Australian Government
Department of Health and Aged Care
Australian Industrial Chemicals Introduction Scheme

Nickel sulfide (NiS)

Assessment statement (CA09926)

20 February 2025

Table of contents

AICIS assessment (CA09926)

Chemical in this assessment

Name	CAS registry number
Nickel sulfide (NiS)	16812-54-7

Reason for the assessment

An application for an assessment certificate under section 31 of the *Industrial Chemicals Act 2019* (the Act).

Certificate application type

AICIS received the application in a Health and Environment Focus type.

Defined scope of assessment

The chemical has been assessed:

- as imported into Australia at up to 10 tonnes once during the five-year period
- as imported in finished end use solid products containing the assessed chemical at up to 30% concentration as a component of a processing agent (catalyst) for use in the petroleum refining industry (production of fuels)
- for use as imported by industrial and professional workers only

Summary of assessment

Summary of introduction, use and end use

The assessed chemical will not be manufactured, reformulated, or re-packaged in Australia. It will be imported into Australia at up to 10 tonnes once during the five years period, packed in sealed 200 L steel drums. The finished end use products containing the assessed chemical at up to 30% concentration, will be transported from the port to the refinery by road and will be temporarily stored at this site in an isolated area until further deployment into the refinery reactors.

The products containing the assessed chemical at up to 30% concentration will not be available to the public and will only be used by specialised catalyst loading contractors at one industrial site as a processing agent for use in the production of fuels.

Human health

Summary of health hazards

The submitted toxicological data on the assessed chemical and analogue chemicals (see **Supporting information** section) indicate that the assessed chemical is:

- of low acute oral toxicity (LD50 > 5,000 mg/kg bw in rats)
- toxic if inhaled (LC50 = 0.67 mg/L in female rats)
- slightly irritating to the skin and eyes
- a skin sensitiser

An analogue chemical (nickel sulfate hexahydrate) caused statistically significant reductions in body weights of rats at 30 mg/kg bw/d in a two-year study. The No Observable Adverse Effect Level (NOAEL) was 10 mg/kg bw/day (equivalent to 2.2 mg Ni/kg bw/day).

No dermal or inhalation repeated dose toxicity data were provided for the assessed chemical.

Information was not available on the repeated dose inhalation toxicity on the assessed chemical. However, based on data available for an analogue chemical (nickel subsulfide) a lowest observed adverse effect concentration (LOAEC) of 0.11 mg/m^3 nickel for lung toxicity was reported in a two-year study (NTP, 1996; NICNAS IMAP report, 2014). Based on the information on the analogue chemical, the assessed chemical is classified as H372: Causes damage to organs through prolonged or repeated exposure through inhalation.

Published limited data from a review report indicate that the assessed chemical has the potential to cause genotoxicity, including DNA damage. Therefore, the assessed chemical is classified as a Category 2 mutagen, according to the GHS criteria (Ni institute, 2024).

Furthermore, nickel compounds such as nickel subsulfide, nickel sulfate and nickel chloride have also been classified as Category 1A carcinogenic substances and Category 2 germ cell mutagens with the hazard statements of ‘May cause cancer by inhalation’ and ‘Suspected of causing genetic defects’, respectively (HCIS Safe Work Australia, 2025).

The International Agency for Research on Cancer (IARC) has also classified nickel compounds as 'Carcinogenic to humans' (Group 1). IARC has highlighted studies showing an increased risk of lung cancer from inhalation exposure to nickel compounds, although these were often mixed exposures, either with soluble nickel or oxidic nickel compounds (IARC, 2012).

Hazard classifications relevant for worker health and safety

Based on the data provided by the applicant and other information available to AICIS, the assessed chemical satisfies the criteria for classification according to the *Globally Harmonized System of Classification and Labelling of Chemicals* (GHS) (UNECE 2017) for hazard classes relevant for worker health and safety as adopted for industrial chemicals in Australia.

