) (U.S. EPA, 1985)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0141_summary.pdf]

NOAEL Human: 0.005 mg/kg/day (water), 0.01 mg/kg/day (food) (U.S. EPA, 1985)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0141_summary.pdf]

LOAEL Rats: No Data

BMDL Rats: 0.05 mg Cd/kg/day (Brzóska et al., 2005a, c, d) [https://www.atsdr.cdc.gov/toxprofiles/tp5-a.pdf]

Oral Slope Factor: N/A

Exposure ratio: 1:1000000 (Air: 6E-4 ug/cu.m)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0141_summary.pdf]

Cadmium (Inhalation)

RfC UF: No data

RfC Human: RfC for cadmium is currently under review (U.S. EPA, 1991).

NOAEL Human: No Data

LOAEL Rats: 0.01 mg Cd/m^3 (HEC) (NTP, 1995) [https://www.atsdr.cdc.gov/toxprofiles/tp5-a.pdf]

BMCL Human: No Data

Slope Factor: 6.1E+0 mg/kg/day (U.S. EPA, 1985) [https://rais.ornl.gov/tox/profiles/cadmium.html]

13. CALCIUM

Calcium is an element that belongs to the alkaline earth metals. It is an element that exists as a double positively-charged ion (Ca2+). Calcium is an element that occurs naturally at varying concentration in most water and, together with magnesium elements they are two of the main components of water hardness. Soft water has low concentrations of calcium, where as hard water has a high concentration of calcium.

Calcium is a very essential element/mineral for all living organisms and certain metabolic process and it is also a vital constituent of bone on mammalian skeletons.

High concentrations of calcium causes aesthetic effects, such as soap lathering and the scaling of domestic appliances. Scaling of domestic appliances, is an undesirable effect that occurs in household appliance, specifically water heating appliances such as kettles, geyser, boilers and some pipes. It results in the less efficient use of electrical power and any other type of fuel that is being used for heating purposes and there is also the partial obstruction of some pipes. Soap lathering, in water that has high concentrations of calcium, the calcium impairs the lathering of the soap through the formation of insoluble salts with long fatty acid chains that will precipitate as scum. This results in the excessive consumption of soap that is used for personal hygiene and in rare cases, the household cleaning operations. The scum that is created is unaesthetic, and it will ultimately lead over a period of time to the marking of the enamelled surfaces such as hand basins and baths. Essentially calcium does not have any major effects on human health, because it's an element that essential for human health (formation of bones and certain metabolic processes). Calcium has been reported to have a protective action against cardiovascular disease. However, the data that is currently available purporting the inverse relationship between the hardness of water or the calcium concentration in water, and the occurrence on cardiovascular disease do not show an unequivocal causal relationship. There is no conclusive evidence that has been recorded to support the claims for an increased incidence of human urinary tract and kidney stones which results from the long-term consumption of water with a high concentration of calcium. Calcium is also known to mitigate against the possible toxicity of certain types of heavy metals.

14. CARBON TETRACHLORIDE

Carbon tetrachloride is used mainly in the production of chlorofluorocarbon refrigerants, foam-blowing agents and solvents. Carbon tetrachloride is released mostly into the atmosphere but also into industrial wastewater. Although it readily migrates from surface water to the atmosphere, levels in anaerobic groundwater may remain elevated for months or even years. Although available data on concentrations in food are limited, the intake from air is expected to be much greater than that from food or drinking-water [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1]. No long-term toxicity data are available for humans with quantified oral exposures to carbon tetrachloride, but case reports identify the liver and kidney as the primary target organs following acute exposures. Evidence of acute oral hepatotoxicity in humans comes from observations of liver enlargement, elevated serum enzyme bilirubin levels, or histopathology. Other acute oral effects in humans include renal toxicity, usually delayed relative to hepatic toxicity and lung effects secondary to renal failure.

Case reports of acute high-level exposure to carbon tetrachloride vapour or long-term occupational exposure provide evidence of hepatotoxic and nephrotoxic effects of carbon tetrachloride in humans. Other effects associated with carbon tetrachloride exposure in humans are gastrointestinal (GI) symptoms (nausea and vomiting, diarrhoea, and abdominal pain) and neurological effects indicative of central nervous system depression (headache, dizziness, and weakness). The liver and kidney are the most prominent targets of carbon tetrachloride in sub-chronic and chronic inhalation studies of laboratory animals. The predominant targets of toxicity of carbon tetrachloride in humans (based on case reports of acute, high-level exposure, or long-term occupational exposure) and experimental animals following inhalation exposure are the liver and kidney. Studies in humans are inadequate to show an association between exposure to carbon tetrachloride and carcinogenicity. It is likely that the carcinogenicity of carbon tetrachloride is secondary to its hepatotoxic effects. There is some evidence for certain types of cancer in occupational populations thought to have had some exposure to carbon tetrachloride, including non-Hodgkin's lymphoma (NHL).
[https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=6].

Carbon tetrachloride (Ingestion)
RfD UF: 1000 (Bruckner *et al*., 1986)
RfD Rats: 0.004 mg/kg-day (Bruckner *et al*., 1986)
NOAEL Rats: No data
LOAEL Rats: No data
BMDL Rats: 3.9 mg/kg-day (Bruckner *et al*., 1986)
Oral Slope Factor: No data

Carbon tetrachloride (Inhalation)
RfC UF: 100 (Nagano *et al*., 2007b; JBRC, 1998)
RfC Rats: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Rats: 14.3 mg/m^3 (Nagano *et al.*, 2007b; JBRC, 1998)

15. CHLORIDE

Chloride is the anion of the element chlorine. Chorine does not occur in nature, but is found only as chloride. The chlorides of sodium, potassium, calcium and magnesium are all highly soluble in water. Chloride is of concern in domestic water supplies because elevated concentrations impart a salty taste to water and accelerate the corrosion rate of metals. High concentrations of chloride can also be detrimental to chloride-sensitive garden plants (South African Domestic Water Use, 2001). [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]. Chloride is a common constituent in water, is highly soluble, and once in solution tends to accumulate. Typically, concentrations of chloride in fresh water range from a few to several hundred mg/L. In sea water the concentration is approximately 19 800 mg/L. Chloride inputs to surface waters can arise from irrigation return flows, sewage effluent discharges and various industrial processes. Chloride can only be removed from water

by energy-intensive processes or ion exchange. Interactions. The taste threshold and the corrosion acceleration threshold of chloride are dependent on the action of other water quality constituents such as associated cations, the pH and the calcium carbonate concentration (South African Domestic Water Use, 2001). [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF].

Chloride is only detectable by taste at concentrations exceeding approximately 200 mg/L. A salty taste becomes quite distinctive at 400 mg/L and objectionable at greater than 600 mg/L. At chloride concentrations greater than 2 000 mg/L nausea may occur, while at 10 000 mg/L vomiting and dehydration may be induced. Chloride accelerates the corrosion rate of iron and certain other metals well below the concentration at which it is detectable by taste. The threshold for an increased corrosion rate is approximately 50 mg/L. At chloride concentrations greater than 200 mg/L, there is likely to be a significant shortening of the lifetime of domestic appliances as a result of corrosion (South African Domestic Water Use, 2001).
[http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]. According to the World Health Organization (1996) chloride concentrations in excess of about 250 mg/litre can give rise to detectable taste in water, but the threshold depends upon the associated cations. Consumers can, however, become accustomed to concentrations in excess of 250 mg/litre. No health-based guideline value is proposed for chloride in drinking-water. [http://www.who.int/water_sanitation_health/dwq/chloride.pdf]

16. CHLORINE

Chlorine is produced in large amounts and widely used both industrially and domestically as an important disinfectant and bleach. In particular, it is widely used in the disinfection of swimming pools and is the most commonly used disinfectant and oxidant in drinking-water treatment. In water, chlorine reacts to form hypochlorous acid and hypochlorites. Present in most disinfected drinking-water at concentrations of 0.2-1 mg/litre. [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1].

Ingestion is unlikely to occur because chlorine is a gas at room temperature. Solutions that are able to generate chlorine (e.g. sodium hypochlorite solutions) may cause corrosive injury if ingested. Most exposures to chlorine occur by inhalation. Chlorine's odour or irritant properties are discernible by most individuals at 0.32 ppm which is less than the OSHA permissible exposure limit (PEL) of 1 ppm. Chlorine's odour or irritant properties generally provide adequate warning of hazardous concentrations. However, prolonged, low-level exposures, such as those that occur in the workplace, can lead to olfactory fatigue and tolerance of chlorine's irritant effects. Chlorine is heavier than air and may cause asphyxiation in poorly ventilated, enclosed, or low-lying areas. In humans and animals exposed to chlorine in drinking-water, no specific adverse treatment-related effects have been observed. [https://www.atsdr.cdc.gov/mmg/mmg.asp?id=198&tid=36]. Due to the chemical relationship between chlorine and monochloramine, reproductive and developmental studies for monochloramine may be used to satisfy data gaps for chlorine.

Chlorine (Ingestion)
RfD UF: 100 (NTP, 1992)
RfD Rats: 0.1 mg/kg-day (NTP, 1992)
NOAEL Rats: 14.4 mg/kg-day (NTP, 1992)
LOAEL Rats: None
BMDL Rats: No data
Oral Slope Factor: No data

Chlorine (Inhalation)
RfC UF: No data
RfC Rats: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Rats: No data

17. CHLOROFORM

Chloroform is also known as trichloro methane or methyl trichloride. It is a colourless liquid with a pleasant, no irritating odour and a slightly sweet taste. Most of the chloroform found in the environment comes from industry. It will only burn when it reaches very high temperatures. Chloroform was one of the first inhaled anaesthetics to be used during surgery, but it is not used for anaesthesia today (ATSDR, 1997). [https://www.atsdr.cdc.gov/ToxProfiles/tp6-c1-b.pdf].

Chloroform enters the environment from chemical companies and paper mills. It is also found in waste water from sewage treatment plants and drinking water to which chlorine has been added. Chlorine is added to most drinking water and many waste waters to destroy bacteria. Small amounts of chloroform are formed as an unwanted product during the process of adding chlorine to water. Chloroform can enter the air directly from factories that make or use it and by evaporating from water and soil that contain it. It can enter water and soil when waste water that contains chlorine is released into water or soil. It may enter water and soil from spills and by leaks from storage and waste sites (ATSDR, 1997). [https://www.atsdr.cdc.gov/ToxProfiles/tp6-c1-b.pdf]. Chloroform can enter your body if you breathe air, eat food, or drink water that contains chloroform. Chloroform easily enters your body through the skin. Therefore, chloroform may also enter your body if you take a bath or shower in water containing chloroform. In addition, you can breathe in chloroform if the shower water is hot enough for chloroform to evaporate (ATSDR, 1997). [https://www.atsdr.cdc.gov/ToxProfiles/tp6-c1-b.pdf]

In humans, chloroform affects the central nervous system (brain), liver, and kidneys after a person breathes air or drinks liquids that contain large amounts of chloroform. Chloroform was used as an anaesthetic during surgery for many years before its harmful effects on the liver and kidneys were recognized. Breathing about 900 parts of chloroform in a million parts of air (900 ppm or 900,000 ppb) for a short time causes fatigue, dizziness, and headache. If you breathe air, eat food, or drink water containing elevated levels of chloroform,

over a long period, the chloroform may damage your liver and kidneys. Large amounts of chloroform can cause sores when the chloroform touches your skin. The EPA sets rules for the amount of chloroform allowed in water. The EPA limit for total trihalomethanes, a class of chemicals that includes chloroform, in drinking water is 100 micrograms per litre (µg/L, 1 µg/L = 1 ppb in water) (ATSDR, 1997). [https://www.atsdr.cdc.gov/ToxProfiles/tp6-c1-b.pdf]

There have been no studies of toxicity or cancer incidence in humans chronically exposed to chloroform (alone) via drinking water. However, there have been a number of epidemiological studies on cancer risk in humans exposed to chlorinated drinking water (e.g. Cantor et al., 1985; McGeehin et al., 1993; King and Marrett, 1996; Doyle et al., 1997; Freedman et al., 1997; Cantor et al., 1998; Hildesheim et al., 1998). Chlorinated drinking water typically contains chloroform, along with other trihalomethanes and a wide variety of other disinfection by products (U.S. EPA, 1994d). It should be noted that humans exposed to chloroform in drinking water are likely to be exposed both by direct ingestion and by inhalation of chloroform gas released from water into indoor air (EPA, 2001) [https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0025tr.pdf]

Chloroform (Ingestion)
RfD UF:100
RfD Dogs: 0.01 mg/kg/d (Heywood et al., 1979)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0025_summary.pdf]
NOAEL Dogs: None (Heywood et al., 1979)
LOAEL Dogs: 12.9 mg/kg/day (Heywood et al., 1979)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0025_summary.pdf]
BMDL Dogs: 1.0 mg/kg/day (Heywood et al., 1979)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0025_summary.pdf]
Oral Slope Factor: N/A

Chloroform (Inhalation)
RfC UF: No data
RfC Human: Not available (EPA has not established a Reference Concentration (RfC) for chloroform)
[https://www.epa.gov/sites/production/files/2016-09/documents/chloroform.pdf]
NOAEL Human: Not available
LOAEL Human: Not available
BMCL Human: Not available
Slope Factor: **0.081 mg/kg/day** (US EPA, 2007)

18. CHROMIUM (VI)

Chromium is an odourless and tasteless metallic element. Chromium is found naturally in rocks, plants, soil and volcanic dust, and animals. Chromium-6 occurs naturally in the environment from the erosion of natural chromium deposits. It can also be produced by industrial processes. There are demonstrated instances of chromium being released to the environment by leakage, poor storage, or inadequate industrial waste disposal practices (EPA, 2017). [https://www.epa.gov/dwstandardsregulations/chromium-drinking-water]

Chromium is used in three basic industries: metallurgical, chemical, and refractory (heat-resistant applications). These industries are the most important industrial sources of chromium in the atmosphere [EPA, 1998; ATSDR, 2000]. In the metallurgical industry, chromium is an important component of stainless steels and various metal alloys. Metal joint prostheses made of chromium alloys are widely used in clinical orthopaedics. In the chemical industry, chromium is used primarily in chrome plating, leather tanning, paint pigments (chromium compounds can be red, yellow, orange, and green), and o wood treatment; smaller amounts in catalysts, copy machine toner, corrosion inhibitors, drilling muds, magnetic tapes, photographic chemicals, safety matches, and water treatment. [https://www.atsdr.cdc.gov/csem/chromium/docs/chromium.pdf]

EPA has a drinking water standard of 0.1 milligrams per litre (mg/l) or 100 parts per billion (ppb) for total chromium. This includes all forms of chromium, including chromium-6 (EPA, 2017). [https://www.epa.gov/dwstandardsregulations/chromium-drinking-water]. The effects of chromium-6 when it is ingested have been the subject of much debate. It is a known fact that when some forms of chromium-6 are inhaled, they can cause cancer. However, experts have disagreed on its toxicity in drinking water due in part to the possible changes to chromium-6 in the stomach when it is ingested. EPA currently regulates total chromium based on noncancerous effects of the chemical such as its ability to cause liver damage, harm the kidney, damage nerve tissues, and cause skin irritations (ACWA, 2017). [http://www.acwa.com/content/chromium-6].

When inhaled, chromium compounds are respiratory tract irritants and can cause pulmonary sensitization. Chronic inhalation of Cr(VI) compounds increases the risk of lung, nasal, and sinus cancer. Severe dermatitis and usually painless skin ulcers can result from contact with Cr(VI) compounds. Chromium compounds can be sensitizers as well as irritants. DHHS, EPA, WHO, and IARC have all recognized Cr(VI) as a human carcinogen. Occupational exposure to Cr(VI) compounds in a number of industries has been associated with increased risk of respiratory system cancers. • Latency for Cr(VI)-induced lung cancer can be greater than 20 years. Some studies indicated that reversible renal tubular damage can occur after low-dose, chronic Cr(VI) exposure. Cr(VI) compounds can cause mild to severe liver abnormalities. Some Cr(VI) compounds, such as potassium dichromate and chromium trioxide, are caustic and irritating to gastrointestinal mucosal tissue.

Ingestion of a lethal dose of chromate can result in cardiovascular collapse. Oral exposure to Cr(VI) compounds may result in haematological toxicity. Potential reproductive effects of chromium in humans have not been adequately investigated. Data indicate that Cr(VI) compounds are teratogenic in animals. Cr(VI) compounds induced DNA damage, gene mutation, sister chromatid exchange, chromosomal aberrations in a

number of targets, including animal cells in vivo and animal and human cells in vitro (ATSDR, 2008) [https://www.atsdr.cdc.gov/csem/chromium/docs/chromium.pdf.]

Chromium (VI) (Ingestion)
RfD UF: 300
RfD Rats: 3E-3 mg/kg-day (MacKenzie et al., 1958)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0144_summary.pdf]
RfD Mice: 9.0E-4 mg/kg-day (ATSDR, 2012) [http://www.michigan.gov/documents/deq/deq-rrd-chem-ChromiumVIDatasheet_527895_7.pdf]
NOAEL Rats: 2.5 mg/kg-day (MacKenzie et al., 1958)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0144_summary.pdf]

LOAEL Rats: None
BMDL Rats: 0.09 mg/kg-day (MacKenzie et al., 1958) [http://www.michigan.gov/documents/deq/deq-rrd-chem-ChromiumVIDatasheet_527895_7.pdf]
Oral Slope Factor: N/A

Chromium (VI) (Inhalation)
RfC UF: 90
RfC Human: 8E-6 mg/m^3 (aerosols) (Lindberg and Hedenstierna, 1983)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0144_summary.pdf]
RfC Rats: 1E-4 mg/m^3 (particulates) (Glaser et al., 1990) (Malsch et al., 1994)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0144_summary.pdf]
NOAEL Human: None
LOAEL Human: 2E-3 mg/m^3 (Lindberg and Hedenstierna, 1983)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0144_summary.pdf]
BMCL Human: 0.01 mg/m^3 (Lindberg and Hedenstierna, 1983)
[http://www.michigan.gov/documents/deq/deq-rrd-chem-ChromiumVIDatasheet_527895_7.pdf]

Slope Factor:
Risk exposure: 1:1000000 (8E-5 ug/m^3) (Mancuso, 1975)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0144_summary.pdf]

19. CHROMIUM (III)

Chromium is an odourless and tasteless metallic element. Chromium is found naturally in rocks, plants, soil and volcanic dust, and animals. Chromium-3 is an essential human dietary element. It is found in many vegetables, fruits, meats, grains, and yeast. There are demonstrated instances of chromium being released to the environment by leakage, poor storage, or inadequate industrial waste disposal practices. Chromium is released to air primarily by combustion processes and metal industries. Non-occupational sources of chromium include contaminated soil, air, water, smoking, and diet (ATSDR) [https://www.atsdr.cdc.gov/csem/chromium/docs/chromium.pdf]. Breathing chromium (III) does not irritate the nose or mouth in most people. There is not enough data to know if chromium (III) causes cancer. Eating small amounts of chromium (III) is healthy but eating too much is harmful. The recommended daily dose of chromium (III) is 50-200 µg. There is not enough data to know if eating large amounts of chromium (III) causes cancer (http://dhss.delaware.gov/dph/files/chromiumfaq.pdf).

Chromium (III) (Ingestion)
RfD UF: 100
RfD Rats: 1.5E+0 mg/kg-day (Ivankovic & Preussman, 1975)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0028_summary.pdf]
NOAEL Rats: 1,468 mg/kg-day (Ivankovic & Preussman, 1975)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0028_summary.pdf]
LOAEL Rats: No Data
BMDL Rats: No Data
Oral Slope Factor: N/A

Chromium (III) (Inhalation)
RfC UF: No data
RfC Human: A number of animal studies confirm that trivalent chromium is poorly absorbed in the gastrointestinal tract. (Donaldson & Barreras (1966); Anderson et al. (1986); Anderson et al. (1983); Visek et al. (1953); Mertz et al. (1965); MacKenzie et al. (1959); Ogawa (1976); Henderson et al. (1979))
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0028tr.pdf]
NOAEL Human: No Data
LOAEL Rats: 3 mg/m^3 (Derelanko et al., 1999) [https://www.atsdr.cdc.gov/toxprofiles/tp7-c8.pdf]
BMCL Human: No Data
Slope Factor: N/A

20. COLOUR

Drinking-water should ideally have no visible colour. Colour in drinking-water is usually due to the presence of coloured organic matter (primarily humic and fulvic acids) associated with the humus fraction of soil. Colour is also strongly influenced by the presence of iron and other metals, either as natural impurities or as corrosion products. It may also result from the contamination of the water source with industrial effluents and may be the first indication of a hazardous situation. The source of colour in a drinking-water supply should be investigated, particularly if a substantial change has taken place. Water that is fit for use water should be clear with no noticeable colour deposits. Common colours include (USA EPA; SAWQGs, 1996):

- Red or Brown Colour – A red, brown or rusty colour is generally indicative of iron or manganese in your water. Iron and manganese may also be found in association with humic acids or lignins. Disadvantages to iron in your water include stains in sinks, or discoloured laundry.
- Yellow Colour – This colouration occurs in areas where the water has passed through swamps and then moved through peat soils. It is more commonly found in surface water supplies and shallow wells. Although the yellow colour may be displeasing, it presents no health hazard, as it is only small particles suspended in the water.
- Blue or Green Colour – A green or blue colour is generally a result of copper, or copper pipes and corrosive water. The copper can cause staining of your fixtures and your laundry. Copper is regulated in drinking water by the EPA at 1.3 PPM. This is at a low enough concentration that the copper cannot be tasted (the taste threshold is around 5 PPM). Copper can become a problem if it is higher than 30 PPM. Effects at this dose are vomiting, diarrhoea, and general gastrointestinal distress.
- Cloudy White or Foamy – Cloudy water is usually due to turbidity (apparent colour). Turbidity is caused by finely divided particles in the water. When light hits the water, it is scattered, giving a cloudy look to the water. The particles may be of either organic or inorganic nature. Neither one causes any harmful effects to the body, although they can cause abrasions to pipes, or possible staining of sinks.

Most people can detect colour above 15 true colour units (TCU) in a glass of water. Levels of colour below 15 TCU are often acceptable to users. High colour from natural organic carbon (e.g. humics) could also indicate a high propensity to produce by-products from disinfection processes. No health-based guideline value is proposed for colour in drinking-water. An arbitrary standard scale has been developed for measuring colour intensity in water samples. When a water is rated as having a colour of 5 units, it means: the colour of this water is equal in intensity to the colour of distilled water containing 5 milligrams of platinum as potassium chloroplatinate per litre. Highly coloured water is objectionable for most process work in the industrial field because excessive colour causes stains. While colour is not a factor of great concern in relation to household applications, excessive colour lacks appeal from an aesthetic standpoint in a domestic water. Further, it can cause staining. U.S. EPA Secondary Drinking Water Regulations and the SA Water Quality Guidelines, 1996 and SANS 241 drinking Water Standard (2015) recommend that a drinking/domestic water possess colour of less than 15 units. In general, colour is reduced or removed from water through the use of coagulation, settling and filtration techniques.

21. COPPER

Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and, at low levels, air. Its average concentration in the earth's crust is about 50 parts copper per million parts soil (ppm) (ATSDR, 2004). Copper also occurs naturally in all plants and animals. It is an essential element for all known living organisms including humans and other animals at low levels of intake. At much higher levels, toxic effects can occur. The most commonly used compound of copper is copper sulphate. Many copper compounds can be recognized by their blue green colour. Copper is extensively mined and processed in various parts of the world and is primarily used as the metal or alloy in the manufacture of wire, sheet metal, pipe, and other metal products. Copper compounds are most commonly used in agriculture to treat plant diseases, like mildew, or for water treatment and as preservatives for wood, leather, and fabrics (ATSDR, 2004).