Health hazards	Hazard category	Hazard statement
Acute toxicity - inhalation	Category 3	H331: Toxic if inhaled
Skin Sensitisation	Category 1	H317: May cause an allergic skin reaction

Genotoxicity	Category 2	H341: Suspected of causing genetic defects
Carcinogenicity	Category 1A	H350i: May cause cancer via inhalation
Specific target organ toxicity (repeated exposure)	Category 1	H372: Causes damage to organs through prolonged or repeated exposure through inhalation

Summary of health risk

Public

The products containing the assessed chemical at up to 30% concentration will not be available for use by the public. When introduced and used in the proposed manner, it is unlikely that the public will be exposed to the assessed chemical.

This assessment does not identify any risks to the public health that require specific risk management measures.

Workers

Limited occupational exposure is expected to the assessed chemical at 30% concentration during use in the production of fuels, including handling at the end of usable life (approximately 5 years). According to the applicant, specialised catalyst loading contractors, special engineering controls and the personal protective equipment (PPE) will be used during these procedures.

Considering the adverse health effects possible through exposure to the assessed chemical, control measures to minimise inhalation, dermal and ocular exposure are needed to manage the risk to workers (see **Means for managing risk** section).

Environment

Summary of environmental hazard characteristics

As the assessed chemical is inorganic, it is excluded from categorisation under the *Australian Environmental Criteria for Persistent, Bioaccumulative and/or Toxic Chemicals* (DCCEEW, 2022).

Environmental hazard classification

A dissolution test conducted on the assessed chemical following OECD TG 105 Shake flask method, coupled with gravimetric determination, showed that soluble nickel was detected. Therefore, the hazard classification for the assessed chemical is based on soluble nickel data. Accordingly, the assessed chemical satisfies the criteria for classification according to the GHS (UNECE, 2017) as Acute Category 1 (H400) and Chronic Category 1 (H410).

Environmental Hazard	Hazard Category	Hazard Statement
Hazardous to the aquatic environment (acute / short-term)	Aquatic Acute 1	H400: Very toxic to aquatic life
Hazardous to the aquatic environment (long-term)	Aquatic Chronic 1	H410 - Very toxic to aquatic life with long lasting effects

Summary of environmental risk

The assessed chemical will be introduced into Australia as a component of solid catalysts for use in the petroleum refining industry.

No environmental exposures of the assessed chemical are expected during manufacture, use or end of life disposals.

A Risk Quotient (PEC/PNEC) for the aquatic compartment was not calculated as the currently available information indicates the assessed chemical will not be released to the environment, untreated. Therefore, it is expected that the environmental risk from the introduction of the assessed chemical can be managed.

Means for managing risk

Recommendation to Safe Work Australia

- It is recommended that Safe Work Australia (SWA) update the *Hazardous Chemical Information System* (HCIS) to include classifications relevant to work health and safety (see **Hazard classifications relevant for worker health and safety**).

Information relating to safe introduction and use

The information in this statement, including recommended hazard classifications, should be used by a person conducting a business or undertaking at a workplace (such as an employer) to determine the appropriate controls under the relevant jurisdiction Work Health and Safety laws.

The following control measures could be implemented to manage the risk arising from exposure to the assessed chemical during loading and unloading:

- Use of engineering controls such as
  - Enclosed and automated systems
  - Adequate workplace ventilation to avoid accumulation of dust or mist
- Use of safe work practices to
  - Avoid contact with skin and eyes
  - Avoid inhalation of dusts or mist
- Use of personal protective equipment (PPE)
  - Impervious gloves
  - Protective clothing
  - Eye protection
  - Respiratory protection

- The storage of the assessed chemical should be in accordance with the Safe Work Australia *Code of Practice for Managing Risks of Hazardous Chemicals in the Workplace* (SWA 2023) or relevant State or Territory Code of Practice.

- As the assessed chemical is a skin sensitiser, the control measures may need to be supplemented with health monitoring for any worker who is at significant risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health.

- A copy of the Safety Data Sheet (SDS) should be easily accessible to workers.

Conclusions

The Executive Director is satisfied that the risks to human health or the environment associated with the introduction and use of the industrial chemical can be managed.