You may be exposed to copper by breathing air, drinking water, eating food, and by skin contact with soil, water and other copper-containing substances. Most copper compounds found in air, water, sediment, soil and rock are strongly attached to dust and dirt or imbedded in minerals. You can take copper into your body upon ingestion of water or soil that contains copper or by inhalation of copper containing dust. In the general population, soluble copper compounds (those that dissolve in water), which are most commonly used in agriculture, are more likely to threaten your health. You may breathe high levels of copper-containing dust if you live or work near copper mines or processing facilities. You may be exposed to levels of soluble copper in your drinking water that are above the acceptable drinking water standard of 1,300 parts copper per billion parts of water (ppb), especially if your water is corrosive and you have copper plumbing and brass water fixtures (ATSDR, 2004).

Long-term exposure to copper dust can irritate your nose, mouth, and eyes, and cause headaches, dizziness, nausea, and diarrhoea. If you drink water that contains higher than normal levels of copper, you may experience nausea, vomiting, stomach cramps, or diarrhoea. Intentionally high intakes of copper can cause liver and kidney damage and even death. We do not know if copper can cause cancer in humans. EPA does not classify copper as a human carcinogen because there are no adequate human or animal cancer studies (ATSDR, 2004).

Copper (Ingestion)
RfD UF: No data
RfD Human: Not Available (IRIS, 1988)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0368_summary.pdf]
RfD Rats: Not Available (IRIS, 1988)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0368_summary.pdf]
NOAEL Human: Not available (IRIS, 1988)
LOAEL Human: Not Available (IRIS, 1988)
BMDL Human: None (IRIS, 1988)
Oral Slope Factor: N/A (IRIS, 1988)

Copper (Inhalation)
RfC UF: No data
RfC Human: Not Available (IRIS, 1988)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0368_summary.pdf]
NOAEL Human: Not Available (IRIS, 1988)
LOAEL Human: Not Available (IRIS, 1988)
BMCL Human: None (IRIS, 1988)
Slope Factor: N/A (IRIS, 1988)

22. CYANIDE

Cyanide compounds are used in a number of industrial processes, including mining, metallurgy, manufacturing, and photography, due to their ability to form stable complexes with a range of metals. Cyanide has been employed extensively in electroplating. The cyanide salts, sodium cyanide (NaCN) and potassium cyanide (KCN), have also been used as rodenticides. Use in industrial processes is the main origin of cyanide in the environment, but cyanide is also released from biomass burning, volcanoes, and natural biogenic processes from higher plants, bacteria, and fungi (ATSDR, 2006). Additionally, cyanogenic compounds, which are converted to cyanide in the body, naturally occur in many plant foods, including cassava root, almonds, millet sprouts, lima beans, soy, spinach, bamboo shoots, and sorghum. Exposure to cyanide also occurs from smoking. https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0060tr.pdf

Available data show that cyanide is absorbed via the oral, inhalation, and dermal routes in humans, although quantitative data on the percent or extent of absorption are limited. Immediately following oral exposure in humans, tissues containing cyanide included the liver, brain, spleen, blood, kidneys, and lungs. Following acute inhalation exposure in humans and animals, cyanide is found in the lung, heart, blood, kidneys, and brain. The major metabolic pathway for cyanide is conversion to thiocyanate, primarily by rhodanese.
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0060tr.pdf].

No epidemiologic studies exist of long-term human exposure to cyanide by the oral route. Information on human oral exposure to cyanide is limited to acute effects following suicide attempts or accidental poisoning. Acute oral exposure to cyanide has been observed to result in typical signs of cyanide poisoning, including central nervous system depression, convulsions, coma, and death. Chronic and subchronic oral studies in experimental animals indicate that the thyroid, central nervous system, and male reproductive organs are sensitive targets of cyanide toxicity.
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0060tr.pdf].

Cyanide has not been subjected to a complete standard battery of genotoxicity assays, although, overall, the available data indicate that cyanide is not genotoxic. No adequate carcinogenicity studies of cyanide are available in animals or humans.

Cyanide (Ingestion)
RfD UF: 3 000
RfD Human: 6.3 x 10^{-4} mg/kg-day
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf]
NOAEL Human: Not available (IRIS, 1988)
LOAEL Human: Not Available (IRIS, 1988)
BMDL Human: 1.9 mg/kg-day
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf]
Oral Slope Factor: N/A (IRIS, 1988)

Copper (Inhalation)
RfC UF: 3 000
RfC Human: 8 X 10^{-4} mg/m^3
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0060_summary.pdf]
NOAEL Human: Not Available (IRIS, 1988)
LOAEL Human: Not Available (IRIS, 1988)
BMCL Human: None (IRIS, 1988)
Slope Factor: N/A (IRIS, 1988)

23. DISSOLVED ORGANIC CARBON

Dissolved organic carbon (DOC) is a general description of the organic material dissolved in water. Organic carbon occurs as the result of decomposition of plant or animal material. Organic carbon present in soil or water bodies may then dissolve when contacted by water. This dissolved organic carbon moves with both surface water and ground water (Government of Saskatchewan, 2009). [http://www.saskh2o.ca/PDF-WaterCommittee/DissolvedOrganicCarbon.pdf]. DOC does not pose health risk itself but may become potentially harmful when in combination with other aspects of your water. When water with high DOC is chlorinated, harmful by products called trihalomethanes may be produced. Trihalomethanes may have long-term effects on health and they should be considered when chlorinating drinking water high in DOC. Organic material in water can cause aesthetic problems such as an unpleasant taste, odour and colour. Organic content is usually higher in surface water than ground water. DOC concentrations greater than 5 mg/L will complicate water treatment and may result in disinfection by-products, such as trihalomethanes, to be formed in amounts exceeding the standards. DOC will also increase colour in the finished water.

24. DDT AND METABOLITES

The structure of DDT permits several different isomeric forms, and commercial products consist predominantly of p,p'-DDT. Its use has been restricted or banned in many parts of the world, although DDT is still used in some countries for the control of vectors that transmit yellow fever, sleeping sickness, typhus, malaria and other insect-transmitted diseases. DDT and its metabolites are persistent in the environment and resistant to

complete degradation by microorganisms. Food is the major source of intake of DDT and related compounds for the general population.

DDT is listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines. IARC classified the DDT complex as a non-genotoxic carcinogen in rodents and a potent promoter of liver tumours. IARC has concluded that there is insufficient evidence in humans and sufficient evidence in experimental animals for the carcinogenicity of DDT (Group 2B) based upon liver tumours observed in rats and mice [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1].

DDT is known to be absorbed by humans in direct proportion to dietary exposure. Humans are predominantly exposed to DDT, DDE, and DDD by eating foods containing small amounts of these compounds. The amount of DDT in food has greatly decreased since DDT was banned in many countries and should continue to decline. The largest fraction of DDT in a person's diet comes from meat, poultry, dairy products, and fish, including the consumption of sport fish. Leafy vegetables generally contain more DDT than other vegetables, possibly because DDT in the air is deposited on the leaves. Infants may be exposed by drinking breast milk.

DDT or its breakdown products are still present in some air, water, and soil samples. However, levels in most air and water samples are presently so low that exposure is of little concern. DDT levels in air have declined to such low levels that it often cannot be detected. In cases where DDT has been detected in air, it is associated with air masses coming from regions where DDT is still used or from the evaporated DDT from contaminated water or soil.

Eating food with large amounts (grams) of DDT over a short time would most likely affect the nervous system. People who swallowed large amounts of DDT became excitable and had tremors and seizures. They also experienced sweating, headache, nausea, vomiting, and dizziness. These effects on the nervous system went away once exposure stopped. The same type of effects would be expected by breathing DDT particles in the air or by contact of the skin with high amounts of DDT. Tests in laboratory animals confirm the effect of DDT on the nervous system [https://www.atsdr.cdc.gov/toxprofiles/tp35.pdf].

Although not common today, exposure to DDT could also occur through inhalation or absorption through the skin during the handling or application of DDT. The primary routes of exposure are inhalation and dermal; however, absorption of DDT from the lungs may not have been significant, and ingestion due to the mucociliary apparatus of the respiratory tract is more likely [https://www.atsdr.cdc.gov/toxprofiles/tp35.pdf].

DDT (Ingestion)
RfD UF: 100 (Laug et al., 1950)
RfD Rats: 5E-04 mg/kg-day (Laug et al., 1950)
NOAEL Rats: 0.05 mg/kg-day (Laug et al., 1950)
LOAEL Rats: 5 ppm (Laug et al., 1950)
BMDL Rats: No data
Oral Slope Factor: 1.5E-1

DDT (Inhalation)
RfC UF: No data
RfC Rats: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Rats: No data

25. *ESCHERICHIA COLI* (*E. COLI*)

Faecal coliforms, and more specifically *Escherichia coli* (*E. coli*), are the most commonly used bacterial indicators of faecal pollution. This indicator group is used to evaluate the quality of wastewater effluents, river water, sea water at bathing beaches, raw water for drinking water supply, treated drinking water, water used for irrigation and aquaculture and recreational waters. The presence of *Escherichia coli* is used to confirm the presence of faecal pollution by warm-blooded animals (often interpreted as human faecal pollution). Some organisms detected as faecal coliforms may not be of human faecal origin but are almost definitely from warm-blooded animals (South African Domestic Water Use, 2001).
[http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]. Faecal coliforms have been shown to represent 93-99% of coliform bacteria in faeces from humans, poultry, cats, dogs and rodents. *Escherichia coli* usually comprises approximately 97% of coliform bacteria in human faeces. Total coliforms are usually enumerated as counts (number of colonies)/100 mL.

Faecal coliforms are primarily used to indicate the presence of bacterial pathogens such as *Salmonella* spp., *Shigella* spp*. Vibrio cholerae*, *Campylobacter jejuni, Campylobacter coli, Yersinia enterocolitica* and pathogenic *E. coli*. These organisms can be transmitted via the faecal/oral route by contaminated or poorly-treated drinking water and may cause diseases such as gastroenteritis, salmonellosis, dysentery, cholera and typhoid fever. The risk of being infected by microbial pathogens correlates with the level of contamination of the water and the amount of contaminated water consumed. Higher concentrations of faecal coliforms in water will indicate a higher risk of contracting waterborne disease, even if small amounts of water are consumed (South African Domestic Water Use, 2001) [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

26. ETHYL BENZENE

Colourless liquid that smells like gasoline. It evaporates at room temperature and burns easily. Ethylbenzene is found naturally in oil. Ethylbenzene is also used in fuels. Consumer products containing ethylbenzene include: gasoline, paints and inks, pesticides, carpet glues, varnishes and paints, tobacco products and automobile products (ATSDR, 2010). Ethylbenzene moves easily into the air from water and soil. Ethylbenzene in soil can also contaminate groundwater. Rapidly broken down in air. In air ethylbenzene is broken down in less than 3 days with the aid of sunlight. In surface water such as rivers and harbours, ethylbenzene breaks down by reacting with other compounds naturally present in water. In the soil, ethylbenzene is broken down by soil bacteria (ATSDR, 2010).

When you breathe air containing ethylbenzene, it enters your body rapidly and almost completely through your lungs. Ethylbenzene in food or water may also rapidly and almost completely enter your body through the digestive tract. It may enter through your skin when you come into contact with liquids containing ethylbenzene (ATSDR, 2010). Exposure to high levels of ethylbenzene in the air for short periods can cause eye and throat irritation. Exposure to higher levels can result in vertigo and dizziness (Delaware Health and Social Services, 2013).

The following effects that ethylbenzene might have on animals, can be assumed to also have an effect on humans. Exposure to relatively low concentrations of ethylbenzene for several days to weeks resulted in potentially irreversible damage to the inner ear and hearing of animals. Exposures to relatively low concentrations of ethylbenzene for several months to years caused kidney damage in animals. There is no clear evidence that ethylbenzene affects fertility. An increase in kidney tumours in rats and lung and liver tumours in mice were found after they were exposed to ethylbenzene in air for 2 years.

The International Agency for Research on Cancer (an expert group that is part of the World Health Organization) has determined that long-term exposure to ethylbenzene may cause cancer in humans. Rats exposed to large amounts of ethylbenzene by mouth had severe damage to the inner ear. Liquid ethylbenzene caused eye damage and skin irritation in rabbits (ATSDR, 2010). [https://www.atsdr.cdc.gov/phs/phs.asp?id=381&tid=66] [http://dhss.delaware.gov/dph/files/ethylbenfaq.pdf]

No information is available about the effects of exposure to ethylbenzene on children. It is likely that children would show the same health effects as adults. There is no evidence to show whether children will have effects at the same exposure levels as adults. There is also no evidence to show whether ethylbenzene causes birth defects in people. Minor birth defects and low birth weights have occurred in new born animals whose mothers were exposed to air contaminated with ethylbenzene during pregnancy.

Ethyl benzene (Ingestion)

RfD UF: 1000

RfD Human: Not Available

RfD Rats: 0.1 mg/kg/day (Wolf et al., 1956)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0051_summary.pdf]
4.0E-2 mg/kg/day (ATSDR, 2010)
[http://www.michigan.gov/documents/deq/deq-rrd-chem-EthylbenzeneDatasheet_527947_7.pdf]

NOAEL Rats: 97 mg/kg/day (Wolf et al., 1956)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0051_summary.pdf]
LOAEL Rats: 291 mg/kg/day (Wolf et al., 1956)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0051_summary.pdf]
BMDL Rats: 10.68 mg/kg/day (Human equivalent concentration) (Mellert et al., 2007)
[https://www.atsdr.cdc.gov/toxprofiles/tp110-c8.pdf]
Oral Slope Factor: 1.1E-2 mg/kg/day (CALEPA, 2011)
[http://www.michigan.gov/documents/deq/deq-rrd-chem-EthylbenzeneDatasheet_527947_7.pdf]

Ethyl Benzene (Inhalation)

RfC UF: 300

RfC Rats & Rabbits: 1E+0 mg/cu.m (Andrew et al., 1981) (Hardin et al., 1981)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0051_summary.pdf]
2.6E+2 mg/cu.m (ATSDR, 2010)
[http://www.michigan.gov/documents/deq/deq-rrd-chem-EthylbenzeneDatasheet_527947_7.pdf]

NOAEL Rats & Rabbits (+HEC): 434 mg/cu.m (100 ppm) (Andrew et al., 1981) (Hardin et al., 1981)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0051_summary.pdf]

LOAEL Rats & Rabbits: 4340 mg/cu.m (1000 ppm) (Andrew et al., 1981) (Hardin et al., 1981)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0051_summary.pdf]
75 ppm (HEC 17.45 ppm) (NTP, 1999)
[https://www.atsdr.cdc.gov/toxprofiles/tp110-c8.pdf]

BMCL (Rats) Human Equivalent Concentration: 154.26 ppm (Cappaert et al., 2000)
[https://www.atsdr.cdc.gov/toxprofiles/tp110-c8.pdf]
63.64 ppm (Gagnaire et al., 2007)
[https://www.atsdr.cdc.gov/toxprofiles/tp110-c8.pdf]

Slope Factor: N/A

27. FLUORIDE

Fluoride occurs naturally in our environment but we consume it in small amounts. Exposure can occur through dietary intake, respiration and fluoride supplements. The most important factor for fluoride presence in alimentation is fluoridated water (Kanduti *et al.*, 2016). [https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4851520/pdf/MSM-28-133.pdf]. Fluoride is also present in products such as: toothpastes, mouth rinses, varnishes, fluoride gels. Other sources include processed foods made with fluoridated water, fluoride-containing pesticides, bottled teas, fluorinated pharmaceuticals, Teflon pans, and mechanically deboned chicken (Fluoride Action Network, 2017). [http://fluoridealert.org/issues/sources]

Current safety standards for fluoride are based on the premise that severe dental fluorosis and crippling skeletal fluorosis are the first adverse effects that fluoride can have on the body (Fluoride Action Network, 2017). These effects represent the crudest, most obvious harm caused by fluoride. Research already shows, in fact, that fluoride can cause arthritic symptoms and bone fracture well before the onset of crippling fluorosis, and can affect many other tissues besides bone and teeth, including the brain and thyroid gland. People with clinical signs of fluorosis can suffer significant symptoms, including chronic joint pain and overt osteoarthritis (Fluoride Action Network, 2017). The National Research Council (NRC) concluded that the allegedly “safe” upper limit of fluoride in water (4 mg/l) is toxic to human health. While the NRC did not determine the safe level, their conclusion means that the safe level is less than 4 times the level added to water (0.7-1.2 mg/l) in community fluoridation programs. This is far too slim a margin to protect vulnerable members of the population, including those who consume high amounts of water (Fluoride Action Network, 2017). [http://fluoridealert.org/issues/health/].

Fluoride (Ingestion)

RfD UF: No data

RfD Human: 6E-2 mg/kg/day (Hodge, 1950)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0053_summary.pdf]

NOAEL Human: 0.06 mg/kg/d (Hodge, 1950)

LOAEL Human: 2 ppm (Hodge, 1950)

BMDL Human: No available data

Oral Slope Factor: N/A

Fluoride (Inhalation)

RfC UF: No data

RfC Human: No available data

NOAEL Human: No available data

LOAEL Human: No available data

BMCL Human: No available data

Slope Factor: No available data

28. GLYPHOSATE & AMINOMETHYLPHOSPHONIC ACID (AMPA)

Glyphosate is a broad-spectrum post-emergence herbicide. It has a high activity when applied to foliage, and it is used worldwide in both agriculture and forestry. Glyphosate is also used for aquatic weed control (IPCS, 1994). AMPA has no commercial use (WHO, 2005) [http://www.who.int/water_sanitation_health/dwq/chemicals/glyphosateampa290605.pdf]
Aminomethylphosphonic acid (AMPA) is a chemical formed through the breakdown of glyphosate (MDH, 2017) [http://www.health.state.mn.us/divs/eh/risk/guidance/gw/ampainfo.pdf]

Glyphosate is chemically stable in water and is not subject to photochemical degradation (FAO/WHO, 1986). The low mobility of glyphosate in soil indicates a minimal potential for the contamination of groundwater. Glyphosate can, however, enter surface and subsurface waters by direct use near aquatic environments or by runoff or leaching from terrestrial applications.

Based on available information, Minnesota Department of Agriculture (MDA) developed a guidance value of 1,000 parts per billion (ppb) for AMPA in drinking water. A person drinking water at or below the guidance value would have little or no risk for health effects [http://www.health.state.mn.us/divs/eh/risk/guidance/gw/ampainfo.pdf]

Several cases of (mostly intentional) intoxications with technical glyphosate herbicide formulation have been reported. A typical symptom is erosion of the gastrointestinal tract. No compound-related effects were observed in a test group of five applicators prior to and after exposure for 1 week. No controlled studies have been conducted in humans (WHO, 2005) [http://www.who.int/water_sanitation_health/dwq/chemicals/glyphosateampa290605.pdf]. AMPA caused minor liver injury and urinary bladder effects in laboratory animals, in addition to decreased body weight gain (MDH, 2017). [http://www.health.state.mn.us/divs/eh/risk/guidance/gw/ampainfo.pdf]

Glyphosate and AMPA (Ingestion)

UF Rats: 100

RfD Rats: 1.00E-01 (Monsanto Co., 1981a)

NOAEL Rats: 10 mg/kg/d (Monsanto Co., 1981a)

LOAEL Rats: No Data

BMDL Rats: No Data

Oral Slope Factor: N/A

Glyphosate and AMPA (Inhalation)

RfC Human: Not evaluated (IRIS, 1987)

NOAEL Human: Not evaluated (IRIS, 1987)

LOAEL Human: Not evaluated (IRIS, 1987)

BMCL Human: Not evaluated (IRIS, 1987)

Slope Factor: Not evaluated (IRIS, 1987)

29. GROSS ALPHA AND BETA PARTICLES

Alpha particles are charged particles, which are emitted from naturally occurring materials (such as uranium, thorium, and radium) and man-made elements (such as plutonium and americium). These alpha emitters are primarily used (in very small amounts) in items such as smoke detectors. In general, alpha particles have a very limited ability to penetrate other materials. In other words, these particles of ionizing radiation can be blocked by a sheet of paper, skin, or even a few inches of air. Nonetheless, materials that emit alpha particles are potentially dangerous if they are inhaled or swallowed, but external exposure generally does not pose a danger (U.S. NRC, 2014). [https://www.nrc.gov/about-nrc/radiation/health-effects/radiation-basics.html]. Alpha particles are normally unable to penetrate the epidermis of the skin, especially when it is a considerable distance from the target. However, when present in large amounts within a close distance, they are able to penetrate the epidermis and enter the body, thus becoming hazardous. [http://laboratorysafetyandmanagement.blogspot.co.za/2011/12/hazards-of-alpha-and-beta-particles-and.html]. Alpha particles can also enter the body via other routes, some of these including: oral ingestion; inhalation; and even absorption into the bloodstream. However, when inside the body, with no epidermis to stop their movements, they are able to travel just enough distances into tissues to cause considerable damage. [http://laboratorysafetyandmanagement.blogspot.co.za/2011/12/hazards-of-alpha-and-beta-particles-and.html]. This can lead to cancer, particularly lung cancer when alpha particles have been inhaled. However, tissues are not the only things that get damaged. If the alpha particles accumulate in an organ, they will also damage the cells of that particular organ resulting in organ damage. [http://laboratorysafetyandmanagement.blogspot.co.za/2011/12/hazards-of-alpha-and-beta-particles-and.html]

Beta particles, which are similar to electrons, are emitted from naturally occurring materials (such as strontium-90). Such beta emitters are used in medical applications, such as treating eye disease.

Humans can be exposed to beta particles in a number of ways. Potassium and carbon found naturally in our bodies are weak beta particle emitters. Direct exposure to beta particles, especially from concentrated emitters, can result in the burning of the skin or erythema. When inside the body, beta particles enter directly into the tissue, causing alteration of cell function, thereby affecting DNA in the cells. With a deeper penetration power, beta particles are able to cause more diverse cellular damage, and can be more hazardous than alpha particles (U.S. NRC, 2014). [https://www.nrc.gov/about-nrc/radiation/health-effects/radiation-basics.html]. Beta particles radiation can result in both acute and chronic health effects. Acute effects are presented when an individual is exposed to a concentrated source of beta particles. Chronic effects are more often observed with a long-term exposure to fairly low levels of beta particles. Exposure to beta particles often cause cancer, dependent on the location where the beta particles accumulate in the body. For example, accumulation of beta particles in the bone or teeth can lead to bone cancer. [http://laboratorysafetyandmanagement.blogspot.co.za/2011/12/hazards-of-alpha-and-beta-particles-and.html]. In general, beta particles are lighter than alpha particles, and they generally have a greater ability to penetrate other materials. As a result, these particles can travel a few feet in the air, and can penetrate skin. Nonetheless, a thin sheet of metal or plastic or a block of wood can stop beta particles (U.S. NRC, 2014).

The drinking water standards are set at 15 pCi/L for gross alpha, and 4 millirems per year (mrem/yr) for beta emitters (Water Research Foundation, 2014) [http://www.waterrf.org/resources/StateOfTheScienceReports/Radionuclides__StateOfTheScience.pdf]

30. IRON

Iron is the second most abundant metal in the earth's crust, of which it accounts for about 5%. Elemental iron is rarely found in nature, as the iron ions Fe2+ and Fe3+ readily combine with oxygen- and sulfur-containing compounds to form oxides, hydroxides, carbonates, and sulphides (Guidelines for drinking-water quality, 1996). Iron is most commonly found in nature in the form of its oxides. [http://www.who.int/water_sanitation_health/dwq/chemicals/iron.pdf]. Iron (as Fe2+) concentrations of 40 µg/litre can be detected by taste in distilled water. In a mineralized spring water with a total dissolved solids content of 500 mg/litre, the taste threshold value was 0.12 mg/litre (Guidelines for drinking-water quality, 1996). In well-water, iron concentrations below 0.3 mg/litre were characterized as unnoticeable, whereas levels of 0.3-3 mg/litre were found acceptable (E. Dahi, personal communication, 1991). [http://www.who.int/water_sanitation_health/dwq/chemicals/iron.pdf]. Iron is used as constructional material, inter alia for drinking-water pipes. Iron oxides are used as pigments in paints and plastics. Other compounds are used as food colours and for the treatment of iron deficiency in humans. Various iron salts are used as coagulants in water treatment (Guidelines for drinking-water quality, 1996). [http://www.who.int/water_sanitation_health/dwq/chemicals/iron.pdf]. The effects of toxic doses of iron in rats and mice include depression, rapid and shallow respiration, coma, convulsions, respiratory failure, and cardiac arrest (Guidelines for drinking-water quality, 1996). Iron dextran complex repeatedly injected subcutaneously or intramuscularly was considered by IARC to be carcinogenic to animals. [http://www.who.int/water_sanitation_health/dwq/chemicals/iron.pdf]

Iron (Ingestion)
UF Human: 1.5
RfD Human: 0.7 mg/kg/day (PPRTV, 2006) (Critical effect: Gastrointestinal toxicity)
[http://www.michigan.gov/documents/deq/deq-rrd-chem-IronDatasheet_527871_7.pdf]
NOAEL Human: No data available
LOAEL Human: 1 mg/kg/day (PPRTV, 2006)
BMDL Human:
Oral Slope Factor: N/A (MDEQ, 2015)

Iron (Inhalation)
RfC Human: No data available
NOAEL Human: No data available
LOAEL Human: No data available
BMCL Human: No data available
Slope Factor: No data available

31. LEAD

Lead is a very soft, dense, ductile metal. Lead is very stable and resistant to corrosion, although acidic water may leach out of pipes, fittings, and solder. It does not conduct electricity. Lead is an effective shield against radiation (CSEM, 2012). [https://www.youtube.com/watch?v=2P_2hUXtwEQ]. Lead exists in both organic and inorganic forms. Inorganic lead can be found in old paint. Lead also occurs in drinking water through leaching from lead-containing pipes, faucets, and solder, which in turn can be found in plumbing of older buildings. Even when lead is not intentionally used in a product, it may contaminate items such as food, water, or alcohol. Lead may contaminate food during, production and processing, packaging and storage (CSEM, 2012). [https://www.youtube.com/watch?v=2P_2hUXtwEQ]. Organic Lead is generally limited to an occupational context. However, organic lead can be more toxic than inorganic lead because the body more readily absorbs it. Potential exposures to organic lead should be taken very seriously (CSEM, 2012). [https://www.youtube.com/watch?v=2P_2hUXtwEQ]

Because of widespread human use of lead, lead is ubiquitous in the environment. These background levels vary depending on historic and ongoing uses in the area. The major exposure pathways for workers are inhalation and ingestion of lead-bearing dust and fumes. Workers in the lead smelting, refining, and manufacturing industries experience the highest and most prolonged occupational exposures to lead (ATSDR 2005). [https://www.youtube.com/watch?v=2P_2hUXtwEQ]. Absorbed lead that is not excreted is exchanged primarily among three compartments: blood, mineralizing tissues (bones and teeth), which typically contain the vast majority of the lead body burden and soft tissue (liver, kidneys, lungs, brain, spleen, muscles, and heart). [https://www.youtube.com/watch?v=2P_2hUXtwEQ]

Effects in children generally occur at lower blood lead levels (BLL) than in adults. The developing nervous system of a child can be affected adversely at BLLs of less than 10 µg/dL. It is often impossible to determine these effects upon clinical examination. There is a wide range of neurological effects associated with lead exposure, some of which may likely be irreversible. Lead exposure can lead to renal effects such as Fanconi-like syndromes, chronic nephropathy, and gout. Most lead-associated renal effects or disease are a result of ongoing chronic or present high acute exposure or can be a latent effect of chronic past lead exposure. Lead inhibits several enzymes critical to the synthesis of haem, causing a decrease in blood haemoglobin.

Today, lead exposure in children only rarely results in anaemia. Lead’s impairment of haem synthesis can affect other haem dependent processes in the body outside of the hematopoietic system. Lead interferes with a hormonal form of vitamin D, which affects multiple processes in the body, including cell maturation and skeletal growth. Health effects associated with exposure to inorganic lead and compounds include, but are not limited to, neurotoxicity, developmental delays, hypertension, impaired hearing acuity, impaired haemoglobin synthesis, and male reproductive impairment. Importantly, many of lead's health effects may occur without overt signs of toxicity. Maternal blood lead, from exogenous and endogenous sources, can cross the placenta and put the foetus at risk. Other potential health effects of lead are currently being studied (CSEM, 2012). [https://www.youtube.com/watch?v=2P_2hUXtwEQ]

Lead (Ingestion)

RfD UF: No data

RfD Human + Animal: Not available (IRIS, 2004)

[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0277_summary.pdf]

NOAEL Human + Animal: Not available (IRIS, 2004)

LOAEL Human + Animal: Not available (IRIS, 2004)

BMDL Human + Rats: (IRIS, 2004)

Oral Slope Factor: N/A (IRIS, 2004)

Lead (Inhalation)

RfC UF: No data

RfC Human + Animal: Not available (IRIS, 2004)

NOAEL Human + Animal: Not available (IRIS, 2004)

LOAEL Human + Animal: Not available (IRIS, 2004)

BMCL Human + Rats: Not available (IRIS, 2004)

Oral Slope Factor: N/A (IRIS, 2004)

32. MAGNESIUM

Magnesium is the eighth most abundant natural element. It makes up 2.5 percent of the Earth's crust and is commonly found in such minerals as magnesite, dolomite, olivine, serpentine, talc, and asbestos. It is present in all natural waters and is a major contributor to water hardness. Ferromagnesian mineral igneous rocks and magnesium carbonates in sedimentary rocks are generally considered to be the principal sources of magnesium in natural waters. Aluminum-magnesium alloys are used in beverage cans, pressure die-cast products, electrical equipment, portable tools, sports equipment, and many other products. Magnesium is used as a deoxidizing and desulphurizing agent in the ferrous metal industry and as a reducing agent in the production of titanium, zirconium, and other reactive metals. Pure magnesium metal is used to protect steel structures from corrosion and has many applications in the chemical industry (Health Canada, 1978). [https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-supporting-documents-magnesium.html]. The estimated daily intake of magnesium for Canadians consuming an average diet is 205 mg for children and between 200 and 300 mg for adults (250 mg/L for adults according to South African guidelines). The intake of magnesium from drinking water varies widely, depending on the hardness of the water. Daily intake from ingesting 1.5 L of water daily would range from 1.5 mg (soft water, 1 mg/L magnesium) to 37.5 mg (hard water, 25 mg/L magnesium). Magnesium in air is not considered to contribute significantly to the total intake of this element (Health Canada, 1978). [https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-supporting-documents-magnesium.html]

The most readily observable adverse effect of magnesium in drinking water is the laxative effect, particularly with magnesium sulphate at concentrations above 700 mg/L. The South African standard is above 400 mg/L (South African Domestic Water Use, 2001). However, the human body can adapt to this laxative effect with time. Toxicity has been reported in the elderly as a result of the extensive use of certain laxatives (magnesium sulphate) and antacids (magnesium hydroxides). This population, however, may also have a reduced renal clearance. At serum concentrations of 5 to 10 meq/L (6 to 12 mg/dL), changes in heartbeat may occur. Skeletal muscle paralysis, respiratory depression, coma, and death occur at plasma concentrations of 15 meq/L (18 mg/dL) (Health Canada, 1978). [https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-supporting-documents-magnesium.html]

Magnesium is one of the major contributors to water hardness, which is discussed in a separate review. Magnesium may also contribute undesirable tastes to drinking water. The taste threshold has been reported to be 100 mg/L for sensitive individuals (between 70-100 mg/L according to South African Standards) and about 500 mg/L for the average person. These levels are well above the magnesium concentrations encountered in most Canadian drinking waters (Health Canada, 1978). [https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-supporting-documents-magnesium.html]

33. MANCOZEB

Mancozeb is registered as a general use pesticide by the U.S. Environmental Protection Agency (EPA) (Cornell University, 1993). [http://pmep.cce.cornell.edu/profiles/extoxnet/haloxyfop-methylparathion/mancozeb-ext.html]. Mancozeb is used to protect many fruit, vegetable, nut and field crops against a wide spectrum of diseases, including potato blight, leaf spot, scab (on apples and pears) and rust (on roses). It is also used for seed treatment of cotton, potatoes, corn, safflower, sorghum, peanuts, tomatoes, flax and cereal grains. Mancozeb is not taken up from the soil by plants. It is a combination of two other chemicals of this class, maneb and zineb. Mancozeb is available as dusts, liquids, water dispersible granules, as wettable powders, and as ready-to-use (R-T-U) formulations (Cornell University, 1993).

Mancozeb has a very low acute toxicity to mammals. No toxicological effects were observed in a long term study with rats fed doses of 5 mg/kg. The major routes of exposure to mancozeb are through the skin or from inhalation. In spray or dust forms, the EBDCs are moderately irritating to the skin and respiratory mucous membranes. Symptoms of poisoning from this class of chemicals include itching, scratchy throat, sneezing, coughing, inflammation of the nose or throat, and bronchitis. There is no evidence of 'neurotoxicity,' nerve tissue destruction or behaviour change, from the EBDCs. However, dithiocarbamates are partially chemically broken down, or metabolized, to carbon disulfide, a neurotoxin capable of damaging nerve tissue (Cornell University, 1993). The thyroid is the target organ for mancozeb. Thyroid effects were observed in multiple studies across species. Thyroid toxicity was manifested as alterations in thyroid hormones, increased thyroid weight, and microscopic thyroid lesions, and thyroid tumours (EPA, 2005). [https://nepis.epa.gov/Exe/tiff2png.cgi/P100BIFA.PNG?-r+75+-g+7+D%3A%5CZYFILES%5CINDEX%20DATA%5C00THRU05%5CTIFF%5C00001752%5CP100BIFA.TIF]

The EPA's cancer concern for mancozeb is limited to risk from the metabolite ethylenethiourea (ETU). The estimated lifetime dietary exposure to ETU from all sources correspond to a cancer risk estimate of 2×10^{-6}, which is within the negligible range of 10^{-6}, and not to be considered of concern (EPA Reregistration Eligibility Decision for Mancozeb, September, 2005) .

[http://msdssearch.dow.com/PublishedLiteratureDOWCOM/dh_0417/0901b80380417adc.pdf?filepath=productsafety/pdfs/noreg/233-00379.pdf&fromPage=GetDoc]. The drinking water exposure assessment for mancozeb addresses concentrations of ETU only, since mancozeb is not expected to remain in drinking water long enough to reach a location that would supply water for human consumption, whether from surface or groundwater sources. Estimated concentrations of ETU, for both surface and ground water sources of drinking water, are low and not of concern (EPA, 2005).

34. MANGANESE

Manganese is a naturally occurring substance found in many types of rocks and soil. Pure manganese is a silver-coloured metal; however, it does not occur in the environment as a pure metal. Rather, it occurs combined with other substances such as oxygen, sulphur, and chlorine. Manganese is a trace element and is necessary for good health (ATSDR, 2012). [https://www.atsdr.cdc.gov/ToxProfiles/tp151-c1-b.pdf]. Manganese is used principally in steel production to improve hardness, stiffness, and strength. It is used in carbon steel, stainless steel, high temperature steel, and tool steel, along with cast iron and super alloys. Manganese occurs naturally in most foods and may be added to food or products made available in nutritional supplements. Manganese is also used in a wide variety of other products, including: fireworks, dry-cell batteries, fertilizer, paints, medical imaging agent and cosmetics. It may also be used as an additive in gasoline to improve the octane rating of the gas (ATSDR, 2012). Small amounts of manganese are used in a pharmaceutical product called mangafodipir trisodium (MnDPDP) to improve lesion detection in magnetic resonance imaging of body organs.

The primary way you can be exposed to manganese is by eating food or source of manganese-containing nutritional supplements. Vegetarians who consume foods rich in manganese such as grains, beans and nuts, as well as heavy exposure tea drinkers, may have a higher intake of manganese than the average person. Certain occupations like welding or working in a factory where steel is made may increase your chances of being exposed to high levels of manganese. Because manganese is a natural component of the environment, you are always exposed to low levels of it in water, air, soil, and food. Manganese is routinely contained in groundwater, drinking water and soil at low levels. Drinking water containing manganese or swimming or bathing in water containing manganese may expose you to low levels of this chemical (ATSDR, 2012). Air also contains low levels of manganese, and breathing air may expose you to it. Releases of manganese into the air occur from: industries using or manufacturing products containing manganese, mining activities, and automobile exhaust. [https://www.atsdr.cdc.gov/ToxProfiles/tp151-c1-b.pdf]

The most common health problems in workers inhaling high levels of manganese involve the nervous system. These health effects include behavioural changes and other nervous system effects, which include movements

that may become slow and clumsy. This combination of symptoms when sufficiently severe is referred to as “manganism.” Other less severe nervous system effects such as slowed hand movements have been observed in some workers exposed to lower concentrations in the work place. The inhalation of a large quantity of dust or fumes containing manganese may cause irritation of the lungs which could lead to pneumonia. Loss of sex drive and sperm damage has also been observed in men exposed to high levels of manganese in workplace air (ATSDR, 2012). [https://www.atsdr.cdc.gov/ToxProfiles/tp151-c1-b.pdf]

Studies in children have suggested that extremely high levels of manganese exposure may produce undesirable effects on brain development, including changes in behaviour and decreases in the ability to learn and remember. In some cases, these same manganese exposure levels have been suspected of causing severe symptoms of manganism disease (including difficulty with speech and walking). We do not know for certain that these changes were caused by manganese alone. We do not know if these changes are temporary or permanent. We do not know whether children are more sensitive than adults to the effects of manganese, but there is some indication from experiments in laboratory animals that they may be (ATSDR. 2012). [https://www.atsdr.cdc.gov/ToxProfiles/tp151-c1-b.pdf]

A study by Kondakis et al. (1989) raised some concern for possible adverse health effects associated with a lifetime consumption of drinking water containing about 2 mg/L of manganese (IRIS, 1995) [https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0373_summary.pdf]. The EPA has established that exposure to manganese in drinking water at concentrations of 1 mg/L for 1 or 10 days is not expected to cause any adverse effects in a child. The EPA has established that lifetime exposure to 0.3 mg/L manganese is not expected to cause any adverse effects. Bottled water The FDA has established that the manganese concentration in bottled drinking water should not exceed 0.05 mg/L (ATSDR, 2012).

In animals ingesting manganese has been shown to cross the blood-brain barrier and a limited amount of manganese is also able to cross the placenta during pregnancy, enabling it to reach a developing foetus. Nervous system disturbances have been observed in animals after very high oral doses of manganese, including changes in behaviour. Sperm damage and adverse changes in male reproductive performance were observed in laboratory animals fed high levels of manganese. Impairments in fertility were observed in female rodents provided with oral manganese before they became pregnant. Illnesses involving the kidneys and urinary tract have been observed in laboratory rats fed very high levels of manganese. These illnesses included inflammation of the kidneys and kidney stone formation (ATSDR, 2012). [https://www.atsdr.cdc.gov/ToxProfiles/tp151-c1-b.pdf]. The EPA concluded that existing scientific information cannot determine whether or not excess manganese can cause cancer (ATSDR, 2012).

Manganese (Ingestion)
RfD UF: 1
RfD Human: 1.4E-1 mg/kg/d (food) (NRC, 1989); (Freeland-Graves et al., 1987); (WHO, 1973)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0373_summary.pdf]
5ug/kg/d (water) (UNITED STATES ENVIRONMENTAL PROTECTION AGENCY, 2009)
[https://www3.epa.gov/region9/water/drinking/files/dwshat-v09.pdf]

NOAEL Human (food): 0.14 mg/kg/day (NRC, 1989); (Freeland-Graves et al., 1987); (WHO, 1973)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0373_summary.pdf]

LOAEL Human: None
BMDL Human: None
Oral Slope Factor: N/A

Manganese (Inhalation)
RfC UF: 1000
RfC Human: 5.00E-5 mg/m^3 (Roels et al., 1992)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0373_summary.pdf]

NOAEL Human: None
LOAEL Human: 0.15 mg/m^3 (Impairment of neurobehavioral function)
0.05 mg/m^3 (HEC) (Occupational exposure to manganese oxides and salts)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0373_summary.pdf]
BMCL Human: None
Slope Factor: None

35. MERCURY

Mercury is used in the electrolytic production of chlorine, in electrical appliances, in dental amalgams and as a raw material for various mercury compounds. Methylation of inorganic mercury has been shown to occur in fresh water and in seawater, although almost all mercury in uncontaminated drinking-water is thought to be in the form of Hg2+. Thus, it is unlikely that there is any direct risk of the intake of organic mercury compounds, especially of alkylmercurials, as a result of the ingestion of drinking water. However, there is a possibility that methylmercury will be converted into inorganic mercury. Food is the main source of mercury in non-occupationally exposed populations; the mean dietary intake of mercury in various countries ranges from 2 to 20 mg/day per person [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1]. Inorganic mercury (metallic mercury and inorganic mercury compounds) enters the air from mining ore deposits, burning coal and waste, and from manufacturing plants. It enters the water or soil from natural deposits, disposal of wastes, and volcanic activity.

Methylmercury may be formed in water and soil by small organisms called bacteria. Methylmercury builds up in the tissues of fish. Larger and older fish tend to have the highest levels of mercury. Oral exposure to mercury such as the ingestion of fish or shellfish contaminated with methylmercury and ingesting contaminated water. Breathing vapours in air from spills, incinerators, and industries that burn mercury-containing fossil fuels results inhalation of mercury. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing foetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems. Short-term exposure to high levels of metallic mercury vapours may cause effects including lung damage, nausea, vomiting, diarrhoea, increases in blood pressure or heart rate, skin rashes, and eye irritation [https://www.atsdr.cdc.gov/toxfaqs/tfacts46.pdf]. The toxic effects of inorganic mercury compounds are seen mainly in the kidney in both humans and laboratory animals following short- and long-term exposure. In rats, effects include increased absolute and relative kidney weights, tubular necrosis, proteinuria and hypoalbuminaemia. In humans, acute oral poisoning results primarily in haemorrhagic gastritis and colitis; the ultimate damage is to the kidney [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1].

Mercury (Ingestion)
RfD UF: No data
RfD Rats: No data
NOAEL Human: None
LOAEL Human: No data
BMDL Human: No data
Oral Slope Factor: No data

Mercury (Inhalation)
RfC UF: 30
RfC Human: 3E-04 mg/cu.m
NOAEL Human: None
LOAEL Human: 0.025 mg/cu.m
BMCL Human: No data

36. MICROCYSTIN

Microcystin is a toxin produced naturally by cyanobacteria, also known as blue-green algae (MDH, 2015). [http://www.health.state.mn.us/divs/eh/risk/guidance/gw/mclrinfo.pdf]. Based on available data, Minnesota Department of Health has derived a guidance value of 0.1 ppb for microcystin-LR in drinking water. A person drinking water at or below this level, whether briefly, occasionally, or daily for a lifetime, would have little or no risk of any health effects from microcystin (MDH, 2015). Microcystin is highly toxic, and even drinking a small amount could be harmful to the liver. Microcystin is considered to help stimulate the growth of cancerous tumours in the liver and colon. You may be exposed to low levels of microcystins through recreational activities such as swimming or boating. Exposure can occur through skin contact, swallowing lake water, or breathing water spray. Your exposure will depend on whether there is an active algal bloom in the water. Children are more likely than adults to be exposed through these routes (MDH, 2015).
[http://www.health.state.mn.us/divs/eh/risk/guidance/gw/mclrinfo.pdf]

Through the recreational use of contaminated water, cyanobacterial blooms of Microcystis, Anabaena, and others have been linked to incidence of human illness in many countries, but no fatalities have been reported (Lambert et al., 1994b). In Canada, human illnesses have been reported in Saskatchewan, with symptoms including stomach cramps, vomiting, diarrhoea, fever, headache, pains in muscles and joints, and weakness (Dillenberg & Dehnel, 1960). Similar symptoms as well as skin, eye, and throat irritation and allergic responses to cyanobacterial toxins in water have also been reported in other countries (Ressom et al., 1994). The reported instances of illnesses are few, but, because they are difficult to diagnose, such illnesses may in fact be more common than has been reported (Carmichael & Falconer, 1993).
[http://www.who.int/water_sanitation_health/dwq/chemicals/cyanobactoxins.pdf]

Microcystin (Ingestion)
RfD UF: No data
NOAEL Mice: 40 ug/kg/d (Fawell et al., 1994)
[http://www.who.int/water_sanitation_health/dwq/chemicals/cyanobactoxins.pdf]

Microcystin (Inhalation)
RfC UF: No data
LOAEL Pigs: 280 ug/kg/d (Falconer et al., 1994)
[http://www.who.int/water_sanitation_health/dwq/chemicals/cyanobactoxins.pdf]

37. MONOCHLORAMINE

Chloramine is a disinfectant used to treat drinking water. It is formed by mixing chlorine with ammonia. Although it is a weaker disinfectant than chlorine, it is more stable and extends disinfectant benefits throughout a water utility's distribution system (a system of pipes water is delivered to homes through). Some water systems use chloramine as a secondary disinfectant to maintain a disinfectant residual throughout the distribution system so that drinking water remains safe as it travels from the treatment facility to the customer (US EPA, 2015). [http://www.mawc.org/sites/default/files/page_attachments/pdf/Chloramine_General_Facts.pdf].

Chloraminated water that meets the EPA standard is safe for drinking and other general household activities such as bathing, cooking, laundry, and cleaning. The water can also be used for gardening (the water is safe for plants) and for watering lawns with no adverse effects (US EPA, 2015). EPA has a standard (the Maximum Residual Disinfectant Level or MRDL) and a health goal (the Maximum Residual Disinfectant Level Goal or MRDLG) for chloramine. The enforceable MRDL is the highest level of a disinfectant allowed in drinking water. The MRDLG is the level of a drinking water disinfectant, below which there is no known or expected risk to health. EPA sets the standard as close to the health goal as feasible, while considering technology, treatment, cost, and risk trade-offs. In the case for chloramine, the MRDL and MRDLG are the same. Maximum Residual Disinfectant Level Goal (MRDLG) 4 milligrams per litre (mg/L) [4 parts per million (ppm)] measured as chlorine as an annual average. Maximum Residual Disinfectant Level (MRDL) 4.0 mg/L (4.0 ppm) measured as chlorine as an annual average (US EPA, 2015). Drinking water chloramine levels that meet the EPA standard are associated with minimal to no risk and should be considered safe. Some people who use water containing chloramine well in excess of the Maximum Residual Disinfectant Level (MRDL) could experience irritating effects to their eyes and nose. Some people who drink water containing chloramine well in excess of the MRDL could experience stomach discomfort or anaemia (US EPA, 2015).