Note:

1. Obligations to report additional information about hazards under s 100 of the *Industrial Chemicals Act 2019* apply.
2. You should be aware of your obligations under environmental, workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Supporting information

Chemical identity

CAS number	16812-54-7
CAS name	Nickel sulfide (NiS)
Molecular formula	NiS
Associated names	Nickel monosulfide Nickel(2+) sulfide Nickel(II) sulfide
Molecular weight (g/mol)	90.76
SMILES (canonical)	S=[Ni]
Representative structure	

Relevant physical and chemical properties

Physical form	Black solid powder
Melting point	> 302 °C
Density	5,314.2 kg/m^3 at 26.09 °C
Water solubility	16.39 mg/L at 20°C
Relative Auto flammability	> 720 °C
Flammability	Not highly flammable
Particle Size	X10 = 6.45 µm X50 = 104.77 µm X90 = 305.30 µm < 2.5 µm = 3.61% < 5 µm = 7.97% < 10 µm = 14.08% Mass median aerodynamic diameter (SMD) = 18.71 µm
Ionisable in the environment	Yes

Human exposure

Workers

At the refinery reactors, the solid products containing the assessed chemical at up to 30% concentration will be loaded from the steel drums into enclosed reactors. According to the applicant, this process will be completed by specialised catalyst loading contractors using refilling equipment under an inert atmosphere with sufficient personal protections as per the well documented site procedures. Special engineering controls such as isolated, automated, specialised processes, inert atmospheric environment, and atmospheric monitoring will be in place. The specialised loading contractors will use the PPE including hazmat life support suits and self-contained respiratory systems during handling of the assessed chemical to minimise worker exposure to the assessed chemical.

Once the assessed chemical is loaded into the reactors, the assessed chemical at up to 30% concentration is expected to remain in enclosed systems over a period of approximately 5 years and will not be available for exposure during the normal use. At the end of usable life, the used product containing the assessed chemical will be unloaded into appropriate containers by specialised contractors using gravity and maintained under inert atmosphere. The used product will then be sent offsite for regeneration or disposed of according to relevant Commonwealth, state, territory and local government legislation. Fresh product containing the assessed chemical at up to 30% concentration will be reloaded into the reactor.

Exposure to the assessed chemical at up to 30% concentration in solid form may be possible during opening of the steel drums containing the assessed chemical at up to 30% concentration, loading to reactor vessels using refilling equipment, bale packing of used bags and cleaning and maintenance processes. However, as mentioned above, exposure to the assessed chemical at up to 30% concentration will be minimised through the use of specialised catalyst loading contractors, special engineering controls and the use of PPE.

Health hazard information

Acute toxicity

Oral

In an acute oral toxicity study (Up and down procedure, OECD TG 425), the assessed chemical was initially administered via oral gavage to a single female Sprague Dawley (SD) rat at 175 mg/kg bw. Five additional animals were dosed at 550, 1,750, and 5,000 mg/kg bw. Animals were observed once daily for 14 days for behaviour changes, signs of gross toxicity and for mortality. All animals gained body weight, appeared active and survived during the study.

There were no gross abnormalities noted for the animals when necropsied at the conclusion of the 14-day observation period at all doses. No signs of systemic toxicity were also noted for all animals at all doses except the only animal dosed at 550 mg/kg bw exhibited irregular respiration, piloerection and ataxia following administration of the assessed chemical. However, the animal recovered by Day 1 and appeared active and healthy for the remainder of the observation period. The acute oral LD50 value for the assessed chemical was determined to be > 5,000 mg/kg bw. Therefore, the assessed chemical is considered of low acute oral toxicity.

Inhalation

In an acute inhalation toxicity study (OECD TG 403), SD rats (n = 10/sex/group) were exposed to the aerosolised assessed chemical in dust form (nose-only exposure) at 0.06, 0.52, 1.21 and 5.21 mg/L for 4 hours, with mass median aerodynamic diameter at up to 3.5 µm of all tested concentrations. Animals were observed for 14 days following exposure. While all animals exposed to 0.06 mg/L showed irregular respiration, two males and six females were also hypoactive in the 0.52 mg/L exposure group. In the 1.21 mg/L exposure group, prior to death, the animals were hypoactive, exhibited irregular respiration, ano-genital staining, facial staining, and were cold to touch. In the 5.21 mg/L exposure group, prior to death, four males and nine females were hypoactive and all animals exhibited irregular respiration, ano-genital staining, facial staining, and were cold to touch. Four surviving males exhibited similar signs.

All animals survived in the 0.06 mg/L exposure group. While one female died within four days in the 0.52 mg/L exposure group, six males and all ten females died within ten days in the 1.21 mg/L exposure group. Six males and all ten females also died or were euthanized for humane reasons within nine days in the 5.21 mg/L exposure group. Gross necropsy of the dead/killed animal in the top three treatment groups revealed discolouration of the lungs, mottled liver, distention or discoloration of the intestines, and/or white frothy liquid in the trachea. In addition, red fluid in the intestine and/or mottled spleen were also noted in the top two treatment groups.

Under the conditions of this study, the acute inhalation LC50 of assessed chemical was determined to be 0.67 mg/L of air in female rats by the study authors, as calculated by Probit Analysis. As the data did not permit the calculation of the LC50 for males by Probit Analysis, the LC50 for male rats was reported as between 0.52 and 1.21 mg/L of air.

Based on the above information, the assessed chemical is classified as toxic if inhaled, according to GHS criteria (Category 3, H331: Toxic if inhaled).

Corrosion/Irritation

Skin irritation

In a skin irritation study (OECD TG 404), an analogue chemical (nickel subsulfide 85% concentration w/w) was applied under semi-occlusive conditions to the shaved skin of 3 New Zealand albino female rabbits for 4 hours. Animals were observed for 72 hours after patch removal. There was no oedema observed at any treated site during this study. Application sites showed signs of very slight erythema for two of the three treated sites within 1 hour of patch removal. Both animals were free from dermal irritation by 72 hours. Under the conditions of this study, the analogue chemical was slightly irritating to the skin and therefore, the assessed chemical is considered as slightly irritating to the skin.

Eye irritation

In an eye irritation study (OECD TG 405), an analogue chemical (0.1 g of nickel subsulfide) was instilled into the conjunctival sac of right eye of 3 female New Zealand White rabbits and the animals were observed for 72 hours. One hour after test substance instillation, all treated eyes exhibited corneal opacity, iritis and conjunctivitis which resolved within 72 hours. Under the conditions of this study, the analogue chemical was determined to be a slight eye irritant in rabbits and therefore, the assessed chemical is considered as slightly irritating to eyes.

Sensitisation

Skin sensitisation

The applicant has not provided any study on skin sensitisation for the assessed chemical or an analogue chemical. However, the applicant provided a published article which indicated that Nickel (metal) remains the most identified contact allergen (Basketter, 2021). The article also stated that “it has proven difficult to demonstrate significant skin-sensitising activity for nickel in toxicology tests, which typically have indicated a weak skin sensitisation potential”. Furthermore, due to lack of human-specific mechanistic route for nickel sensitisation that animals lack, both *in vivo* and *in vitro* assays are not predictive of dermal sensitisation hazard or potency for nickel. It also noted that *in silico* methods are not designed for metal allergens that can contribute to the analysis for metal allergens.

Therefore, based on the above information and that nickel metal is also classified as Category 1 skin sensitiser (HCIS Safe Work Australia, 2025), the assessed chemical is considered a Category 1 skin sensitiser, with a hazard classification for skin sensitisation (H317: Category 1 May cause an allergic skin reaction).

Repeat dose toxicity

Oral

In an oral carcinogenicity study (OECD TG 451), an analogue chemical (nickel sulfate hexahydrate) was administered by oral gavage to Fischer 344 rats (n = 60/sex/dose) for 2 years. Based on the findings from a 90-day range-finding study, exposure levels of 0 (control), 10 mg/kg bw/day (low dose), 30 mg/kg bw/day (medium dose) and 50 mg/kg bw/day (high dose) were selected.

There were no treatment-related clinical abnormalities observed in any group during this study. While a variety of clinical signs were noted during the two-year study period, the type and incidence of clinical signs observed in the treatment groups were generally comparable to those observed in the control group.

A few statistically significant differences in the haematology data were observed in males and females in the treatment groups. However, none of these differences were suggestive of a hyperplastic (i.e., leukemic) response and none of these changes were considered toxicologically meaningful since they were small and did not follow a consistent dose-related pattern.