Monochloramine (Ingestion)

RfD UF: 100

RfD Rats: 1.00E-1 mg/kg/day (NTP, 1992)

NOAEL Rats: 9.5 mg/kg/d (NTP, 1992)

LOAEL Rats: No Data

BMDL Rats: No Data

Oral Slope Factor: N/A

Monochloramine (Inhalation)

RfC UF: No data

RfC Human: No Data

NOAEL Human: No Data

LOAEL Human: No Data

BMCL Human: No Data

Slope Factor: No Data

38. NICKEL

Pure nickel is a hard, silvery-white metal, which has properties that make it very desirable for combining with other metals to form mixtures called alloys. Some of the metals that nickel can be alloyed with are iron, copper, chromium, and zinc. These alloys are used in making metal coins and jewellery and in industry for making items such as valves and heat exchangers. Most nickel is used to make stainless steel. There are also compounds consisting of nickel combined with many other elements, including chlorine, sulphur, and oxygen. Many of these nickel compounds are water soluble (dissolve fairly easily in water) and have a characteristic green colour. Nickel and its compounds have no characteristic odour or taste. Nickel compounds are used for nickel plating, to colour ceramics, to make some batteries, and as substances known as catalysts that increase the rate of chemical reactions (ATSDR, 2005).[https://www.atsdr.cdc.gov/ToxProfiles/tp15-c1-b.pdf]

Food is the major source of exposure to nickel. You may also be exposed to nickel by breathing air, drinking water, or smoking tobacco containing nickel. Skin contact with soil, bath or shower water, or metals containing nickel, as well as, metals plated with nickel can also result in exposure. Stainless steel and coins contain nickel. Some jewellery is plated with nickel or made from nickel alloys (ATSDR, 2005). Much of the nickel found in air, soil, sediment, and rock is so strongly attached to dust and soil particles or embedded in minerals that it is not readily taken up by plants and animals and, therefore, cannot easily affect your health. In water and waste water, nickel can exist either dissolved in water or attached to material suspended in water (ATSDR, 2005). [https://www.atsdr.cdc.gov/ToxProfiles/tp15-c1-b.pdf]

The most common harmful health effect of nickel in humans is an allergic reaction. The most common reaction is a skin rash at the site of contact. In some sensitized people, dermatitis (a type of skin rash) may develop in an area of the skin that is away from the site of contact (ATSDR, 2005). The most serious harmful health effects from exposure to nickel, such as chronic bronchitis, reduced lung function, and cancer of the lung and nasal sinus, have occurred in people who have breathed dust containing certain nickel compounds while working in nickel refineries or nickel processing plants. Lung and nasal sinus cancers occurred in workers who were exposed to more than 10 mg nickel/m^3 as nickel compounds that were hard to dissolve (such as nickel subsulfide). Exposure to high levels of nickel compounds that dissolve easily in water (soluble) may also result in cancer when nickel compounds that are hard to dissolve (less soluble) are present, or when other chemicals that can produce cancer are present (ATSDR, 2005). EPA recommends that drinking water levels for nickel should not be more than 0.1 mg per litre. [https://www.atsdr.cdc.gov/ToxProfiles/tp15-c1-b.pdf]

The EPA, U.S. Department of Health and Human Services and The International Agency for Research on Cancer has determined that nickel refinery dust and nickel subsulfide are human carcinogens. These cancer classifications were based on studies of nickel workers and laboratory animals. [https://www.atsdr.cdc.gov/ToxProfiles/tp15-c1-b.pdf]

Nickel (Ingestion)
RfD UF: 300
RfD Human: 2.00E-2
NOAEL Human: 5 mg/kg/d
LOAEL Human: 50 mg/kg/d
BMDL Rats: N/A
Oral Slope Factor: N/A

Nickel (Inhalation)
RfC UF: No data
RfC Human: No Data
NOAEL Human : 0.0052 mg/m^3 (ATSDR, 2005)
LOAEL Human: No Data
BMCL Human: No Data
Slope Factor: N/A

39. NITRATE

Nitrate is a naturally occurring ionic species that are part of the earth's nitrogen cycle. Nitrate typically exist in the environment in highly water-soluble forms, in association with other ions such as sodium and potassium. Salts completely dissociate in aqueous environments (ATSDR, 2015). [https://www.atsdr.cdc.gov/ToxProfiles/tp204-c1-b.pdf]. Nitrate is generally stable in the environment; however, it may be reduced to nitrite through biological processes involving plants, microbes, etc. In nature, plants utilize nitrate as an essential nutrient. In commerce, the majority of nitrate is used in inorganic fertilizers. Additional uses of commercial nitrate and nitrite include food preservation and the production of munitions and explosives. The major source of overexposure of the general population to nitrate is via ingestion of water and foods that contain nitrate. Most people are not exposed to levels of nitrate and/or nitrite that would cause adverse health effects. Young infants (<6 months of age) appeared to be particularly sensitive to the effects of nitrite on haemoglobin after consuming formula prepared with drinking water that contained nitrate at levels higher than recommended limits; some of these infants died. The cause of methemoglobinemia (a change to haemoglobin that decreases the ability to transport oxygen to tissues) in many of these infants may have been gastroenteritis from bacteria or viruses in the drinking water or from other sources not related to nitrate. Some children and adults who ate food or drank fluids that contained unusually high levels of nitrite experienced decreases in blood pressure, increased heart rate, reduced ability of the blood to carry oxygen to tissues, headaches, abdominal cramps, vomiting, and even death (ATSDR, 2015). [https://www.atsdr.cdc.gov/ToxProfiles/tp204-c1-b.pdf]. The International Agency for Research on Cancer (IARC) determined that there is inadequate evidence for the carcinogenicity of nitrate in food or drinking water and limited evidence for the carcinogenicity of nitrite in food (based on association with increased incidence of stomach cancer) (ATSDR, 2015). [https://www.atsdr.cdc.gov/ToxProfiles/tp204-c1-b.pdf]

The EPA lists maximum contaminant levels (MCL) and maximum contaminant level goals (MCLG) of 10 mg/L (or ppm) for nitrate (as nitrate-nitrogen; ~44 mg nitrate/L) in the 2012 Edition of the Drinking Water Standards and Health Advisories. The FDA lists 10 mg/L nitrate (as nitrogen; ~44 mg nitrate/L), and 10 mg/L total nitrate and nitrite (as nitrogen) as allowable levels in bottled water. OSHA has not set a legal limit for nitrate or nitrite in air. [https://www.atsdr.cdc.gov/ToxProfiles/tp204-c1-b.pdf]

Nitrate (Ingestion)
RfD UF: 1
RfD Humans: 1.60E+0 mg/kg/d (Bosch et al., 1950; Walton, 1951)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0076_summary.pdf]
LOAEL Human: 1.8-3.2 mg/kg/d (Bosch et al., 1950; Walton, 1951)
NOAEL Human: 1.6 mg/kg/d (Bosch et al., 1950; Walton, 1951)
BMCL: Not evaluated (IRIS, 1991)
Risk Factor: N/A

Nitrate (Inhalation)
RfC UF: No data
RfC Humans: Not evaluated (IRIS, 1991)
LOAEL Human: Not evaluated (IRIS, 1991)
NOAEL Human: Not evaluated (IRIS, 1991)

40. NITRITE AS N

Nitrite (NO_2) is not usually present in significant concentrations except in a reducing environment, since nitrate is the most stable oxidation state. It can be formed by the microbial reduction of nitrate. Nitrite can also be formed chemically in distribution pipes by *Nitrosomonas* bacteria during stagnation of nitrate-containing and oxygen-poor drinking-water in galvanized steel pipes or if chloramination is used to provide a residual disinfectant. In general, the most important source of human exposure to nitrite is through vegetables and through meat in the diet (nitrite is used as a preservative in many cured meats) and ingestion of water containing nitrate/nitrite. In some circumstances, however, drinking-water can make a significant contribution to nitrate and, occasionally, nitrite intake. In the case of bottle-fed infants, drinking-water can be the major external source of exposure to nitrate and nitrite. In humans, methemoglobinemia forms as a consequence of the reaction of nitrite with haemoglobin in the red blood cells to form methaemoglobin, which binds oxygen tightly and does not release it, so blocking oxygen transport. Although most absorbed nitrite is oxidized to nitrate in the blood, residual nitrite can react with haemoglobin. High levels of methaemoglobin (greater than 10%) formation can give rise to cyanosis, referred to as blue-baby syndrome [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1].

Nitrate and nitrite could enter your body from the air you breathe; however, you are not likely to be exposed to amounts of nitrate or nitrite in the air that might cause adverse health effects [https://www.atsdr.cdc.gov/ToxProfiles/tp204-c1-b.pdf].

Nitrite (Ingestion)
RfD UF: 1 (Walton, 1951)
RfD Human: 1E-1 mg/kg-day (Walton, 1951)
NOAEL Human: 1.0 mg/kg/day (Walton, 1951)
LOAEL Human: 11-20 ppm (Walton, 1951)
BMDL Human: No data
Oral Slope Factor: No data

Nitrite (Inhalation)
RfC UF: No data
RfC Human: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data

41. pH

Although pH usually has no direct impact on consumers, it is one of the most important operational water quality parameters. Careful attention to pH control is necessary at all stages of water treatment to ensure satisfactory water clarification and disinfection. For effective disinfection with chlorine, the pH should preferably be less than 8; however, lower-pH water (approximately pH 7 or less) is more likely to be corrosive. The pH of the water entering the distribution system must be controlled to minimize the corrosion of water mains and pipes in household water systems. Alkalinity and calcium management also contribute to the stability of water and control its aggressiveness to pipes and appliances. Failure to minimize corrosion can result in the contamination of drinking-water and in adverse effects on its taste and appearance. The optimum pH required will vary in different supplies according to the composition of the water and the nature of the construction materials used in the distribution system, but it is usually in the range 6.5-8.5. Extreme values of pH can result from accidental spills, treatment breakdowns and insufficiently cured cement mortar pipe linings or cement mortar linings applied when the alkalinity of the water is low. No health-based guideline value has been proposed for pH. [http://apps.who.int/iris/bitstream/10665/44584/1/9789241548151_eng.pdf]

42. PHENOLS

Phenol is a solid at room temperature and normal atmospheric pressure consisting of white crystals that turn pink or red when exposed to air and light. It has a burning taste and a distinctive odour. The compound has limited solubility in water (6. 7 g/100 ml) and is soluble in most organic solvents. Phenol is mainly used for the manufacture of phenolic resins, bisphenol A, and caprolactam. Other products are alkylphenols, xylenol, cresol, and adipic acid. Minor uses include the production of germicidal paints, pharmaceutical products, dyes, and indicators, and the use of phenols as a laboratory reagent, a slimicide, and a general disinfectant. [http://apps.who.int/iris/bitstream/handle/10665/39958/9241510889-eng.pdf?sequence=1]. Phenol is considered to be quite toxic to humans via oral exposure. Exposure of the general population to phenol mainly occurs by inhalation. Minor oral exposure may arise through the consumption of smoked food or drinking-water.

Gastrointestinal irritation was reported following ingestion of phenol. Painless blanching, or effects ranging from erythema to corrosion and deep necrosis, occurred following dermal exposure. Main systemic effects included cardiac dysrhythmias, respiratory distress, metabolic acidosis, renal failure, dark urine, methaemoglobinaemia, neurological effects, cardiovascular shock, coma, and death. The lowest reported fatal dose was 4.8 g by ingestion; death occurred within 19 minutes. Symptoms associated with inhalation of phenol included anorexia, weight loss, headache, vertigo, salivation, acidosis, and dark urine. No cases of death following this type of exposure have been reported. Phenol is highly irritating to the skin, eyes, and mucous membranes in humans after acute (short-term) inhalation or dermal exposures. [http://apps.who.int/iris/bitstream/handle/10665/39958/9241510889-eng.pdf?sequence=1].

Phenols (Ingestion)
RfD UF: 300
RfD Rat: 3E-1 mg/kg-day (Argus Research Laboratories, 1997)
NOAEL Rat: 12-40 mg/kg (http://apps.who.int/iris/bitstream/handle/10665/39958/9241510889-eng.pdf?sequence=1)
LOAEL Rat: No data
BMDL Rat: 93 mg/kg-day (Argus Research Laboratories, 1997)
Oral Slope Factor: No data

Phenols (Inhalation)
RfC UF: No data
RfC Human: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data

43. POTASSIUM

Potassium is an alkali metal which reacts violently with water to form positively-charged potassium ions. Potassium always occurs in water in association with anions, usually chloride, but can also occur with sulphate, bicarbonate, or nitrate. Potassium is the main intracellular cation in living organisms and is an essential dietary element (South African Domestic Water Use, 2001). [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

Potassium salts are highly soluble in water and precipitation does not occur on evaporation until very high concentrations are reached and potassium therefore has a strong tendency to remain in water. Since sodium salts are generally cheaper than the corresponding potassium salts, industries predominantly use sodium rather than potassium salts. Therefore, sodium is usually found at higher concentrations than potassium in wastes and brines. High concentrations of potassium may occur in runoff from irrigated lands, and from fertilizer production and domestic wastes (South African Domestic Water Use, 2001)
[http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

Potassium is an important intracellular cation and the total dietary intake ranges from 1.6-4.7 g/day, depending on age (IOM, 2004) (South African Domestic Water Use, 2001). At high concentrations (>400 mg/L) potassium imparts a bitter taste to water, and consumption can induce nausea and vomiting. Consequently, excessive concentrations of potassium salts ingested orally are relatively harmless to healthy adults, since the protective vomiting reflex rids the system of dangerous excesses. Healthy humans are relatively insensitive to any harmful effects caused by potassium, but electrolyte disturbances can occur, particularly in infants or in patients with kidney pathologies on a potassium-restricted diet (South African Domestic Water Use, 2001).
[http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

Although concentrations of potassium normally found in drinking-water are generally low and do not pose health concerns, the high solubility of potassium chloride and its use in treatment devices such as water softeners can lead to significantly increased exposure. In the United Kingdom, a survey carried out for the Regional Heart Study (Powell, Bailey & Jolly, 1987) found a mean potassium concentration of 2.5 mg/l in drinking-water. Data from Canada indicate that average concentrations of potassium in raw and treated drinking water in different areas vary between <1 and 8 mg/L. However, concentrations ranged up to 51 mg/l in Saskatchewan, which is the largest production area for potassium chloride in Canada (Health Canada, 2008)
[http://www.who.int/water_sanitation_health/water-quality/guidelines/chemicals/potassium-background.pdf?ua=1]

44. SELENIUM

Selenium is present in the Earth's crust, often in association with sulfur-containing minerals. Selenium is an essential trace element, and foodstuffs such as cereals, meat and fish are the principal source of selenium in the general population. Levels in food also vary greatly according to geographical area of production. [http://www.who.int/water_sanitation_health/dwq/GDW12rev1and2.pdf?ua=1].

People receive the majority of their daily intake of selenium from eating food, and to a lesser extent, from water intake. People could be exposed to too much selenium if they eat a lot of locally grown grains and vegetables or animal products that have built up high levels of selenium. Humans are normally not exposed to large amounts of selenium in the air, unless selenium dust or volatile selenium compounds are formed in their workplace. Selenium commonly enters the air from burning coal or oil. Occupations in which humans may be exposed to selenium in the air are the metal industries, selenium-recovery processes, paint manufacturing, and special trades [https://www.atsdr.cdc.gov/toxprofiles/tp92.pdf].

In humans, the toxic effects of long-term selenium exposure are manifested in nails, hair and liver. Effects on synthesis of a liver protein were also seen in a small group of patients with rheumatoid arthritis given selenium at a rate of 0.25 mg/day in addition to selenium from food. No clinical or biochemical signs of selenium toxicity were reported in a group of 142 persons with a mean daily intake of 0.24 mg (maximum 0.72 mg) from food. Acute oral doses of selenite and other selenium compounds cause symptoms such as nausea, diarrhoea, abdominal pain, chills, tremor, numbness in limbs, irregular menstrual bleeding, and marked hair loss. High dietary intakes of selenium have been investigated in selenium-rich areas of South Dakota, USA. Symptoms in people with high urinary selenium levels included gastrointestinal disturbances, discoloration of the skin, and decayed teeth [http://apps.who.int/iris/bitstream/handle/10665/75424/WHO_SDE_WSH_03.04_13_eng.pdf;jsessionid=A13D58523F63568B2802F7FBCDBD7767?sequence=1]

Selenium (Ingestion)
RfD UF: 3 (Yang et al., 1989b)
RfD Human: 5E-3 mg/kg/day (Yang et al., 1989b)
NOAEL Human: 0.015 mg/kg/day (Yang et al., 1989b)
LOAEL Human: 0.023 mg/kg/day
BMDL Human: No data
Oral Slope Factor: No data

Selenium (Inhalation)
RfC UF: No data
RfC Human: No data
NOAEL Human: No data
LOAEL Human: No data
BMCL Human: No data

45. SODIUM

Sodium is an alkali metal which reacts with water to form highly soluble, positively-charged sodium ions. It is an essential dietary element important for the electrolyte balance and the maintenance of many essential physiological functions. Sodium is present in all food to varying degrees (South African Domestic Water Use, 2001). [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

Sodium is ubiquitous in the environment and usually occurs as sodium chloride, but sometimes as sodium sulphate, bicarbonate or even nitrate. Sodium is found as solid sodium chloride (rock salt) in areas where geological deposits occur. The levels of sodium in surface waters are generally low in areas of high rainfall and high in arid areas with low mean annual precipitation. Sodium is highly soluble in water and does not precipitate when water evaporates, unless saturation occurs. Hence, water in arid areas often contains elevated concentrations of sodium. High concentrations also occur in sea water, at approximately 11 g/L. Industrial wastes, especially processes that give rise to brines, contain elevated concentrations of sodium. Sodium is also present at high concentrations in domestic waste water; this is in part due to the addition of table salt (sodium chloride) to foods. Furthermore, with re-use or recycling of water, the sodium concentration will tend to increase with each cycle or addition of sodium to the water. For this reason, sodium concentrations are elevated in runoffs or leachates from irrigated soils (South African Domestic Water Use, 2001).
[http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

The taste threshold for sodium in water varies from 135-200 mg/L, depending on the associated anion. The common ones include chloride, sulphate, nitrate, bicarbonate and carbonate. Sodium intake can exacerbate certain disease conditions. Persons suffering from hypertension, cardiovascular or renal diseases, should restrict their sodium intake. In the case of bottle-fed infants, sodium intake should also be restricted (South African Domestic Water Use, 2001).
[http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

46. SULPHATE

Sulphate is the oxy-anion of sulphur in the +VI oxidation state and forms salts with various cations such as potassium, sodium, calcium, magnesium, barium, lead and ammonium. Potassium, sodium, magnesium and ammonium sulphates are highly soluble, whereas calcium sulphate is partially soluble and barium and lead sulphates are insoluble. Consumption of excessive amounts of sulphate in drinking water typically results in diarrhoea. Sulphate imparts a bitter or salty taste to water, and is associated with varying degrees of unpalatability (South African Domestic Water Use, 2001). [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

Sulphate is a common constituent of water and arises from the dissolution of mineral sulphates in soil and rock, particularly calcium sulphate (gypsum) and other partially soluble sulphate minerals. Since most sulphates are soluble in water, and calcium sulphate relatively soluble, sulphates when added to water tend to accumulate to progressively increasing concentrations. Sulphates are discharged from acid mine wastes and many other industrial processes such as tanneries, textile mills and processes using sulphuric acid or

sulphates. Sulphates can be removed or added to water by ion exchange processes, and microbiological reduction or oxidation can interconvert sulphur and sulphate. Atmospheric sulphur dioxide, discharged on combustion of fossil fuels, can give rise to sulphuric acid in rainwater (acid-rain) and as such, this results in the return of sulphate to surface waters in the environment (South African Domestic Water Use, 2001). [http://www.dwa.gov.za/iwqs/wq_guide/Pol_saWQguideFRESH_vol1_Domesticuse.PDF]

High concentrations of sulphate exert predominantly acute health effects (diarrhoea). These are temporary and reversible since sulphate is rapidly excreted in the urine. Individuals exposed to elevated sulphate concentrations in their drinking water for long periods, usually become adapted and cease to experience these effects. Sulphate concentrations of 600 mg/L and more cause diarrhoea in most individuals and adaptation may not occur. Sulphate imparts a salty or bitter taste to water. The taste threshold for sulphate falls in the range of 200-400 mg/L and depends on whether the sulphate is predominantly associated with either sodium, potassium, calcium or magnesium, or mixtures thereof. Elevated sulphate concentrations also increase the erosion rate of metal fittings in distribution systems. According to the World Health Organization (2004) no health-based guideline value for sulphate in drinking water is proposed. However, there is an increasing likelihood of complaints arising from a noticeable taste as concentrations in water increase above 500 mg/L. [http://www.who.int/water_sanitation_health/dwq/chemicals/sulfate.pdf]

47. TASTE AND ODOUR

Taste and odour can originate from natural inorganic and organic chemical contaminants and biological sources or processes (e.g. aquatic microorganisms), from contamination by synthetic chemicals, from corrosion or as a result of problems with water treatment (e.g. chlorination). Taste and odour may also develop during storage and distribution as a result of microbial activity (WHO, 2017). In the assessment of drinking water quality, the sense of taste is more useful in detecting inorganic constituents, while the sense of smell detects organic constituents more effectively (DWA, 1996). Since taste and odour work together it is often difficult to distinguish the two. Odour concerns include:

- Strong Chlorine taste or smell – Generally this occurs when the water is treated at the water treatment plant to disinfect it. The addition of chlorine is used to kill off bacteria and other harmful microorganisms.
- Metallic taste – Some water systems have a high mineral concentration giving the consumer a salty or soda taste. In the case of Iron and Manganese, a strong metallic taste is readily detected.
- Rotten egg odour – This is usually a result of decaying organic deposits underground. As water flows through these areas, hydrogen sulphide gas is picked up, and when this water reaches the surface or comes out of the tap, the gas is released into the air. Hydrogen sulphide gas produces the rotten egg odour, can be corrosive to plumbing at high concentrations, and can tarnish silver rapidly. In large enough quantities. As little as 0.5 PPM (parts per million) can be tasted in drinking water.
- Musty or unnatural smells – These smells are normally a result of organic matter or even some pesticides in the water supply. Even very low amounts can introduce unpleasant odours into the water.

In many cases, aesthetic problems will be prevented by optimizing conventional treatment processes such as coagulation, sedimentation and chlorination. However, if specific treatment is deemed necessary, aeration, granular or powdered activated carbon and ozonation are generally effective techniques in removing organic chemicals and some inorganic chemicals, such as hydrogen sulfide, that cause tastes and odours.

Tastes and odours caused by disinfectants are best controlled through careful operation of the disinfection process and pretreatment to remove precursors. Manganese can be removed by chlorination followed by filtration. Techniques for removing hydrogen sulfide include aeration, granular activated carbon, filtration and oxidation. Ammonia can be removed by biological nitrification. Precipitation softening or cation exchange can reduce hardness. Other taste- and odour-causing inorganic chemicals (e.g. chloride and sulfate) are generally not amenable to treatment. [http://apps.who.int/iris/bitstream/10665/44584/1/9789241548151_eng.pdf]

Determination of Threshold Odour Numbers:

The measurement for odour is expressed in terms of the threshold Odour Number (TON), which is defined as the greatest dilution of sample with odour free water that yields a final water with an odour which is just detectable under carefully controlled test conditions (SAWQGs, 1996).