There was no apparent treatment-related effect on mortality in males over the two-year period. Similar mortality rates were observed across control and treated groups (i.e., 60%, 48%, 50% and 57% for control, 10 mg/kg bw/day, 30 mg/kg bw/day and 50 mg/kg bw/day groups, respectively). Survival in the females was statistically reduced in a dose-related manner at the 30 (43% mortality) and 50 (50% mortality) mg/kg/day dose levels as compared to study controls (23% mortality).

Male rats showed a reduction in body weight gain in a dose-related manner at the 10, 30 and 50 mg/kg bw/day, reduction being approximately 5%, 11% and 12% respectively. A lesser level of reduction in body weight gain was also noted in female rats, reduction being approximately 4%, 8% and 10% respectively. The reduced body weight gain reached the level of biological significance (i.e., > 10% decrease) in the males at 30 and 50 mg/kg bw/day and in females at 50 mg/kg bw/day. This significant weight reduction indicates that the maximum tolerated dose

was reached in this study for both males and females. The reduced weight gain was not accompanied by similar reductions in food consumption in male and female animals.

Several gross necropsy findings observed in the treatment groups were comparable to those observed in the control group and consistent with findings commonly seen in aging rats on a long-term study. Similarly, microscopic findings observed in the treatment groups were not considered to be related to the treatment with the analogue chemical and were either secondary to toxicity or incidental backgrounds.

Under the conditions of this study and based on the effects on body weight in male animals, the NOAEL for the analogue chemical was 10 mg/kg bw/day, equivalent to 2.2 mg Ni/kg bw/day.

Inhalation

No data are available for the assessed chemical.

In a two-year study conducted with F344 rats, 63 males and 63 females were exposed to 0, 0.15 or 1 mg/m^3 nickel subsulfide (equivalent to 0, 0.11 or 0.72 mg/m^3 nickel, respectively) for six hours/day, five days a week for two years. Exposure to nickel subsulfide did not affect survival when compared with the controls. Observable adverse effects included rapid and shallow breathing following each exposure period. Lung-specific effects included a significant increase in lung weights compared with controls across all exposure groups when assessed at seven and 15 months. Non-neoplastic lung pathology included fibrosis, chronic active inflammation in the lung and bronchial lymph node hyperplasia, which was significantly increased in males and females at an exposure concentration ≥ 0.15 mg/m^3. There was also a significant increase in atrophy of the olfactory epithelium in males and females and chronic active inflammation in the nose of females at the highest exposure dose (NTP, 1996; NICNAS IMAP report, 2014).

In the two-year study conducted with B6C3F1 mice, 80 males and 80 females were exposed to 0, 0.6 or 1.2 mg/m^3 nickel subsulfide (equivalent to 0, 0.44, 0.88 mg/m^3 of nickel, respectively) under the same conditions as above. Exposure to nickel subsulfide did not affect survival when compared with the controls. Observable adverse effects included rapid and shallow breathing following each exposure period. Lung-specific effects included a significant increase in lung weights compared with controls across all exposure groups when assessed at seven and 15 months. Non-neoplastic lung pathology included fibrosis, chronic active inflammation in the lung and bronchial lymph node hyperplasia, which was significantly increased in males and females at an exposure concentration of ≥ 0.6 mg/m^3. Similar to the study conducted in F344 rats as above, there was a significant increase in atrophy of the olfactory epithelium in males and females and a significant increase in acute inflammation and degeneration of the olfactory epithelium in females across both exposure concentrations, respectively (NTP, 1996; NICNAS IMAP report, 2014).

Based on the two-year studies in F344 rats, a LOAEC of 0.11 mg/m^3 nickel for non-neoplastic lung toxicity was reported.

Based on the data available as summarised above for an analogue chemical (nickel subsulfide) (NICNAS IMAP report, 2014), the assessed chemical is classified as causing damage to organs through prolonged or repeated exposure through inhalation, according to the GHS criteria (STOT rep. exp. 1, H372: Causes damage to organs through prolonged or repeated exposure through inhalation). This classification was based on the established LOAEC of 0.11 mg/m^3 nickel (analogue chemical) for lung toxicity reported in a two year study (NTP, 1996; NICNAS IMAP report, 2014).