Sample diluted to 200 mL	Threshold Odour number (TON)	Sample diluted to 200 mL	Threshold Odour number (TON)
200	1	8.3	24
100	2	5.7	35
70	3	4	50
50	4	2.8	70
35	6	2	100
25	8	1.4	140
17	12	1	200

TON = (A + B)/ A

A – Volume of Sample with odour

B – Volume of Pure Water with no odour Added

If A was a 100 ml sample and 100 ml of water had to be added to not detect the odour – the TON would be 2. TON = (100 + 100)/ 100. Ref: http://www.water-research.net/odor.htm.

48. TOLUENE

Toluene is a clear, colourless liquid with a distinctive smell. It is a good solvent (a substance that can dissolve other substances). Toluene occurs naturally in crude oil and in the tolu tree. It is produced in the process of making gasoline and other fuels from crude oil and in making coke from coal (Division of Toxicology and Human Health Sciences, 2015). [https://www.atsdr.cdc.gov/ToxProfiles/tp56-c1-b.pdf]. Toluene can be

released into the air, water, and soil at places where it is produced or used. Toluene is commonly found in air, particularly when there is heavy vehicular traffic. Toluene can enter surface waters and groundwater (wells) from solvent and petroleum products spills. Toluene can also leak from underground storage tanks at gasoline stations and other facilities. When toluene-containing products are placed in landfills or waste disposal sites, toluene can enter the soil and water near the waste site. Toluene in surface soils rapidly evaporates into the air (Division of Toxicology and Human Health Sciences, 2015). [https://www.atsdr.cdc.gov/ToxProfiles/tp56-c1-b.pdf]

Toluene enters the environment when you use materials that contain it, such as paints, paint thinners, adhesives, fingernail polish, and gasoline; it evaporates rapidly from these materials and becomes mixed with the air you breathe. Toluene can enter your body from the air, water, or soil. After being taken into your body, the majority of toluene is removed from your body within a day; however, a small amount may accumulate in fat tissue with daily exposure (Division of Toxicology and Human Health Sciences, 2015). A serious health concern is that toluene may have an effect on your nervous system (brain and nerves). Nervous system effects can be temporary, such as headaches, dizziness, or unconsciousness. However, effects such as incoordination, cognitive impairment, and vision and hearing loss may become permanent with repeated exposure, especially at concentrations associated with intentional solvent abuse. Other health effects of potential concern may include immune, kidney, liver, and reproductive effects. Single exposures to toluene or repeated exposures over a few weeks can cause headaches and sleepiness, and can impair your ability to think clearly. Low to moderate, day-after-day exposure to toluene in your workplace can cause tiredness, confusion, weakness, drunken-type actions, memory loss, nausea, and loss of appetite. These symptoms usually disappear when exposure is stopped (Division of Toxicology and Human Health Sciences, 2015).

Toluene (Ingestion)

RfD UF:3000

RfD Rats: 0.08 mg/kg/day (NTP, 1990)

[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0118_summary.pdf]

NOAEL Mice: 22 mg/kg-d (Hsieh et al., 1989)

[Hsieh GC, RP Sharma, RDR Parker. 1989. Immuno-toxicological Evaluation of Toluene Exposure via Drinking Water in Mice. Env Res 49:93-103].

312 mg/kg/d (NTP, 1989)

[http://www.michigan.gov/documents/deq/deq-rrd-chem-TolueneDatasheet_527509_7.pdf]

LOAEL Mice: 625 mg/kg (NTP, 1989)

[http://www.michigan.gov/documents/deq/deq-rrd-chem-TolueneDatasheet_527509_7.pdf]

BMDL Rats: 238 mg/kg-day (NTP, 1990)

[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0118_summary.pdf]

Oral Slope Factor: N/A

Toluene (Inhalation)

RfC UF:10

RfC Human: 5 mg/m^3 (Foo et al., 1990)

[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0118_summary.pdf]

10 mg/m^3 (Zavalic et al., 1998a)

NOAEL Human: 128 mg/m^3 (NTP, 1990)

Numerous human studies have identified NOAELs in the range of 25-50 ppm toluene for individual neurological effects (Cavalleri et al., 2000; Eller et al., 1999; Nakatsuka et al., 1992; Neubert et al., 2001; Schaper et al., 2003; Zavalic et al., 1998a; Zupanic et al., 2002). These studies were designed to measure effects on subjective symptoms (e.g. headache, dizziness), colour vision, neurological and psychomotor functioning, and hearing. Several studies have shown statistically significant effects in workers in the range of 83-132 ppm on at least one of the following neurological effects: colour vision, auditory evoked brain potentials, neurobehavioral parameters, and neurological functioning (Abbate et al., 1993; Boey et al., 1997; Eller et al., 1999; Foo et al., 1990; Neubert et al. 2001; Vrca et al., 1995, 1996, 1997; Zavalic et al., 1998a).

LOAEL Human: 40-132ppm

(Vrca et al., 1995) (Neubert et al., 2001) (Zavalic et al., 1998a)

BMCL Human: 374 mg/m^3

(Zavalic et al., 1998a)

Slope Factor: N/A

49. TOTAL DISSOLVED SOLIDS

Total dissolved solids (TDS) is the term used to describe the inorganic salts and small amounts of organic matter present in solution in water. The principal constituents are usually calcium, magnesium, sodium, and potassium cations and carbonate, hydrogen carbonate, chloride, sulphate, and nitrate anions [http://www.who.int/water_sanitation_health/dwq/chemicals/tds.pdf]. TDS in water supplies originate from natural sources, sewage, urban and agricultural run-off, and industrial wastewater. Salts used for road de-icing can also contribute to the TDS loading of water supplies. Concentrations of TDS from natural sources have been found to vary. No recent data on health effects associated with the ingestion of TDS in drinking-water appear to exist; however, associations between various health effects and hardness, rather than TDS content, have been investigated in many studies. It was reported that mortality from all categories of ischaemic heart disease and acute myocardial infarction was increased in a community with high levels of soluble solids, calcium, magnesium, sulphate, chloride, fluoride, alkalinity, total hardness, and pH when compared with one in which levels were lower. Reliable data on possible health effects associated with the ingestion of TDS in drinking water are not available. The results of early epidemiological studies suggest that even low concentrations of TDS in drinking-water may have beneficial effects, although adverse effects have been reported in two limited investigations [http://www.who.int/water_sanitation_health/dwq/chemicals/tds.pdf].

50. TOTAL HARDNESS

Water hardness is the traditional measure of the capacity of water to react with soap, hard water requiring considerably more soap to produce a lather. Hard water often produces a noticeable deposit of precipitate (e.g. insoluble metals, soaps or salts). The principal natural sources of hardness in water are dissolved polyvalent metallic ions from sedimentary rocks, seepage and runoff from soils. Calcium and magnesium, the two principal ions, are present in many sedimentary rocks, the most common being limestone and chalk [http://www.who.int/water_sanitation_health/dwq/chemicals/hardness.pdf]. Small water supplies using groundwater often encounter significant levels of hardness, but some larger surface water supplies also have the same issue. Both calcium and magnesium are essential minerals and beneficial to human health in several respects. Inadequate intake of either nutrient can result in adverse health consequences. Inadequate intakes of calcium have been associated with increased risks of osteoporosis, nephrolithiasis (kidney stones), colorectal cancer, hypertension and stroke, coronary artery disease, insulin resistance and obesity. Low magnesium levels are associated with endothelial dysfunction, increased vascular reactions, elevated circulating levels of C-reactive protein (a proinflammatory marker that is a risk factor for coronary heart disease) and decreased insulin sensitivity. The major cause of hypermagnesaemia is renal insufficiency associated with a significantly decreased ability to excrete magnesium. Increased intake of magnesium salts may cause a temporary adaptable change in bowel habits (diarrhoea) [http://www.who.int/water_sanitation_health/dwq/chemicals/hardness.pdf].

51. TURBIDITY

Turbidity in water is caused by suspended particles or colloidal matter that obstructs light transmission through the water. It may be caused by inorganic or organic matter or a combination of the two. Microorganisms (bacteria, viruses and protozoa) are typically attached to particulates, and removal of turbidity by filtration will significantly reduce microbial contamination in treated water. Turbidity in some groundwater sources is a consequence of inert clay or chalk particles or the precipitation of non-soluble reduced iron and other oxides when water is pumped from anaerobic waters, whereas turbidity in surface waters may be the result of particulate matter of many types and is more likely to include attached microorganisms that are a threat to health. Turbidity in distribution systems can occur as a result of the disturbance of sediments and biofilms but is also from the ingress of dirty water from outside the system.

In addition, turbidity can seriously interfere with the efficiency of disinfection by providing protection for organisms, and much of water treatment is directed at removal of particulate matter before disinfection. This not only will increase the efficacy of disinfection by chemical disinfectants such as chlorine and ozone, but is an essential step in ensuring the effectiveness of physical disinfection processes such as ultraviolet irradiation, because light transmission through water is impaired by particulates. Removal of particulate matter by coagulation and sedimentation and by filtration is an important barrier in achieving safe drinking-water. Achieving low turbidity by filtration (before disinfection) of water from surface sources and groundwaters where raised turbidity occurs − for instance, where these are under the influence of surface waters − is strongly recommended to ensure microbially safe water.

Turbidity can also have a negative impact on consumer acceptability of water as a result of visible cloudiness. Although turbidity per se (e.g. from groundwater minerals or from post-precipitation of calcium carbonate from lime treatment) is not necessarily a threat to health, it is an important indicator of the possible presence of contaminants that would be of concern for health, especially from inadequately treated or unfiltered surface water. Data are emerging that show an increasing risk of gastro intestinal infections that correlates with high turbidity and turbidity events in distribution. This may be because turbidity is acting as an indicator of possible sources of microbial contamination. Therefore, turbidity events should be investigated and the causes corrected, whereas turbidity should be minimized as far as is possible within the constraints of the type of system and the resources available as one part of the management of distribution to achieve water safety.

Turbidity is also an important consideration when investment decisions are made regarding sources and treatment for water supplies and should be identified in the water safety plan as a hazard that needs to be controlled. Turbidity is measured by nephelometric turbidity units (NTU) and can be initially noticed by the naked eye above approximately 4.0 NTU. However, to ensure effectiveness of disinfection, turbidity should be no more than 1 NTU and preferably much lower. Large, well-run municipal supplies should be able to achieve less than 0.5 NTU before disinfection at all times and should be able to average 0.2 NTU or less. Surface water (and groundwater under the influence of surface water) treatment systems that achieve less than 0.3 NTU prior to disinfection will have demonstrated that they have significant barriers against pathogens that adsorb to particulate matter. Of particular importance is the fact that this will be a good indicator that they are removing chlorine-resistant pathogens such as *Cryptosporidium*.

Small water supplies where resources are very limited and where there is limited or no treatment may not be able to achieve such low levels of turbidity. In these cases, the aim should be to produce water that has turbidity of at least less than 5 NTU and, if at all possible, below 1 NTU. For many of these small and usually rural supplies, measuring turbidity below 5 NTU may present a significant cost challenge, and thus providing low-cost measuring systems that can measure lower turbidities is an important requirement. Occasionally, turbidity can be caused by minute air bubbles released when water has a high dissolved air content. Such turbidity clears rapidly upwards through the surface but can cause concern for consumers, and efforts should be made to manage distribution systems to ensure that this does not happen. [http://apps.who.int/iris/bitstream/10665/44584/1/9789241548151_eng.pdf]

52. URANIUM

Uranium is a naturally occurring radioactive element. Natural uranium is a mixture of three isotopes: 234U, 235U, and 238U. The most common isotope is 238U; it makes up about 99% of natural uranium by mass. All three isotopes behave the same chemically, but they have different radioactive properties (ATSDR, 2013).

Uranium is almost as hard as steel and much denser than lead. Natural uranium is used to make enriched uranium; depleted uranium is the leftover product. Enriched uranium is used to make fuel for nuclear power plants. Depleted uranium is used as a counterbalance on helicopter rotors and airplane control surfaces, as a shield to protect against ionizing radiation, as a component of munitions to help them penetrate enemy armoured vehicles, and as armour in some parts of military vehicles (ATSDR, 2013). Industries involved in mining, milling, and processing of uranium can also release it into the environment. Inactive uranium industries may continue to release uranium into the environment. For most people, food and drinking water are the main sources of uranium exposure. Root crops such as potatoes, parsnips, turnips, and sweet potatoes contribute the highest amounts of uranium to the diet. The amount of uranium in these foods is directly related to the amount of uranium in the soil in which they are grown (ATSDR, 2013).

People who work with materials and products that contain uranium may be exposed at work. This includes workers who mine, mill, or process uranium or make items that contain uranium. People who work with phosphate fertilizers may also be exposed to higher levels of uranium. People who live near uranium mining, processing, and manufacturing facilities could be exposed to more uranium than the general population. People may also be exposed if they live near areas where depleted uranium weapons are used (ATSDR, 2013). Natural and depleted uranium have the identical chemical effect on your body. The health effects of natural and depleted uranium are due to chemical effects and not to radiation. Uranium's main target is the kidneys. Kidney damage has been seen in humans and animals after inhaling or ingesting uranium compounds. However, kidney damage has not been consistently found in soldiers who have had uranium metal fragments in their bodies for several years. Ingesting water-soluble uranium compounds will result in kidney effects at lower doses than following exposure to insoluble uranium compounds. Workers who inhaled uranium hexafluoride have experienced respiratory irritation and accumulation of fluid in the lungs. However, these effects were attributed to the irritant hydrofluoric acid rather than the uranium. Inhaled insoluble uranium compounds can also damage the respiratory tract (ATSDR, 2013).

Rats ingesting uranium over a long time had neurobehavioral changes and changes in the levels of certain chemicals in the brain. Uranium has been shown to decrease fertility in some studies of rats and mice; other studies have not found this effect. Very soluble uranium compounds on the skin caused skin irritation and mild skin damage in animals (ATSDR, 2013). Neither the National Toxicology Program (NTP), International Agency for Research on Cancer (IARC), nor the EPA have classified natural uranium or depleted uranium with respect to carcinogenicity (ATSDR, 2013). [https://www.atsdr.cdc.gov/ToxProfiles/tp150-c1-b.pdf]

Uranium (Ingestion)
RfD UF: 1000
RfD Rabbits: 3E-3 mg/kg/day
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0259_summary.pdf]
NOAEL Rabbits: None
LOAEL Rabbits: 2.8 mg/kg/day
BMDL Rabbits: Not available (EPA, 1993)
Oral Slope Factor: N/A

Uranium (Inhalation)
RfC UF: Not available
RfC Human: Not available (EPA, 1993)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0259_summary.pdf]
NOAEL Human: Not available (EPA, 1993)
LOAEL Human: Not available (EPA, 1993)
BMCL Human: Not available (EPA, 1993)
Slope Factor: N/A

53. XYLENE

Also known as xylol or dimethylbenzene. Xylene is primarily a synthetic chemical. Chemical industries produce xylene from petroleum. Xylene also occurs naturally in petroleum and coal tar and is formed during forest fires, to a small extent. It is a colourless, flammable liquid with a sweet odour (ATSDR, 2007). [https://www.atsdr.cdc.gov/ToxProfiles/tp71-c1-b.pdf]. It is primarily used as a solvent (a liquid that can dissolve other substances) in the printing, rubber, and leather industries. Along with other solvents, xylene is also widely used as a cleaning agent, a thinner for paint, and in varnishes. Xylene is used, to a lesser extent, as a material in the chemical, plastics, and synthetic fibre industries and as an ingredient in the coating of fabrics and papers. Xylene is found in small amounts in airplane fuel and gasoline. Xylene evaporates and burns easily. Xylene does not mix well with water; however, it does mix with alcohol and many other chemicals. Most people begin to smell xylene in air at 0.08-3.7 parts of xylene per million parts of air (ppm) and in water at 0.53-1.1 ppm. Xylene is a liquid, and it can leak into soil, surface water (creeks, streams, rivers), or groundwater. Xylene can enter the environment when it is made, packaged, shipped, or used. Since xylene evaporates easily, most xylene that gets into soil and water (if not trapped underground) is expected to go into

the air where it is broken down by sunlight into other less harmful chemicals within a couple of days. Xylene below the soil surface may travel down through the soil and enter underground water (groundwater). Xylene may remain in groundwater for several months before it is finally broken down by small organisms. You are most likely to be exposed to xylene by breathing it in contaminated air. Xylene is sometimes released into water and soil as a result of the use, storage, and transport of petroleum products. You may be exposed to xylene by drinking or eating xylene-contaminated water or food. You may also come in contact with xylene from a variety of consumer products, including gasoline, paint, varnish, shellac, rust preventives, and cigarette smoke. Skin contact with products containing xylene, such as solvents, lacquers, paint thinners and removers, and pesticides may also expose you to xylene.

No health effects have been noted at the background levels that people are exposed to on a daily basis. Short term exposure of people to high levels of xylene can cause irritation of the skin, eyes, nose, and throat; difficulty in breathing; impaired function of the lungs; delayed response to a visual stimulus; impaired memory; stomach discomfort; and possible changes in the liver and kidneys. Both short- and long-term exposure to high concentrations of xylene can also cause a number of effects on the nervous system, such as headaches, lack of muscle coordination, dizziness, confusion, and changes in one's sense of balance. Some people exposed to very high levels of xylene for a short period of time have died. Information from animal studies is not adequate to determine whether or not xylene causes cancer in humans. Both the International Agency for Research on Cancer (IARC) and EPA have found that there is insufficient information to determine whether or not xylene is carcinogenic and consider xylene not classifiable as to its human carcinogenicity.

Xylene (Ingestion)
RfD UF:1000
RfD Rats: 0.2 mg/kg-day (NTP, 1986)
[https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0270_summary.pdf]
NOAEL Rats: 250 mg/kg-day (NTP, 1986)
200 mg/kg-day (Wolfe, 1988a)
150 mg/kg-day (Condie et al., 1988)
LOAEL Rats: 500 mg/kg-day (NTP, 1986)
800 mg/kg-day (Wolfe, 1988a)
750 mg/kg-day (Condie et al., 1988)
BMDL Rats: No Data
Oral Slope Factor: N/A

Xylene (Inhalation)
RfC UF: 300
RfC Rats: 2.2E+2 mg/m^3 (ATSDR, 2007)
0.1 mg/m^3 (Korsak et al., 1994)
NOAEL Rats: 50 ppm (Korsak et al., 1994)
39 mg/m^3 (HEC) (Korsak et al., 1994)
LOAEL Rats: 100ppm (Korsak et al., 1994)
78 mg/m^3 (HEC) (Korsak et al., 1994)
BMCL Human: No Data
Slope Factor: N/A

54. ZINC

Zinc is one of the most common elements in the Earth's crust. Zinc is found in the air, soil, and water and is present in all foods. In its pure elemental (or metallic) form, zinc is a bluish-white, shiny metal. Powdered zinc is explosive and may burst into flames if stored in damp places. Metallic zinc has many uses in industry. A common use for zinc is to coat steel and iron as well as other metals to prevent rust and corrosion; this process is called galvanization. Metallic zinc is also mixed with other metals to form alloys such as brass and bronze (ATSDR, 2005). Zinc compounds that may be found at hazardous waste sites are zinc chloride, zinc oxide, zinc sulphate, and zinc sulphide. Most zinc ore found naturally in the environment is in the form of zinc sulphide. Zinc sulphide and zinc oxide are used to make white paints, ceramics, and other products. Zinc oxide is also used in producing rubber. Zinc compounds, such as zinc acetate, zinc chloride, and zinc sulphate, are used in preserving wood and in manufacturing and dyeing fabrics. Zinc chloride is also the major ingredient in smoke from smoke bombs. Zinc compounds are used by the drug industry as ingredients in some common products, such as vitamin supplements, sun blocks, diaper rash ointments, deodorants, athlete's foot preparations, acne and poison ivy preparations, and antidandruff shampoos (ATSDR, 2005). [https://www.atsdr.cdc.gov/ToxProfiles/tp60-c1-b.pdf]

Zinc enters the air, water, and soil as a result of both natural processes and human activities. Most zinc enters the environment as the result of mining, purifying of zinc, lead, and cadmium ores, steel production, coal burning, and burning of wastes (ATSDR, 2005). Zinc is an essential element needed by your body in small amounts. We are exposed to zinc compounds in food. Zinc is also present in most drinking water. Drinking water or other beverages may contain high levels of zinc if they are stored in metal containers or flow through pipes that have been coated with zinc to resist rust (ATSDR, 2005). [https://www.atsdr.cdc.gov/ToxProfiles/tp60-c1-b.pdf]

Zinc can enter the body through the digestive tract when you eat food or drink water containing it. Zinc can also enter through your lungs if you inhale zinc dust or fumes from zinc-smelting or zinc welding operations on your job. The amount of zinc that passes directly through the skin is relatively small (ATSDR, 2005). [https://www.atsdr.cdc.gov/ToxProfiles/tp60-c1-b.pdf]

Inhaling large amounts of zinc (as zinc dust or fumes from smelting or welding) can cause a specific short-term disease called metal fume fever, which is generally reversible once exposure to zinc ceases. However, very little is known about the long-term effects of breathing zinc dust or fumes (ATSDR, 2005). Taking too much zinc into the body through food, water, or dietary supplements can also affect health. The levels of zinc that produce adverse health effects are much higher than the Recommended Dietary Allowances (RDAs) for zinc of 11 mg/day for men and 8 mg/day for women. If large doses of zinc (10-15 times higher than the RDA) are taken by mouth even for a short time, stomach cramps, nausea, and vomiting may occur. Ingesting high levels of zinc for several months may cause anaemia, damage the pancreas, and decrease levels of high density lipoprotein (HDL) cholesterol (ATSDR, 2005). [https://www.atsdr.cdc.gov/ToxProfiles/tp60-c1-b.pdf]
EPA has determined that because of lack of information, zinc is not classifiable as to its human carcinogenicity. EPA has stated that drinking water should contain no more than 5 mg of zinc per litre of water (5 mg/L or 5 ppm) because of taste (ATSDR, 2005). [https://www.atsdr.cdc.gov/ToxProfiles/tp60-c1-b.pdf]

Zinc (Ingestion)

RfD UF: 3

RfD Human: 0.3 mg/kg/d (Yadrick et al., 1989), (Fischer et al., 1984), (Davis et al., 2000), (Milne et al., 2001)

NOAEL Human: None

LOAEL Human: 0.91 mg/kg/d (Yadrick et al., 1989), (Fischer et al., 1984), (Davis et al., 2000), (Milne et al., 2001)

BMDL Human: No Data

Oral Slope Factor: N/A

Zinc (Inhalation)

RfC UF: No data

RfC Human: Not available at this time (EPA, 2005)

NOAEL Human: Not available at this time (EPA, 2005)

LOAEL Human: Not available at this time (EPA, 2005)

BMCL Human: N/A

Slope Factor: N/A

APPENDIX C: DSS OUTPUT REPORTING SHEETS OF THE RISK BASED THRESHOLD CRITERIA OF APPLICABLE WATER QUALITY CONSTITUENTS PER DOMESTIC USE CATEGORY

All Domestic Water Use Categories – Constituents Report sheet

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Exposure
Acrylamide	mg/l	0.032	No negative health impacts.	0.065	No negative health impacts.	0.097	Confusion, disorientation, memory disturbances and hallucinations.	0.129	Skin irritation, fatigue, foot weakness and sensory changes.	Ingestion
Aluminium	mg/l	0.1	No effects	0.3	Minor effects	0.5	Noticeable discolouration occurs in association with iron or manganese	0.6	Severe adverse effects occur in the presence of iron or manganese	Physical
Ammonia	mg/l	15	No negative health impacts.	147	No negative health impacts.	279	Influences metabolism.	411	Cells mutagenicity.	Human Health
Antimony	mg/l	0.012	No negative health impacts.	0.111	No negative health impacts.	0.6	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	10.5	Respiratory and eye problems, staining of tooth surface.	Human Health
Arsenic	mg/l	0.015	No negative health impacts.	0.039	No negative health impacts.	4.139	Vomiting, abdominal pain and diarrhoea.	8.238	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Human Health
Asbestos	mg/l	7.269	No negative health impacts.	14.538	No negative health impacts.	21.808	Bronchial diseases/illnesses.	29.077	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Human Health
Atrazine	mg/l	52.5	No negative health impacts.	105	No negative health impacts.	427.5	Lowering of the immune system.	750	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Human Health
Barium	mg/l	6	No negative health impacts.	6.3	No negative health impacts.	6.6	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	6.9	Cardiovascular (hypertension) effects, toxic.	Human Health
Benzene	mg/l	0.007	No negative health impacts.	0.014	No negative health impacts.	0.021	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.028	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Human Health
Benzo(a)pyrene	mg/l	0.003	No negative health impacts.	0.006	No negative health impacts.	0.009	Red blood cell damage, leading to anaemia; suppressed immune system.	0.012	Affects foetal development and Reproductive organs in females.	Human Health
Boron	mg/l	6	No negative health impacts.	12	No negative health impacts.	18	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	24	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Human Health
Bromide	mg/l	21	No negative health impacts.	210	No negative health impacts.	399	Nausea and vomiting (gastrointestinal symptoms).	588	No major long-term health effects observed.	Human Health
Cadmium	mg/l	0.016	No negative health impacts.	0.032	No negative health impacts.	0.048	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.065	Kidney dysfunction, kidney, liver, bone and blood damage.	Human Health

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Exposure
Calcium	mg/l	0.06	No negative health impacts.	0.12	No negative health impacts.	0.18	No major effects on human health.	0.24	No conclusive evidence.	Human Health
	mg/l	10	Possible corrosive effects (appliances/equipment)	32	No scaling, Insignificant effect on lathering of soap (Bathing; Laundry)	80	Increased scaling (appliances); lathering of soap slightly impaired (bathing/laundry)	150	High degree of scaling (appliances); impaired lathering of soap(bathing/laundry)	Physical
Carbon tetrachloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Human Health
Chloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Human Health
	mg/l	50	No effects	200	Increased corrosion effects in appliances	600	Noticeable increase in corrosion rates in appliances; Distinctly salty taste of water	1200	Objectionable salty taste; Likelihood of rapid corrosion in appliances	Physical
Chlorine	mg/l	3	No negative health impacts.	4.32	No negative health impacts.	5.64	Acts as an Oestrogen Mimic, Causes Weight Gain.	6.96	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Human Health
Chloroform	mg/l	0.323	No negative health impacts.	16.154	No negative health impacts.	216.462	Cardiac arrhythmias and abnormalities of the liver and kidneys.	416.769	Degrades blood, liver, and kidney.	Human Health
Chromium (VI)	mg/l	0.097	No negative health impacts.	0.614	No negative health impacts.	1.362	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	1.615	Genotoxic effects, lung cancer.	Human Health
Colour	TCU	1	No visual effects.	5	No visual effects.	12	No visual effects.	15	Taste and staining effects due to the presence of iron or manganese.	Physical
Copper	mg/l	0.42	No negative health impacts.	0.816	No negative health impacts.	79.908	Vomiting, diarrhoea, stomach cramps, and nausea.	159	Liver damage and kidney disease.	Human Health
Cyanide	mg/l	0.019	No negative health impacts.	0.038	No negative health impacts.	0.057	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.076	Chronic effects on thyroid and nervous system.	Human Health
Dissolved Organic Carbon	mg/l	1	No negative health impacts.	5	Slight taste odour, and colour effect.	10	Significant taste, colour and odour effects.	20	Severe taste, colour and odour effects.	Human Health
	mg/l	5	No effects	10	Slight taste odour, and colour effect	20	Significant taste, colour and odour effects	25	Severe taste, colour and odour effects	Physical
DDT and metabolites	mg/l	0.016	No negative health impacts.	1.615	No negative health impacts.	3.215	Low observed effects.	4.83	Liver, nervous and reproductive systems.	Human Health

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Exposure
Electrical Conductivity	mS/m	70	No effects on appliances/equipment.	150	No effects on appliances/equipment.	300	Slight scaling or corrosion of appliances and piping may be expected.	450	Increasing scaling and corrosion of appliances/equipment.	Physical
Ethylbenzene	mg/l	1.5	No negative health impacts.	1456.5	No negative health impacts.	2910.75	No observed short-term effects.	4365	No observed long-term effects.	Human Health
Fluoride	mg/l	1.5	No negative health impacts.	1.8	No negative health impacts.	30	Mild dental fluorosis.	60	Severe effects on skeletal tissues (bones and teeth).	Human Health
	mg/l	0.5	No effects	1	No effects	1.5	Discolouration of dental enamel occurs in sensitive, susceptible users	2	Discolouration of dental enamel occurs in sensitive, susceptible users	Physical
Glyphosate and AMPA	mg/l	1.5	No negative health impacts.	150	No negative health impacts.	300	No observed short-term effects.	750	Erosion of the gastrointestinal tract.	Human Health
Iron	mg/l	4.05	No negative health impacts.	8.1	No negative health impacts.	12.15	Unlikely to cause adverse effects in healthy persons.	20.25	Unlikely to cause adverse effects in healthy persons.	Human Health
	mg/l	0.1	no effects	0.3	Slight taste effects, staining of laundry and fixtures; slimy coatings in plumbing equipment begin to occur	1	increased taste and colour effects, increased staining of laundry and fixtures; increased problems with plumbing	10	Objectionable taste and appearance; Pronounced staining of laundry and fixtures, and effects on plumbing (slimy coatings)	Physical
Lead	mg/l	0.075	No negative health impacts.	0.15	No negative health impacts.	0.225	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	0.3	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Human Health
Magnesium	mg/l	30	no effects	70	slight scaling problems (together with calcium), slight impairment of soap lathering (bathing; laundry)	100	Slightly bitter taste; lathering of soap moderately impaired; scaling problems encountered (together with calcium)	200	unacceptable bitter taste; increased scaling problems	Physical
Manganese	mg/l	4.2	No negative health impacts.	9	No negative health impacts.	13.8	No observed effects.	18.6	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Human Health
	mg/l	0.05	no effects	0.15	slight taste effect; slight discolouration of water; slight staining of white clothes	1	increasing taste and colour; pale brown discolouration of water, moderate staining of clothes and fixtures	5	Extreme staining; aesthetically unacceptable to users	Physical
Monochloramine	mg/l	3	No negative health impacts.	285	No negative health impacts.	567	No minor health effects observed.	849	No major Health effects observed.	Human Health
Mercury	mg/l	3	No negative health impacts.	6.9	No negative health impacts.	10.8	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	14.7	Mental disturbances, tremors and gingivitis, kidney failure.	Human Health
Nickel	mg/l	0.646	No negative health impacts.	161.538	No negative health impacts.	888.462	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	1615.385	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Exposure
Nitrate	mg/l	24	No negative health impacts.	48	No negative health impacts.	72	Poor transportation of oxygen into the blood	96	Congenital malformations.	Human Health
Odour	TON	1	No noticeable odour.	2	Noticeable odour.	5	Strong odour objectionable to users.	10	Odour becomes stronger and more objectionable.	Physical
Acidic pH	-	7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Human Health
	unitless	7	No effects	6	No effects, however slight taste effects may be noted on occasion.	5	Slightly sour taste	4	Taste effect, sour taste;	Physical
Alkaline pH	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Human Health
	unitless	7	No effects	9	No effects, however slight taste effects may be noted on occasion.	10	Bitter taste	11	Soapy taste	Physical
Phenols	mg/l	9	No negative health impacts.	690	No negative health impacts.	2070	Increasing risk of negative health effects.	2790	Severe health effects	Human Health
Selenium	mg/l	0.15	No negative health impacts.	0.45	No negative health impacts.	0.75	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	1.2	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Human Health
Sodium	mg/l	100	No effect	200	Threshold for taste. Faintly salty taste	400	Slightly salty taste	1000	Very salty taste	Physical
Sulphate	mg/l	200	No effect	400	Slight taste noticeable	600	Definite salty or bitter taste	1000	Very strong salty and bitter	Physical
Toluene	mg/l	0.15	No negative health impacts.	3.84	No negative health impacts	601.68	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	995.28	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Human Health
Total Dissolved Solids	mg/l	450	No effects on appliances/equipment, A slight taste effect may be detected above 45 mS/m depending on the taste threshold.	1000	No effects on appliances/equipment, Noticeable salty taste but well tolerated.	2000	Marked salty taste. Slight scaling or corrosion of appliances and piping may be expected	3000	Extremely salty and bitter taste. Corrosive and increased scaling of appliances/equipment	Physical
Total Hardness	mg Ca CO3/l	50	Soft (but some corrosion of appliances).	100	Moderately soft (some protection against corrosion).	200	Moderately hard (some scaling of appliances; soap lathering impaired).	300	Very hard (effect on taste, severe scaling of appliances; soap lathering severely impaired).	Physical
Turbidity	NTU	0	No adverse effects.	1	Slight aesthetic effect.	5	Turbidity is visible. Slightly cloudy appearance.	10	Severe appearance, taste and odour effects.	Human Health

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Exposure
Uranium (238)	mg/l	0.09	No negative health impacts.	0.45	No negative health impacts.	0.81	No observed effects.	1.26	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Human Health
Xylene	mg/l	6	No negative health impacts.	5370	No negative health impacts.	10185	Disturbances of cognitive abilities, balance, and coordination.	15000	Damage to the central nervous system, liver and kidneys.	Human Health
Zinc	mg/l	9	No negative health impacts.	15.09	No negative health impacts.	21.18	Stomach cramps, nausea and vomiting.	27.3	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Human Health
	mg/l	3	No effect.	5	Slight opalescence or bitter taste.	10	Clearly discernible bitter taste and opalescence.	50	Bitter taste; strong opalescence.	Physical

Drinking Constituents Report Sheet

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Acrylamide	mg/l	0.032	No negative health impacts.	0.065	No negative health impacts.	0.097	Confusion, disorientation, memory disturbances and hallucinations.	0.129	Skin irritation, fatigue, foot weakness and sensory changes.	Ingestion
Ammonia	mg/l	15	No negative health impacts.	147	No negative health impacts.	279	Influences metabolism.	411	Cells mutagenicity.	Ingestion
Antimony	mg/l	0.012	No negative health impacts.	0.111	No negative health impacts.	0.6	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	10.5	Respiratory and eye problems, staining of tooth surface.	Ingestion
Arsenic	mg/l	0.015	No negative health impacts.	0.039	No negative health impacts.	4.139	Vomiting, abdominal pain and diarrhoea.	8.238	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Ingestion
Asbestos	mg/l	7.269	No negative health impacts.	14.538	No negative health impacts.	21.808	Bronchial diseases/illnesses.	29.077	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Ingestion
Atrazine	mg/l	52.5	No negative health impacts.	105	No negative health impacts.	427.5	Lowering of the immune system.	750	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Ingestion
Barium	mg/l	6	No negative health impacts.	6.3	No negative health impacts.	6.6	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	6.9	Cardiovascular (hypertension) effects, toxic.	Ingestion
Benzene	mg/l	0.007	No negative health impacts.	0.014	No negative health impacts.	0.021	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.028	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Ingestion
Benzo(a)pyrene	mg/l	0.003	No negative health impacts.	0.006	No negative health impacts.	0.009	Red blood cell damage, leading to anaemia; suppressed immune system.	0.012	Affects foetal development and Reproductive organs in females.	Ingestion
Boron	mg/l	6	No negative health impacts.	12	No negative health impacts.	18	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	24	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Ingestion
Bromide	mg/l	21	No negative health impacts.	210	No negative health impacts.	399	Nausea and vomiting (gastrointestinal symptoms).	588	No major long-term health effects observed.	Ingestion
Cadmium	mg/l	0.016	No negative health impacts.	0.032	No negative health impacts.	0.048	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.065	Kidney dysfunction, kidney, liver, bone and blood damage.	Ingestion
Calcium	mg/l	0.06	No negative health impacts.	0.12	No negative health impacts.	0.18	No major effects on human health.	0.24	No conclusive evidence.	Ingestion
Carbon tetrachloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Chloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Ingestion
Chlorine	mg/l	3	No negative health impacts.	4.32	No negative health impacts.	5.64	Acts as an Oestrogen Mimic, Causes Weight Gain.	6.96	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Ingestion
Chloroform	mg/l	0.323	No negative health impacts.	16.154	No negative health impacts.	216.462	Cardiac arrhythmias and abnormalities of the liver and kidneys.	416.769	Degrades blood, liver, and kidney.	Ingestion
Chromium (VI)	mg/l	0.097	No negative health impacts.	0.614	No negative health impacts.	1.362	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	1.615	Genotoxic effects, lung cancer.	Ingestion
Colour	TCU	1	No visual effects.	5	No visual effects.	12	No visual effects.	15	Taste and staining effects due to the presence of iron or manganese.	Aesthetic
Copper	mg/l	0.42	No negative health impacts.	0.816	No negative health impacts.	79.908	Vomiting, diarrhoea, stomach cramps, and nausea.	159	Liver damage and kidney disease.	Ingestion
Cyanide	mg/l	0.019	No negative health impacts.	0.038	No negative health impacts.	0.057	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.076	Chronic effects on thyroid and nervous system.	Ingestion
Dissolved Organic Carbon	mg/l	1	No negative health impacts.	5	Slight taste odour, and colour effect.	10	Significant taste, colour and odour effects.	20	Severe taste, colour and odour effects.	Aesthetic
DDT and metabolites	mg/l	0.016	No negative health impacts.	1.615	No negative health impacts.	3.215	Low observed effects.	4.83	Liver, nervous and reproductive systems.	Ingestion
Electrical Conductivity	mS/m	70	No negative health impacts.	150	Noticeable salty taste but well tolerated.	300	Marked salty taste.	450	Extremely salty and bitter taste.	Aesthetic
Fluoride	mg/l	1.5	No negative health impacts.	1.8	No negative health impacts.	30	Mild dental fluorosis.	60	Severe effects on skeletal tissues (bones and teeth).	Ingestion
Glyphosate and AMPA	mg/l	3	No negative health impacts.	300	No negative health impacts.	600	No observed short-term effects.	1500	Erosion of the gastrointestinal tract.	Ingestion
Iron	mg/l	4.05	No negative health impacts.	8.1	No negative health impacts.	12.15	Unlikely to cause adverse effects in healthy persons.	20.25	Unlikely to cause adverse effects in healthy persons.	Ingestion
Lead	mg/l	0.075	No negative health impacts.	0.15	No negative health impacts.	0.225	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	0.3	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Ingestion
Magnesium	mg/l	1	No adverse effects on taste.	2	No adverse effects on taste.	5	Taste effects are present.	10	Severe taste effects present.	Aesthetic
Manganese	mg/l	4.2	No negative health impacts.	9	No negative health impacts.	13.8	No observed effects.	18.6	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Ingestion
Monochloramine	mg/l	3	No negative health impacts.	285	No negative health impacts.	567	No minor health effects observed.	849	No major Health effects observed.	Ingestion
Mercury	mg/l	3	No negative health impacts.	6.9	No negative health impacts.	10.8	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	14.7	Mental disturbances, tremors and gingivitis, kidney failure.	Ingestion
Nickel	mg/l	0.646	No negative health impacts.	161.538	No negative health impacts.	888.462	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	1615.385	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Nitrate	mg/l	24	No negative health impacts.	48	No negative health impacts.	72	Poor transportation of oxygen into the blood	96	Congenital malformations.	Ingestion
Odour	TON	1	No noticeable odour.	2	Noticeable odour.	5	Strong odour objectionable to users.	10	Odour becomes stronger and more objectionable.	Aesthetic
Acidic pH	-	7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Ingestion
Alkaline pH	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Ingestion
Phenols	mg/l	9	No negative health impacts.	690	No negative health impacts.	2070	Increasing risk of negative health effects.	2790	Severe health effects	Ingestion
Selenium	mg/l	0.15	No negative health impacts.	0.45	No negative health impacts.	0.75	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	1.2	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Ingestion
Sodium	mg/l	100	Faintly salty taste.	200	Slightly salty taste.	600	Distinctly salty taste.	1000	Very salty taste.	Aesthetic
Sulphate	mg/l	200	Slight taste noticeable.	400	Definite salty or bitter taste.	600	Pronounced salty or bitter taste.	1000	Very strong salty and bitter.	Aesthetic
Toluene	mg/l	0.15	No negative health impacts.	3.84	No negative health impacts	601.68	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	995.28	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Ingestion
Total Dissolved salts	mg/l	450	A slight taste effect may be detected.	1000	Noticeable salty taste but well tolerated.	2000	Marked salty taste.	3400	Extremely salty and bitter taste.	Aesthetic
Total Hardness	mg/l	25	A slight taste effect may be detected.	150	Noticeable taste but well tolerated.	300	Marked unpleasant taste.	600	Extremely unpleasant taste.	Aesthetic
Turbidity	NTU	0.1	No adverse effects.	1	Slight aesthetic effect.	5	Turbidity is visible. Slightly cloudy appearance.	10	Severe appearance, taste and odour effects.	Aesthetic
Uranium (238)	mg/l	0.09	No negative health impacts.	0.45	No negative health impacts.	0.81	No observed effects.	1.26	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Ingestion
Xylene	mg/l	6	No negative health impacts.	5370	No negative health impacts.	10185	Disturbances of cognitive abilities, balance, and coordination.	15000	Damage to the central nervous system, liver and kidneys.	Ingestion
Zinc	mg/l	9	No negative health impacts.	15.09	No negative health impacts.	21.18	Stomach cramps, nausea and vomiting.	27.3	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Ingestion

Bathing Constituents Report Sheet

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Acrylamide	mg/l	4.308	No negative health impacts.	8.615	No negative health impacts.	12.923	Confusion, disorientation, memory disturbances and hallucinations.	17.231	Skin irritation, fatigue, foot weakness and sensory changes.	Ingestion
		140.14	No negative health impacts.	280.28	No negative health impacts.	420.42	Confusion, disorientation, memory disturbances and hallucinations.	560.559	Skin irritation, fatigue, foot weakness and sensory changes.	Dermal Contact
		0	No negative health impacts.	0.001	No negative health impacts.	0.003	Confusion, disorientation, memory disturbances and hallucinations.	0.004	Skin irritation, fatigue, foot weakness and sensory changes.	Inhalation
Ammonia	mg/l	2000	No negative health impacts.	19600	No negative health impacts.	37200	Influences metabolism.	54800	Cells mutagenicity.	Ingestion
		9.091	No negative health impacts.	89.091	No negative health impacts.	169.091	Influences metabolism.	249.091	Cells mutagenicity.	Dermal Contact
		98.039	No negative health impacts.	372.549	No negative health impacts.	1823.529	Influences metabolism.	2686.275	Cells mutagenicity.	Inhalation
Antimony	mg/l	1.6	No negative health impacts.	14.8	No negative health impacts.	80	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	1400	Respiratory and eye problems, staining of tooth surface.	Ingestion
		0.007	No negative health impacts.	0.067	No negative health impacts.	0.364	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	6.364	Respiratory and eye problems, staining of tooth surface.	Dermal Contact
Arsenic	mg/l	1.938	No negative health impacts.	5.169	No negative health impacts.	551.815	Vomiting, abdominal pain and diarrhoea.	1098.462	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Ingestion
		0.126	No negative health impacts.	0.336	No negative health impacts.	35.832	Vomiting, abdominal pain and diarrhoea.	71.329	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Dermal Contact
		0	No negative health impacts.	0	No negative health impacts.	0	Vomiting, abdominal pain and diarrhoea.	0.001	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Inhalation
Asbestos	mg/l	969.231	No negative health impacts.	1938.462	No negative health impacts.	2907.692	Bronchial diseases/illnesses.	3876.923	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Ingestion
		62.937	No negative health impacts.	125.874	No negative health impacts.	188.811	Bronchial diseases/illnesses.	251.748	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Dermal Contact
Atrazine	mg/l	7000	No negative health impacts.	14000	No negative health impacts.	57000	Lowering of the immune system.	100000	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Ingestion
		31.818	No negative health impacts.	63.636	No negative health impacts.	259.091	Lowering of the immune system.	454.545	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Dermal Contact
Barium	mg/l	800	No negative health impacts.	840	No negative health impacts.	880	Vomiting, abdominal cramps, and watery	920	Cardiovascular (hypertension) effects, toxic.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
							diarrhoea are typically reported shortly after ingestion.			
		1.818	No negative health impacts.	1.909	No negative health impacts.	2	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	2.091	Cardiovascular (hypertension) effects, toxic.	Dermal Contact
Benzene	mg/l	0.948	No negative health impacts.	1.895	No negative health impacts.	2.843	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	3.791	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Ingestion
		0.003	No negative health impacts.	0.006	No negative health impacts.	0.009	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.011	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Dermal Contact
		0.171	No negative health impacts.	0.051	No negative health impacts.	23.09	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	46.751	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Inhalation
Benzo(a)pyrene	mg/l	0.393	No negative health impacts.	0.786	No negative health impacts.	1.179	Red blood cell damage, leading to anaemia; suppressed immune system.	1.572	Affects foetal development and Reproductive organs in females.	Ingestion
		0	No negative health impacts.	0	No negative health impacts.	0	Red blood cell damage, leading to anaemia; suppressed immune system.	0	Affects foetal development and Reproductive organs in females.	Dermal Contact
Boron	mg/l	800	No negative health impacts.	1600	No negative health impacts.	2400	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	3200	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Ingestion
		3.636	No negative health impacts.	7.273	No negative health impacts.	10.909	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	14.545	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Dermal Contact
Bromide	mg/l	2800	No negative health impacts.	28000	No negative health impacts.	53200	Nausea and vomiting (gastrointestinal symptoms).	78400	No major long-term health effects observed.	Ingestion
		12.727	No negative health impacts.	127.273	No negative health impacts.	241.818	Nausea and vomiting (gastrointestinal symptoms).	356.364	No major long-term health effects observed.	Dermal Contact
Cadmium	mg/l	2.154	No negative health impacts.	4.308	No negative health impacts.	6.462	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	8.615	Kidney dysfunction, kidney, liver, bone and blood damage.	Ingestion
		0.07	No negative health impacts.	0.14	No negative health impacts.	0.21	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory	0.28	Kidney dysfunction, kidney, liver, bone and blood damage.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
							disturbances, liver injury, convulsions, shock and renal failure.			
		0.001	No negative health impacts.	0.003	No negative health impacts.	0.004	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.005	Kidney dysfunction, kidney, liver, bone and blood damage.	Inhalation
Calcium	mg/l	8	No negative health impacts.	16	No negative health impacts.	24	No major effects on human health.	32	No conclusive evidence.	Ingestion
		0.018	No negative health impacts.	0.036	No negative health impacts.	0.055	No major effects on human health.	0.073	No conclusive evidence.	Dermal Contact
Carbon tetrachloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Ingestion
		0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Dermal Contact
		0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Inhalation
Chloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Ingestion
		0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Dermal Contact
Chlorine	mg/l	400	No negative health impacts.	576	No negative health impacts.	752	Acts as an Oestrogen Mimic, Causes Weight Gain.	928	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Ingestion
		1.818	No negative health impacts.	2.618	No negative health impacts.	3.418	Acts as an Oestrogen Mimic, Causes Weight Gain.	4.218	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Dermal Contact
Chloroform	mg/l	43.077	No negative health impacts.	2153.846	No negative health impacts.	28861.538	Cardiac arrhythmias and abnormalities of the liver and kidneys.	55569.231	Degrades blood, liver, and kidney.	Ingestion
		2.797	No negative health impacts.	139.86	No negative health impacts.	1874.126	Cardiac arrhythmias and abnormalities of the liver and kidneys.	3608.392	Degrades blood, liver, and kidney.	Dermal Contact
Chromium (VI)	mg/l	12.923	No negative health impacts.	81.846	No negative health impacts.	181.655	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	215.385	Genotoxic effects, lung cancer.	Ingestion
		0.42	No negative health impacts.	2.657	No negative health impacts.	5.898	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	6.993	Genotoxic effects, lung cancer.	Dermal Contact
		0.002	No negative health impacts.	0.106	No negative health impacts.	0.317	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	0.422	Genotoxic effects, lung cancer.	Inhalation
Colour	TCU	1	No visual effects.	5	No visual effects.	12	No visual effects.	15	Taste and staining effects due to the presence of iron or manganese.	Aesthetic