Genotoxicity

In a review of mutagenicity of the assessed chemical, many *in vitro* genetic toxicity studies were identified characterising the genetic toxicity of the assessed chemical (Ni institute, 2024). Genotoxicity effects reported included chromosomal aberrations, morphological transformation, DNA strand breakage, and mutation frequency. Most studies clarified the type of NiS (i.e., crystalline, amorphous) assessed in the study and several evaluated the comparative genotoxicity of multiple types.

In conclusion, the review reported that the assessed chemical has the potential to cause genotoxicity, including DNA damage, under laboratory conditions in a number of cell lines. However, in a number of these studies, negative and positive controls were not incorporated, nor were statistical analyses implemented to determine the significance of findings. The review also stated that *in vivo* studies with the assessed chemical have not been found. Taken together with the results of *in vivo* studies conducted with analogue chemical compounds, the weight of the evidence indicated that a genotoxic potential should be considered for the assessed chemical and supports classification of the assessed chemical as a Category 2 mutagen (H341: Suspected of causing genetic defects).

Carcinogenicity

No data are available for the assessed chemical. However, nickel subsulfide is classified as hazardous, Category 1 with the risk phrase ‘May cause cancer by inhalation’ (HCIS Safe Work Australia, 2025). IARC has classified nickel compounds as 'Carcinogenic to humans' (Group 1).

In a two-year study conducted with F344 rats, 63 males and 63 females were exposed to 0, 0.15 or 1 mg/m^3 of an analogue chemical (nickel subsulfide) (equivalent to 0, 0.11 or 0.72 mg/m^3 nickel, respectively) for six hours/ day, five days a week for two years. There was a significant increase in the number of alveolar/bronchiolar adenomas or carcinomas in male and female F344 rats (1 mg/m^3) after two years of exposure. Furthermore, there was a significant increase in the number of bilateral neoplastic lesions of the adrenal medulla in male (≥ 0.15 mg/m^3) and female rats (≥ 1 mg/m^3) exposed to nickel subsulfide. However, compared with the two-year study in rats, the two-year study in B6C3F1 mice exposed to 0, 0.6 or 1.2 mg/m^3 nickel subsulfide (equivalent to 0, 0.44, 0.88 mg/m^3 of nickel, respectively) did not report a significant increase in the number of alveolar/bronchiolar adenomas or carcinomas in males or females after two years of exposure (NTP, 1996; NICNAS IMAP report, 2014).

There are limited data with respect to epidemiological studies on the assessed chemical or on the analogue chemical (nickel subsulfide) (NICNAS IMAP report, 2014). IARC has highlighted studies with an increased risk of lung cancer from exposure to nickel subsulfide, although these were often mixed exposures, either with soluble nickel or oxidic nickel compounds. A cohort of nickel plant (Clydach—United Kingdom) cleaners exposed to insoluble nickel compounds (oxidic and sulfidic mainly) were reported to have a high rate of lung cancer. This study was taken as sufficient evidence of carcinogenicity in humans of nickel subsulfide (IARC, 2012; NICNAS IMAP report, 2014).

Therefore, based on the above information, the assessed chemical is classified as a Category 1A carcinogen via inhalation, according to the GHS criteria (H350i: May cause cancer via inhalation).

Environmental exposure

Exposures to the environment during manufacturing, reformulation or re-packaging processes are not expected. The catalysts containing the assessed chemical will be imported into Australia as fully finished products containing the assessed chemical and will be transported from port to the refinery by road. It may be temporarily stored at refinery sites until it is deployed into refinery reactors. No environmental release is expected during these processes.

In the unlikely event of accidental spills or leaks during storage, transport, activation and loading, the assessed chemical is expected to be collected for re-use or metal reclamation to the extent practicable.

Environmental exposures during loading the catalyst containing the assessed chemical into reactors are also expected to be minimal, as specialised procedures will be in place during these processes.

Once the catalysts are loaded into the reactors, the assessed chemical is expected to remain in the enclosed systems over a period of approximately 5 years and is not expected to have any environmental release during use.