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Copper	mg/l	56	No negative health impacts.	108.8	No negative health impacts.	10654.4	Vomiting, diarrhoea, stomach cramps, and nausea.	21200	Liver damage and kidney disease.	Ingestion
		0.127	No negative health impacts.	0.247	No negative health impacts.	24.215	Vomiting, diarrhoea, stomach cramps, and nausea.	48.182	Liver damage and kidney disease.	Dermal Contact
Cyanide	mg/l	2.52	No negative health impacts.	5.04	No negative health impacts.	7.56	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	10.08	Chronic effects on thyroid and nervous system.	Ingestion
		0.011	No negative health impacts.	0.023	No negative health impacts.	0.034	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.046	Chronic effects on thyroid and nervous system.	Dermal Contact
Dissolved Organic Carbon	mg/l	1	No negative health impacts.	5	Slight taste odour, and colour effect.	10	Significant taste, colour and odour effects.	20	Severe taste, colour and odour effects.	Aesthetic
DDT and metabolites	mg/l	2.154	No negative health impacts.	215.385	No negative health impacts.	428.615	Low observed effects.	644	Liver, nervous and reproductive systems.	Ingestion
		0.029	No negative health impacts.	2.92	No negative health impacts.	5.81	Low observed effects.	8.73	Liver, nervous and reproductive systems.	Dermal Contact
		0	No negative health impacts.	0	No negative health impacts.	0	Low observed effects.	0	Liver, nervous and reproductive systems.	Inhalation
Electrical Conductivity	mS/m	70	No negative health impacts.	150	Noticeable salty taste but well tolerated.	300	Marked salty taste.	450	Extremely salty and bitter taste.	Aesthetic
Fluoride	mg/l	200	No negative health impacts.	240	No negative health impacts.	4000	Mild dental fluorosis.	8000	Severe effects on skeletal tissues (bones and teeth).	Ingestion
		0.909	No negative health impacts.	1.091	No negative health impacts.	18.182	Mild dental fluorosis.	36.364	Severe effects on skeletal tissues (bones and teeth).	Dermal Contact
Glyphosate and AMPA	mg/l	400	No negative health impacts.	40000	No negative health impacts.	80000	No observed short-term effects.	200000	Erosion of the gastrointestinal tract.	Ingestion
		1.818	No negative health impacts.	181.818	No negative health impacts.	363.636	No observed short-term effects.	909.091	Erosion of the gastrointestinal tract.	Dermal Contact
Iron	mg/l	540	No negative health impacts.	1080	No negative health impacts.	1620	Unlikely to cause adverse effects in healthy persons.	2700	Unlikely to cause adverse effects in healthy persons.	Ingestion
		1.227	No negative health impacts.	2.455	No negative health impacts.	3.682	Unlikely to cause adverse effects in healthy persons.	6.136	Unlikely to cause adverse effects in healthy persons.	Dermal Contact
Lead	mg/l	10	No negative health impacts.	20	No negative health impacts.	30	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	40	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Ingestion
		0.023	No negative health impacts.	0.045	No negative health impacts.	0.068	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	0.091	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Dermal Contact
Magnesium	mg/l	1	No adverse effects on taste.	2	No adverse effects on taste.	5	Taste effects are present.	10	Severe taste effects present.	Aesthetic
Manganese	mg/l	560	No negative health impacts.	1200	No negative health impacts.	1840	No observed effects.	2480	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
		1.273	No negative health impacts.	2.727	No negative health impacts.	4.182	No observed effects.	5.636	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Dermal Contact
		0.01	No negative health impacts.	2.451	No negative health impacts.	6.127	No observed effects.	9.804	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Inhalation
Monochloramine	mg/l	400	No negative health impacts.	38000	No negative health impacts.	75600	No minor health effects observed.	113200	No major Health effects observed.	Ingestion
		0	No negative health impacts.	0	No negative health impacts.	0	No minor health effects observed.	0	No major Health effects observed.	Dermal Contact
Mercury	mg/l	400	No negative health impacts.	920	No negative health impacts.	1440	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	1960	Mental disturbances, tremors and gingivitis, kidney failure.	Ingestion
		1.818	No negative health impacts.	4.182	No negative health impacts.	6.545	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	8.909	Mental disturbances, tremors and gingivitis, kidney failure.	Dermal Contact
		0.059	No negative health impacts.	0.485	No negative health impacts.	1.125	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	1.765	Mental disturbances, tremors and gingivitis, kidney failure.	Inhalation
Nickel	mg/l	86.154	No negative health impacts.	21538.462	No negative health impacts.	118461.538	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	215384.615	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Ingestion
		2.797	No negative health impacts.	699.301	No negative health impacts.	3846.154	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	6993.007	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Dermal Contact
Nitrate	mg/l	3200	No negative health impacts.	6400	No negative health impacts.	9600	Poor transportation of oxygen into the blood	12800	Congenital malformations.	Ingestion
		14.545	No negative health impacts.	29.091	No negative health impacts.	43.636	Poor transportation of oxygen into the blood	58.182	Congenital malformations.	Dermal Contact
Odour	TON	1	No noticeable odour.	2	Noticeable odour.	5	Strong odour objectionable to users.	10	Odour becomes stronger and more objectionable.	Aesthetic
Acidic pH	-	7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Ingestion
		7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Dermal Contact
Alkaline pH	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Ingestion
		7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Dermal Contact
Phenols	mg/l	1200	No negative health impacts.	92000	No negative health impacts.	276000	Increasing risk of negative health effects.	372000	Severe health effects	Ingestion
		0.191	No negative health impacts.	14.673	No negative health impacts.	44.019	Increasing risk of negative health effects.	59.33	Severe health effects	Dermal Contact
Selenium	mg/l	20	No negative health impacts.	60	No negative health impacts.	100	Gastrointestinal disturbances, dermatitis, dizziness,	160	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
							lassitude and a garlic odour to the breath.			
		0.091	No negative health impacts.	0.273	No negative health impacts.	0.455	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	0.727	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Dermal Contact
Sodium	mg/l	100	Faintly salty taste.	200	Slightly salty taste.	600	Distinctly salty taste.	1000	Very salty taste.	Aesthetic
Sulphate	mg/l	200	Slight taste noticeable.	400	Definite salty or bitter taste.	600	Pronounced salty or bitter taste.	1000	Very strong salty and bitter.	Aesthetic
Toluene	mg/l	20	No negative health impacts.	512	No negative health impacts	80224	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	132704	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Ingestion
		0.091	No negative health impacts.	2.327	No negative health impacts	364.655	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	603.2	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Dermal Contact
		1960.784	No negative health impacts.	3921.569	No negative health impacts	5882.353	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	7843.137	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Inhalation
Total Dissolved salts	mg/l	450	A slight taste effect may be detected.	1000	Noticeable salty taste but well tolerated.	2000	Marked salty taste.	3400	Extremely salty and bitter taste.	Aesthetic
Total Hardness	mg/l	25	A slight taste effect may be detected.	150	Noticeable taste but well tolerated.	300	Marked unpleasant taste.	600	Extremely unpleasant taste.	Aesthetic
Turbidity	NTU	0.1	No adverse effects.	1	Slight aesthetic effect.	5	Turbidity is visible. Slightly cloudy appearance.	10	Severe appearance, taste and odour effects.	Aesthetic
Uranium (238)	mg/l	12	No negative health impacts.	60	No negative health impacts.	108	No observed effects.	168	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Ingestion
		0.055	No negative health impacts.	0.273	No negative health impacts.	0.491	No observed effects.	0.764	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Dermal Contact
Xylene	mg/l	800	No negative health impacts.	716000	No negative health impacts.	1358000	Disturbances of cognitive abilities, balance, and coordination.	2000000	Damage to the central nervous system, liver and kidneys.	Ingestion
		3.636	No negative health impacts.	3254.545	No negative health impacts.	6172.727	Disturbances of cognitive abilities, balance, and coordination.	9090.909	Damage to the central nervous system, liver and kidneys.	Dermal Contact
		19.608	No negative health impacts.	7647.059	No negative health impacts.	11470.588	Disturbances of cognitive abilities, balance, and coordination.	15294.118	Damage to the central nervous system, liver and kidneys.	Inhalation
Zinc	mg/l	1200	No negative health impacts.	2012	No negative health impacts.	2824	Stomach cramps, nausea and vomiting.	3640	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
		2.727	No negative health impacts.	4.573	No negative health impacts.	6.418	Stomach cramps, nausea and vomiting.	8.273	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Dermal Contact
		3	No visible effects.	5	No visible effects	10	Taste and colour effects present.	700	Severe taste and colour effects present.	Aesthetic

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Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Acrylamide	mg/l	0.129	No negative health impacts.	0.258	No negative health impacts.	0.388	Confusion, disorientation, memory disturbances and hallucinations.	0.517	Skin irritation, fatigue, foot weakness and sensory changes.	Ingestion
	mg/l	330.33	No negative health impacts.	660.659	No negative health impacts.	990.989	Confusion, disorientation, memory disturbances and hallucinations.	1321.319	Skin irritation, fatigue, foot weakness and sensory changes.	Dermal Contact
Ammonia	mg/l	60	No negative health impacts.	588	No negative health impacts.	1116	Influences metabolism.	1644	Cells mutagenicity.	Ingestion
	mg/l	21.429	No negative health impacts.	210	No negative health impacts.	398.571	Influences metabolism.	587.143	Cells mutagenicity.	Dermal Contact
Antimony	mg/l	0.048	No negative health impacts.	0.444	No negative health impacts.	2.4	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	42	Respiratory and eye problems, staining of tooth surface.	Ingestion
	mg/l	0.017	No negative health impacts.	0.159	No negative health impacts.	0.857	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	15	Respiratory and eye problems, staining of tooth surface.	Dermal Contact
Arsenic	mg/l	0.058	No negative health impacts.	0.155	No negative health impacts.	16.554	Vomiting, abdominal pain and diarrhoea.	32.954	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Ingestion
	mg/l	0.297	No negative health impacts.	0.791	No negative health impacts.	84.462	Vomiting, abdominal pain and diarrhoea.	168.132	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Dermal Contact
Asbestos	mg/l	29.077	No negative health impacts.	58.154	No negative health impacts.	87.231	Bronchial diseases/illnesses.	116.308	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Ingestion
	mg/l	148.352	No negative health impacts.	296.703	No negative health impacts.	445.055	Bronchial diseases/illnesses.	593.407	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Dermal Contact
Atrazine	mg/l	210	No negative health impacts.	420	No negative health impacts.	1710	Lowering of the immune system.	3000	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Ingestion
	mg/l	75	No negative health impacts.	150	No negative health impacts.	610.714	Lowering of the immune system.	1071.429	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Dermal Contact
Barium	mg/l	24	No negative health impacts.	25.2	No negative health impacts.	26.4	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	27.6	Cardiovascular (hypertension) effects, toxic.	Ingestion
	mg/l	4.286	No negative health impacts.	4.5	No negative health impacts.	4.714	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	4.929	Cardiovascular (hypertension) effects, toxic.	Dermal Contact
Benzene	mg/l	0.028	No negative health impacts.	0.057	No negative health impacts.	0.085	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.114	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Ingestion
	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Dermal Contact
Benzo(a)pyrene	mg/l	0.012	No negative health impacts.	0.024	No negative health impacts.	0.035	Red blood cell damage, leading to anaemia; suppressed immune system.	0.047	Affects foetal development and Reproductive organs in females.	Ingestion
	mg/l	0.007	No negative health impacts.	0.013	No negative health impacts.	0.02	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.027	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Boron	mg/l	24	No negative health impacts.	48	No negative health impacts.	72	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	96	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Ingestion
	mg/l	8.571	No negative health impacts.	17.143	No negative health impacts.	25.714	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	34.286	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Dermal Contact
Bromide	mg/l	84	No negative health impacts.	840	No negative health impacts.	1596	Nausea and vomiting (gastrointestinal symptoms).	2352	No major long-term health effects observed.	Ingestion
	mg/l	30	No negative health impacts.	300	No negative health impacts.	570	Nausea and vomiting (gastrointestinal symptoms).	840	No major long-term health effects observed.	Dermal Contact
Cadmium	mg/l	0.065	No negative health impacts.	0.129	No negative health impacts.	0.194	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.258	Kidney dysfunction, kidney, liver, bone and blood damage.	Ingestion
	mg/l	0.165	No negative health impacts.	0.33	No negative health impacts.	0.495	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.659	Kidney dysfunction, kidney, liver, bone and blood damage.	Dermal Contact
Calcium	mg/l	0.24	No negative health impacts.	0.48	No negative health impacts.	0.72	No major effects on human health.	0.96	No conclusive evidence.	Ingestion
	mg/l	0.043	No negative health impacts.	0.086	No negative health impacts.	0.129	No major effects on human health.	0.171	No conclusive evidence.	Dermal Contact
Carbon tetrachloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Ingestion
	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Dermal Contact
Chloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Ingestion
	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Dermal Contact
Chlorine	mg/l	12	No negative health impacts.	17.28	No negative health impacts.	22.56	Acts as an Oestrogen Mimic, Causes Weight Gain.	27.84	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Ingestion
	mg/l	4.286	No negative health impacts.	6.171	No negative health impacts.	8.057	Acts as an Oestrogen Mimic, Causes Weight Gain.	9.943	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Dermal Contact
Chloroform	mg/l	1.292	No negative health impacts.	64.615	No negative health impacts.	865.846	Cardiac arrhythmias and abnormalities of the liver and kidneys.	1667.077	Degrades blood, liver, and kidney.	Ingestion
	mg/l	6.593	No negative health impacts.	329.67	No negative health impacts.	4417.582	Cardiac arrhythmias and abnormalities of the liver and kidneys.	8505.495	Degrades blood, liver, and kidney.	Dermal Contact
Chromium (VI)	mg/l	0.388	No negative health impacts.	2.455	No negative health impacts.	5.45	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	6.462	Genotoxic effects, lung cancer.	Ingestion
	mg/l	0.989	No negative health impacts.	6.264	No negative health impacts.	13.902	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	16.484	Genotoxic effects, lung cancer.	Dermal Contact
Colour	TCU	1	No visual effects.	5	No visual effects.	12	No visual effects.	15	Taste and staining effects due to the presence of iron or manganese.	Aesthetic
Copper	mg/l	1.68	No negative health impacts.	3.264	No negative health impacts.	319.632	Vomiting, diarrhoea, stomach cramps, and nausea.	636	Liver damage and kidney disease.	Ingestion
	mg/l	0.3	No negative health impacts.	0.583	No negative health impacts.	57.077	Vomiting, diarrhoea, stomach cramps, and nausea.	113.571	Liver damage and kidney disease.	Dermal Contact
Cyanide	mg/l	0.076	No negative health impacts.	0.151	No negative health impacts.	0.227	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.302	Chronic effects on thyroid and nervous system.	Ingestion

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
	mg/l	0.027	No negative health impacts.	0.054	No negative health impacts.	0.081	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.108	Chronic effects on thyroid and nervous system.	Dermal Contact
Dissolved Organic Carbon	mg/l	1	No negative health impacts.	5	Slight taste odour, and colour effect.	10	Significant taste, colour and odour effects.	20	Severe taste, colour and odour effects.	Aesthetic
DDT and metabolites	mg/l	0.065	No negative health impacts.	6.462	No negative health impacts.	12.858	Low observed effects.	19.32	Liver, nervous and reproductive systems.	Ingestion
	mg/l	0.069	No negative health impacts.	6.882	No negative health impacts.	13.696	Low observed effects.	20.579	Liver, nervous and reproductive systems.	Dermal Contact
Electrical Conductivity	mS/m	70	No negative health impacts.	150	Noticeable salty taste but well tolerated.	300	Marked salty taste.	450	Extremely salty and bitter taste.	Aesthetic
Fluoride	mg/l	6	No negative health impacts.	7.2	No negative health impacts.	120	Mild dental fluorosis.	240	Severe effects on skeletal tissues (bones and teeth).	Ingestion
	mg/l	2.143	No negative health impacts.	2.571	No negative health impacts.	42.857	Mild dental fluorosis.	85.714	Severe effects on skeletal tissues (bones and teeth).	Dermal Contact
Glyphosate and AMPA	mg/l	12	No negative health impacts.	1200	No negative health impacts.	2400	No observed short-term effects.	6000	Erosion of the gastrointestinal tract.	Ingestion
	mg/l	4.286	No negative health impacts.	428.571	No negative health impacts.	857.143	No observed short-term effects.	2142.857	Erosion of the gastrointestinal tract.	Dermal Contact
Iron	mg/l	16.2	No negative health impacts.	32.4	No negative health impacts.	48.6	Unlikely to cause adverse effects in healthy persons.	81	Unlikely to cause adverse effects in healthy persons.	Ingestion
	mg/l	2.893	No negative health impacts.	5.786	No negative health impacts.	8.679	Unlikely to cause adverse effects in healthy persons.	14.464	Unlikely to cause adverse effects in healthy persons.	Dermal Contact
Lead	mg/l	0.3	No negative health impacts.	0.6	No negative health impacts.	0.9	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	1.2	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Ingestion
	mg/l	0.054	No negative health impacts.	0.107	No negative health impacts.	0.161	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	0.214	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Dermal Contact
Magnesium	mg/l	1	No adverse effects on taste.	2	No adverse effects on taste.	5	Taste effects are present.	10	Severe taste effects present.	Aesthetic
Manganese	mg/l	16.8	No negative health impacts.	36	No negative health impacts.	55.2	No observed effects.	74.4	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Ingestion
	mg/l	3	No negative health impacts.	6.429	No negative health impacts.	9.857	No observed effects.	13.286	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Dermal Contact
Monochloramine	mg/l	12	No negative health impacts.	1140	No negative health impacts.	2268	No minor health effects observed.	3396	No major Health effects observed.	Ingestion
	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No minor health effects observed.	0	No major Health effects observed.	Dermal Contact
Mercury	mg/l	12	No negative health impacts.	27.6	No negative health impacts.	43.2	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	58.8	Mental disturbances, tremors and gingivitis, kidney failure.	Ingestion
	mg/l	4.286	No negative health impacts.	9.857	No negative health impacts.	15.429	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	21	Mental disturbances, tremors and gingivitis, kidney failure.	Dermal Contact
Nickel	mg/l	2.585	No negative health impacts.	646.154	No negative health impacts.	3553.846	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	6461.538	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Ingestion
	mg/l	6.593	No negative health impacts.	1648.352	No negative health impacts.	9065.934	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	16483.516	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Nitrate	mg/l	96	No negative health impacts.	192	No negative health impacts.	288	Poor transportation of oxygen into the blood	384	Congenital malformations.	Ingestion
	mg/l	34.286	No negative health impacts.	68.571	No negative health impacts.	102.857	Poor transportation of oxygen into the blood	137.143	Congenital malformations.	Dermal Contact
Odour	TON	1	No noticeable odour.	2	Noticeable odour.	5	Strong odour objectionable to users.	10	Odour becomes stronger and more objectionable.	Aesthetic
Acidic pH	-	7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Ingestion
	-	4.615	No negative health impacts.	4.484	No negative health impacts.	4.286	No observed effects.	3.956	No observed effects.	Dermal Contact
Alkaline pH	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Ingestion
	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Dermal Contact
Phenols	mg/l	36	No negative health impacts.	2760	No negative health impacts.	8280	Increasing risk of negative health effects.	11160	Severe health effects	Ingestion
	mg/l	0.451	No negative health impacts.	34.586	No negative health impacts.	103.759	Increasing risk of negative health effects.	139.85	Severe health effects	Dermal Contact
Selenium	mg/l	0.6	No negative health impacts.	1.8	No negative health impacts.	3	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	4.8	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Ingestion
	mg/l	0.214	No negative health impacts.	0.643	No negative health impacts.	1.071	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	1.714	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Dermal Contact
Sodium	mg/l	100	Faintly salty taste.	200	Slightly salty taste.	600	Distinctly salty taste.	1000	Very salty taste.	Aesthetic
Sulphate	mg/l	200	Slight taste noticeable.	400	Definite salty or bitter taste.	600	Pronounced salty or bitter taste.	1000	Very strong salty and bitter.	Aesthetic
Toluene	mg/l	0.6	No negative health impacts.	15.36	No negative health impacts	2406.72	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	3981.12	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Ingestion
	mg/l	0.214	No negative health impacts.	5.486	No negative health impacts	859.543	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	1421.829	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Dermal Contact
Total Dissolved salts	mg/l	450	A slight taste effect may be detected.	1000	Noticeable salty taste but well tolerated.	2000	Marked salty taste.	3400	Extremely salty and bitter taste.	Aesthetic
Total Hardness	mg/l	25	A slight taste effect may be detected.	150	Noticeable taste but well tolerated.	300	Marked unpleasant taste.	600	Extremely unpleasant taste.	Aesthetic
Turbidity	NTU	0.1	No adverse effects.	1	Slight aesthetic effect.	5	Turbidity is visible. Slightly cloudy appearance.	10	Severe appearance, taste and odour effects.	Aesthetic
Uranium (238)	mg/l	0.36	No negative health impacts.	1.8	No negative health impacts.	3.24	No observed effects.	5.04	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Ingestion
	mg/l	0.129	No negative health impacts.	0.643	No negative health impacts.	1.157	No observed effects.	1.8	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Dermal Contact
Xylene	mg/l	24	No negative health impacts.	21480	No negative health impacts.	40740	Disturbances of cognitive abilities, balance, and coordination.	60000	Damage to the central nervous system, liver and kidneys.	Ingestion
	mg/l	8.571	No negative health impacts.	7671.429	No negative health impacts.	14550	Disturbances of cognitive abilities, balance, and coordination.	21428.571	Damage to the central nervous system, liver and kidneys.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Zinc	mg/l	36	No negative health impacts.	60.36	No negative health impacts.	84.72	Stomach cramps, nausea and vomiting.	109.2	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Ingestion
	mg/l	6.429	No negative health impacts.	10.779	No negative health impacts.	15.129	Stomach cramps, nausea and vomiting.	19.5	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Dermal Contact
	mg/l	3	No visible effects.	5	No visible effects	10	Taste and colour effects present.	700	Severe taste and colour effects present.	Aesthetic

Laundry Constituents Report Sheet

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Acrylamide	mg/l	330.33	No negative health impacts.	660.659	No negative health impacts.	990.989	Confusion, disorientation, memory disturbances and hallucinations.	1321.319	Skin irritation, fatigue, foot weakness and sensory changes.	Dermal Contact
Ammonia	mg/l	21.429	No negative health impacts.	210	No negative health impacts.	398.571	Influences metabolism.	587.143	Cells mutagenicity.	Dermal Contact
Antimony	mg/l	0.017	No negative health impacts.	0.159	No negative health impacts.	0.857	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	15	Respiratory and eye problems, staining of tooth surface.	Dermal Contact
Arsenic	mg/l	0.297	No negative health impacts.	0.791	No negative health impacts.	84.462	Vomiting, abdominal pain and diarrhoea.	168.132	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Dermal Contact
Asbestos	mg/l	148.352	No negative health impacts.	296.703	No negative health impacts.	445.055	Bronchial diseases/illnesses.	593.407	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Dermal Contact
Atrazine	mg/l	75	No negative health impacts.	150	No negative health impacts.	610.714	Lowering of the immune system.	1071.429	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Dermal Contact
Barium	mg/l	4.286	No negative health impacts.	4.5	No negative health impacts.	4.714	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	4.929	Cardiovascular (hypertension) effects, toxic.	Dermal Contact
Benzene	mg/l	0.007	No negative health impacts.	0.013	No negative health impacts.	0.02	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.027	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Dermal Contact
Benzo(a)pyrene	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Red blood cell damage, leading to anaemia; suppressed immune system.	0	Affects foetal development and Reproductive organs in females.	Dermal Contact
Boron	mg/l	8.571	No negative health impacts.	17.143	No negative health impacts.	25.714	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	34.286	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Dermal Contact
Bromide	mg/l	30	No negative health impacts.	300	No negative health impacts.	570	Nausea and vomiting (gastrointestinal symptoms).	840	No major long-term health effects observed.	Dermal Contact
Cadmium	mg/l	0.165	No negative health impacts.	0.33	No negative health impacts.	0.495	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.659	Kidney dysfunction, kidney, liver, bone and blood damage.	Dermal Contact
Calcium	mg/l	0.043	No negative health impacts.	0.086	No negative health impacts.	0.129	No major effects on human health.	0.171	No conclusive evidence.	Dermal Contact
	mg/l	10	Insignificant effect on lathering of soap.	32	Lathering of soap slightly impaired.	80	Impaired lathering of soap.	150	Lathering of soap severely impaired.	Physical damage.
Carbon tetrachloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Dermal Contact
Chloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Chlorine	mg/l	4.286	No negative health impacts.	6.171	No negative health impacts.	8.057	Acts as an Oestrogen Mimic, Causes Weight Gain.	9.943	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Dermal Contact
Chloroform	mg/l	6.593	No negative health impacts.	329.67	No negative health impacts.	4417.582	Cardiac arrhythmias and abnormalities of the liver and kidneys.	8505.495	Degrades blood, liver, and kidney.	Dermal Contact
Chromium (VI)	mg/l	0.989	No negative health impacts.	6.264	No negative health impacts.	13.902	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	16.484	Genotoxic effects, lung cancer.	Dermal Contact
Colour	TCU	1	No visual effects.	5	No visual effects.	12	No visual effects.	15	Taste and staining effects due to the presence of iron or manganese.	Aesthetic
Copper	mg/l	0.3	No negative health impacts.	0.583	No negative health impacts.	57.077	Vomiting, diarrhoea, stomach cramps, and nausea.	113.571	Liver damage and kidney disease.	Dermal Contact
	mg/l	0	No negative impacts.	0	No negative impacts.	0	No negative impacts.	0	No negative impacts.	Physical damage.
Cyanide	mg/l	0.027	No negative health impacts.	0.054	No negative health impacts.	0.081	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.108	Chronic effects on thyroid and nervous system.	Dermal Contact
Dissolved Organic Carbon	mg/l	1	No negative health impacts.	5	Slight taste odour, and colour effect.	10	Significant taste, colour and odour effects.	20	Severe taste, colour and odour effects.	Aesthetic
DDT and metabolites	mg/l	0.069	No negative health impacts.	6.882	No negative health impacts.	13.696	Low observed effects.	20.579	Liver, nervous and reproductive systems.	Dermal Contact
Electrical Conductivity	mS/m	70	No negative health impacts.	150	Noticeable salty taste but well tolerated.	300	Marked salty taste.	450	Extremely salty and bitter taste.	Aesthetic
Fluoride	mg/l	2.143	No negative health impacts.	2.571	No negative health impacts.	42.857	Mild dental fluorosis.	85.714	Severe effects on skeletal tissues (bones and teeth).	Dermal Contact
Glyphosate and AMPA	mg/l	4.286	No negative health impacts.	428.571	No negative health impacts.	857.143	No observed short-term effects.	2142.857	Erosion of the gastrointestinal tract.	Dermal Contact
Iron	mg/l	2.893	No negative health impacts.	5.786	No negative health impacts.	8.679	Unlikely to cause adverse effects in healthy persons.	14.464	Unlikely to cause adverse effects in healthy persons.	Dermal Contact
	mg/l	0.1	No effect.	0.3	Staining of laundry.	1	Increased staining of laundry.	10	Pronounced staining of laundry.	Physical damage.
Lead	mg/l	0.054	No negative health impacts.	0.107	No negative health impacts.	0.161	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	0.214	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Dermal Contact
Magnesium	mg/l	1	No adverse effects on taste.	2	No adverse effects on taste.	5	Taste effects are present.	10	Severe taste effects present.	Aesthetic
	mg/l	30	No effects.	70	Slight impairment of soap lathering.	100	Lathering of soap moderately impaired.	200	Lathering of soap significantly impaired.	Physical damage.
Manganese	mg/l	3	No negative health impacts.	6.429	No negative health impacts.	9.857	No observed effects.	13.286	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Dermal Contact
	mg/l	0.05	No effects.	0.1	Slight staining of white clothes.	1	Moderate staining of clothes.	5	Extreme staining.	Physical damage.
Monochloramine	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No minor health effects observed.	0	No major Health effects observed.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Mercury	mg/l	4.286	No negative health impacts.	9.857	No negative health impacts.	15.429	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	21	Mental disturbances, tremors and gingivitis, kidney failure.	Dermal Contact
Nickel	mg/l	6.593	No negative health impacts.	1648.352	No negative health impacts.	9065.934	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	16483.516	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Dermal Contact
Nitrate	mg/l	34.286	No negative health impacts.	68.571	No negative health impacts.	102.857	Poor transportation of oxygen into the blood	137.143	Congenital malformations.	Dermal Contact
Odour	TON	1	No noticeable odour.	2	Noticeable odour.	5	Strong odour objectionable to users.	10	Odour becomes stronger and more objectionable.	Aesthetic
Acidic pH	-	7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Dermal Contact
Alkaline pH	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Dermal Contact
Phenols	mg/l	0.451	No negative health impacts.	34.586	No negative health impacts.	103.759	Increasing risk of negative health effects.	139.85	Severe health effects	Dermal Contact
Selenium	mg/l	0.214	No negative health impacts.	0.643	No negative health impacts.	1.071	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	1.714	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Dermal Contact
Sodium	mg/l	100	Faintly salty taste.	200	Slightly salty taste.	600	Distinctly salty taste.	1000	Very salty taste.	Aesthetic
Sulphate	mg/l	200	Slight taste noticeable.	400	Definite salty or bitter taste.	600	Pronounced salty or bitter taste.	1000	Very strong salty and bitter.	Aesthetic
Toluene	mg/l	0.214	No negative health impacts.	5.486	No negative health impacts	859.543	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	1421.829	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Dermal Contact
Total Dissolved salts	mg/l	450	A slight taste effect may be detected.	1000	Noticeable salty taste but well tolerated.	2000	Marked salty taste.	3400	Extremely salty and bitter taste.	Aesthetic
Total Hardness	mg/l	25	A slight taste effect may be detected.	150	Noticeable taste but well tolerated.	300	Marked unpleasant taste.	600	Extremely unpleasant taste.	Aesthetic
	mg/l	100	No negative effects.	150	Slight impairment of soap lathering.	200	Soap lathering impaired.	300	Soap lathering severely impaired.	Physical damage.
Turbidity	NTU	0.1	No adverse effects.	1	Slight aesthetic effect.	5	Turbidity is visible. Slightly cloudy appearance.	10	Severe appearance, taste and odour effects.	Aesthetic
	NTU	0.1	No effects.	1	Slight staining of laundry.	5	Moderate staining of laundry.	10	Extreme staining of laundry.	Physical damage.
Uranium (238)	mg/l	0.129	No negative health impacts.	0.643	No negative health impacts.	1.157	No observed effects.	1.8	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Dermal Contact
Xylene	mg/l	8.571	No negative health impacts.	7671.429	No negative health impacts.	14550	Disturbances of cognitive abilities, balance, and coordination.	21428.571	Damage to the central nervous system, liver and kidneys.	Dermal Contact
Zinc	mg/l	6.429	No negative health impacts.	10.779	No negative health impacts.	15.129	Stomach cramps, nausea and vomiting.	19.5	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Dermal Contact

Household Use Report Sheet

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Acrylamide	mg/l	330.33	No negative health impacts.	660.659	No negative health impacts.	990.989	Confusion, disorientation, memory disturbances and hallucinations.	1321.319	Skin irritation, fatigue, foot weakness and sensory changes.	Dermal Contact
Ammonia	mg/l	21.429	No negative health impacts.	210	No negative health impacts.	398.571	Influences metabolism.	587.143	Cells mutagenicity.	Dermal Contact
Antimony	mg/l	0.017	No negative health impacts.	0.159	No negative health impacts.	0.857	Distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and is excreted in faeces and urine.	15	Respiratory and eye problems, staining of tooth surface.	Dermal Contact
Arsenic	mg/l	0.297	No negative health impacts.	0.791	No negative health impacts.	84.462	Vomiting, abdominal pain and diarrhoea.	168.132	Confirmed carcinogenic, numbness and tingling of the extremities, muscle cramping, death.	Dermal Contact
Asbestos	mg/l	148.352	No negative health impacts.	296.703	No negative health impacts.	445.055	Bronchial diseases/illnesses.	593.407	Asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx.	Dermal Contact
Atrazine	mg/l	75	No negative health impacts.	150	No negative health impacts.	610.714	Lowering of the immune system.	1071.429	Affect neuroendocrine function, leading to disruption of the oestrous cycle or developmental effects.	Dermal Contact
Barium	mg/l	4.286	No negative health impacts.	4.5	No negative health impacts.	4.714	Vomiting, abdominal cramps, and watery diarrhoea are typically reported shortly after ingestion.	4.929	Cardiovascular (hypertension) effects, toxic.	Dermal Contact
Benzene	mg/l	0.007	No negative health impacts.	0.013	No negative health impacts.	0.02	Impacts central nervous system causing dizziness, nausea, vomiting, headache and drowsiness.	0.027	Pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia, death.	Dermal Contact
Benzo(a)pyrene	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Red blood cell damage, leading to anaemia; suppressed immune system.	0	Affects foetal development and Reproductive organs in females.	Dermal Contact
Boron	mg/l	8.571	No negative health impacts.	17.143	No negative health impacts.	25.714	Irritation of the eye, the upper respiratory tract, and the nasopharynx.	34.286	Effects on the reproductive system, boron poisoning, central nervous system stimulation, depression, skin eruptions.	Dermal Contact
Bromide	mg/l	30	No negative health impacts.	300	No negative health impacts.	570	Nausea and vomiting (gastrointestinal symptoms).	840	No major long-term health effects observed.	Dermal Contact
Cadmium	mg/l	0.165	No negative health impacts.	0.33	No negative health impacts.	0.495	Osteomalacia, nausea, vomiting, diarrhoea, muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.	0.659	Kidney dysfunction, kidney, liver, bone and blood damage.	Dermal Contact
Calcium	mg/l	0.043	No negative health impacts.	0.086	No negative health impacts.	0.129	No major effects on human health.	0.171	No conclusive evidence.	Dermal Contact
Carbon tetrachloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	Impacts central nervous system, depression, and liver and kidney toxicity.	0	Damage/toxicity of the liver and kidneys.	Dermal Contact
Chloride	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	0	No negative health impacts.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Chlorine	mg/l	4.286	No negative health impacts.	6.171	No negative health impacts.	8.057	Acts as an Oestrogen Mimic, Causes Weight Gain.	9.943	Increased Risk of Cancer, asthmatic attacks, destroys cells and tissues inside our body.	Dermal Contact
Chloroform	mg/l	6.593	No negative health impacts.	329.67	No negative health impacts.	4417.582	Cardiac arrhythmias and abnormalities of the liver and kidneys.	8505.495	Degrades blood, liver, and kidney.	Dermal Contact
Chromium (VI)	mg/l	0.989	No negative health impacts.	6.264	No negative health impacts.	13.902	Gastrointestinal disorders, haemorrhagic diathesis, and convulsions.	16.484	Genotoxic effects, lung cancer.	Dermal Contact
Colour	TCU	1	No visual effects.	5	No visual effects.	12	No visual effects.	15	Taste and staining effects due to the presence of iron or manganese.	Aesthetic
Copper	mg/l	0.3	No negative health impacts.	0.583	No negative health impacts.	57.077	Vomiting, diarrhoea, stomach cramps, and nausea.	113.571	Liver damage and kidney disease.	Dermal Contact
	mg/l	0	No negative impacts.	0	No negative impacts.	0	No negative impacts.	0	No negative impacts.	Physical damage.
Cyanide	mg/l	0.027	No negative health impacts.	0.054	No negative health impacts.	0.081	Lower vitamin B12 levels and hence exacerbate vitamin B12 deficiency.	0.108	Chronic effects on thyroid and nervous system.	Dermal Contact
Dissolved Organic Carbon	mg/l	1	No negative health impacts.	5	Slight taste odour, and colour effect.	10	Significant taste, colour and odour effects.	20	Severe taste, colour and odour effects.	Aesthetic
DDT and metabolites	mg/l	0.069	No negative health impacts.	6.882	No negative health impacts.	13.696	Low observed effects.	20.579	Liver, nervous and reproductive systems.	Dermal Contact
Electrical Conductivity	mS/m	70	No negative health impacts.	150	Noticeable salty taste but well tolerated.	300	Marked salty taste.	450	Extremely salty and bitter taste.	Aesthetic
Fluoride	mg/l	2.143	No negative health impacts.	2.571	No negative health impacts.	42.857	Mild dental fluorosis.	85.714	Severe effects on skeletal tissues (bones and teeth).	Dermal Contact
Glyphosate and AMPA	mg/l	4.286	No negative health impacts.	428.571	No negative health impacts.	857.143	No observed short-term effects.	2142.857	Erosion of the gastrointestinal tract.	Dermal Contact
Iron	mg/l	2.893	No negative health impacts.	5.786	No negative health impacts.	8.679	Unlikely to cause adverse effects in healthy persons.	14.464	Unlikely to cause adverse effects in healthy persons.	Dermal Contact
	mg/l	0.1	No effect.	0.3	Staining of laundry.	1	Increased staining of laundry.	10	Pronounced staining of laundry.	Physical damage.
Lead	mg/l	0.054	No negative health impacts.	0.107	No negative health impacts.	0.161	Behaviour and learning problems, lower IQ and hyperactivity, slowed growth, hearing problems, anaemia.	0.214	Neurodevelopment effects, impaired renal function, adverse pregnancy outcomes.	Dermal Contact
Magnesium	mg/l	1	No adverse effects on taste.	2	No adverse effects on taste.	5	Taste effects are present.	10	Severe taste effects present.	Aesthetic
	mg/l	30	No effects.	70	Slight impairment of soap lathering.	100	Lathering of soap moderately impaired.	200	Lathering of soap significantly impaired.	Physical damage.
Manganese	mg/l	3	No negative health impacts.	6.429	No negative health impacts.	9.857	No observed effects.	13.286	Manganism, lethargy, increased muscle tone, tremor and mental disturbances.	Dermal Contact
	mg/l	0.05	No effects.	0.1	Slight staining of white clothes.	1	Moderate staining of clothes.	5	Extreme staining.	Physical damage.
Monochloramine	mg/l	0	No negative health impacts.	0	No negative health impacts.	0	No minor health effects observed.	0	No major Health effects observed.	Dermal Contact
Mercury	mg/l	4.286	No negative health impacts.	9.857	No negative health impacts.	15.429	Pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock.	21	Mental disturbances, tremors and gingivitis, kidney failure.	Dermal Contact

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description	Most sensitive exposure route
Nickel	mg/l	6.593	No negative health impacts.	1648.352	No negative health impacts.	9065.934	Nausea, vomiting, abdominal discomfort, diarrhoea, visual disturbance, headaches, giddiness, and coughing.	16483.516	Nickel allergy (contact dermatitis), lung fibrosis, cardiovascular and kidney diseases, cancer of the respiratory tract.	Dermal Contact
Nitrate	mg/l	34.286	No negative health impacts.	68.571	No negative health impacts.	102.857	Poor transportation of oxygen into the blood	137.143	Congenital malformations.	Dermal Contact
Odour	TON	1	No noticeable odour.	2	Noticeable odour.	5	Strong odour objectionable to users.	10	Odour becomes stronger and more objectionable.	Aesthetic
Acidic pH	-	7	No negative health impacts.	6.8	No negative health impacts.	6.5	No observed effects.	6	No observed effects.	Dermal Contact
Alkaline pH	-	7	No negative health impacts.	7.2	No negative health impacts.	7.5	Irritate eyes, skin and mucous membranes, gastrointestinal problems.	8	No observed long-term effects.	Dermal Contact
Phenols	mg/l	0.451	No negative health impacts.	34.586	No negative health impacts.	103.759	Increasing risk of negative health effects.	139.85	Severe health effects	Dermal Contact
Selenium	mg/l	0.214	No negative health impacts.	0.643	No negative health impacts.	1.071	Gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.	1.714	Hair and fingernail loss, damage to kidney and liver tissue, and the nervous and circulatory systems.	Dermal Contact
Sodium	mg/l	100	Faintly salty taste.	200	Slightly salty taste.	600	Distinctly salty taste.	1000	Very salty taste.	Aesthetic
Sulphate	mg/l	200	Slight taste noticeable.	400	Definite salty or bitter taste.	600	Pronounced salty or bitter taste.	1000	Very strong salty and bitter.	Aesthetic
Toluene	mg/l	0.214	No negative health impacts.	5.486	No negative health impacts	859.543	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	1421.829	Impairment of central nervous system, irritation of mucous membranes. Fatigue and drowsiness.	Dermal Contact
Total Dissolved salts	mg/l	450	A slight taste effect may be detected.	1000	Noticeable salty taste but well tolerated.	2000	Marked salty taste.	3400	Extremely salty and bitter taste.	Aesthetic
Total Hardness	mg/l	25	A slight taste effect may be detected.	150	Noticeable taste but well tolerated.	300	Marked unpleasant taste.	600	Extremely unpleasant taste.	Aesthetic
	mg/l	100	No negative effects.	150	Slight impairment of soap lathering.	200	Soap lathering impaired.	300	Soap lathering severely impaired.	Physical damage.
Turbidity	NTU	0.1	No adverse effects.	1	Slight aesthetic effect.	5	Turbidity is visible. Slightly cloudy appearance.	10	Severe appearance, taste and odour effects.	Aesthetic
	NTU	0.1	No effects.	1	Slight staining of laundry.	5	Moderate staining of laundry.	10	Extreme staining of laundry.	Physical damage.
Uranium (238)	mg/l	0.129	No negative health impacts.	0.643	No negative health impacts.	1.157	No observed effects.	1.8	Non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity.	Dermal Contact
Xylene	mg/l	8.571	No negative health impacts.	7671.429	No negative health impacts.	14550	Disturbances of cognitive abilities, balance, and coordination.	21428.571	Damage to the central nervous system, liver and kidneys.	Dermal Contact
Zinc	mg/l	6.429	No negative health impacts.	10.779	No negative health impacts.	15.129	Stomach cramps, nausea and vomiting.	19.5	Anaemia, nervous system disorders, damage to the pancreas and lowered levels of “good” cholesterol.	Dermal Contact

Appliances Constituents Report Sheet

Constituent	Units	Ideal	Risk Description	Acceptable	Risk Description	Tolerable	Risk Description	Unacceptable	Risk Description
Calcium	mg/l	32	No scaling effects on appliances and equipment.	50	Increased scaling on appliances.	80	High degree of scaling	150	Severe scaling.
Chloride	mg/l	50	No negative effects.	200	Noticeable increase in corrosion rates in appliances.	600	Likelihood of rapid corrosion in appliances.	1200	Very corrosive to appliances.
Electrical Conductivity	mg/l	70	No effects on appliances and/or equipment.	150	Slight scaling or corrosion of appliances and piping may be expected.	300	Increasing scaling and corrosion of appliances and/or equipment.	450	Corrosive and increased scaling of appliances and/or equipment.
Iron	mg/l	0.1	No effect.	0.3	Staining of fixtures; slimy coatings on plumbing equipment.	1	Increased staining of fixtures, increased problems with plumbing.	10	Pronounced staining of fixtures, significant effects on plumbing.
Magnesium	mg/l	30	Slight scaling problems.	70	Scaling problems encountered (especially together with calcium).	100	Increased scaling problems.	200	Severe scaling problems.
Acidic pH	-	0	No negative effects.	0	No negative effects.	0	No negative effects.	0	No negative effects.
Alkaline pH	-	0	No negative effects.	0	No negative effects.	0	No negative effects.	0	No negative effects.
Total Dissolved Salts	mg/l	450	No effects on appliances and/or equipment.	1000	Slight scaling or corrosion of appliances and piping may be expected.	2000	Increasing scaling and corrosion of appliances and/or equipment.	3000	Corrosive and increased scaling of appliances and/or equipment.
Total Hardness	mg/l	100	No negative effects.	150	Slight scaling of appliances.	200	Moderately hard (some scaling of appliances).	300	Hard (scaling of appliances).

Gardening Report Sheet

	Fitness-for-use	Relative crop yield (%)	% of time yield is within relative crop yield category, as affected by:			
			Salinity (EC)	Boron	Chloride	Sodium
ROOT ZONE EFFECTS	Ideal	90-100	<57	< 0.4	< 208	< 2.99
	Acceptable	80-90	57-75	0.4-0.67	208-269	2.99-3.27
	Tolerable	70-80	75-92	0.67-0.93	269-331	3.27-3.54
	Unacceptable	<70	>92	> 0.93	> 331	> 3.54

			Irrigation water concentration that may cause the corresponding degree of leaf scorching under sprinkler irrigation	
LEAF SCROCHING BY Cl AND Na	Fitness-for-use	Degree of leaf-scorching	Chloride (Cl) (mg/L)	Sodium (Na) (mg/L)
	Ideal	None	<70	<50
	Acceptable	Slight	70-135	50-83
	Tolerable	Moderate	135-180	83-115
	Unacceptable	Severe	>180	>115

	Fitness-for-use	Excess infections per 1000 people p.a.	Irrigation water concentration predicted to give rise to the indicated excess infections per 1000 persons p.a. (*E. coli* counts per 100 mL)
MIRCOBIAL CONTAMINATION	Ideal	<1	<351
	Acceptable	1-3	351-1052
	Tolerable	3-10	1052-3506
	Unacceptable	>10	>3506