The used catalysts will be sent offsite for regeneration or disposed of according to relevant Commonwealth, state, territory and local government legislation, or exported for metal reclamation processes. Any potential disposal to landfill requires prior consent from appropriate local, State and Federal government authorities.

Environmental fate

Dissolution, speciation and partitioning

The behaviour of the nickel (2^+) ion is strongly dependent on the chemistry of the environmental compartment into which it is released.

In natural waters (pH 5–9), nickel (2^+) ions may adsorb to Fe/Mn oxides or dissolved organic matter (DOM), or form complexes with inorganic ligands (OH, SO, Cl or NH) (IPCS, 1991). These interactions produce a complex mixture of nickel species and compounds that are largely determined by the chemistry of the aquatic compartment. In aquatic systems, > 90% of nickel is associated with particulate matter or sediments (Hart, 1982). However, this distribution between phases is affected by pH. At pH > 6, nickel (2^+) readily adsorbs to suspended organic matter or precipitates with iron and manganese hydroxides (ANZECC, 2000a). Conversely, in acidic waters (pH < 6), adsorption of nickel (2^+) to organic matter plays a minor role and ionic nickel is relatively mobile (ANZECC, 2000a).

The partitioning of nickel in soil has a complex dependence on soil properties, but is mainly determined by soil pH, cation exchange capacity (CEC) and the concentration of both clay and DOM (Danish-EPA, 2015). Recently, the median partition coefficient (log K_D) value for nickel of 2.74 was reported in a large-scale European geochemical survey of 500 soils with varying physical and chemical properties (Janik, et al., 2015).

Degradation

No information on the degradation of the assessed chemical was provided. The assessed chemical is inorganic, and therefore excluded from persistence classification.

Bioaccumulation

Conventional measures of bioaccumulation as applied to organic chemicals are not appropriate for metal ions. However, Nickel does not bioaccumulate to a significant extent in aquatic or terrestrial organisms (ECB, 2008; NICNAS IMAP report, 2020). Although data are limited for the biomagnification of nickel, most studies indicate that biomagnification does not occur in aquatic (ECB, 2008) or terrestrial (Commonwealth of Australia, 2011) food chains.

Predicted environmental concentration (PEC)

The predicted environmental concentration (PEC) has not been calculated as release of the assessed chemical to the aquatic environment is not expected based on its assessed use patterns.

Environmental effects

Effects on aquatic Life

Acute toxicity

The following median lethal concentration (LC50) and inhibitory concentration (IC50) values for model organisms were supplied for dissolvable Ni in solution.

Taxon	Endpoint	Method
Fish	96 hr LC50 = 400 µg Ni/L at 21 mg/L $CaCO_3$ hardness level	*Pimephales promelas* (flathead minnow) US EPA method Static conditions Measured concentration
Invertebrate	48 hr LC50 = 0.13 µg Ni/L at 300-320 mg/L $CaCO_3$ hardness, pH 8-8.5	*Ceriodaphnia dubia* (water flea) Immobility/mortality Method not specified Measured concentration
Algae	96 hr IC50 = 17 µg Ni^{+2}/L	*Ankistrodesmus falcatus* (green algae) Growth rate OECD TG 201 method Static conditions Nominal concentration

Chronic toxicity

The following effect concentration (EC10) values for model organisms were supplied for dissolvable Ni in solution.

Taxon	Endpoint	Method
Fish	12-day EC10 = 10 µg dissolved Ni/L at 0.25 mg Ca/L, pH 6.4	*Melanotaenia splendida* (Eastern rainbow fish) Number hatched US EPA 2002 test method Time weighted measured concentration
Invertebrates	30-day EC10 = 1.1 dissolved Ni^{2+} µg/L	*Lymnaea stagnalis* (great pond snail) Growth Unspecified test method Arithmetic mean measured concentration

Predicted no-effect concentration (PNEC)

Default guideline values are published for nickel in the *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*. These values represent thresholds above which further assessment of potential toxicity may be required to ensure environmental quality and have been normalised using a water hardness of 30 mg $CaCO_3$/L. For marine ecosystem, a high reliability guideline value for protection of 95% of marine species has been determined to be 7 µg Ni/L (ANZECC, 2000b).

Categorisation of environmental hazard

As the assessed chemical is inorganic, it is excluded from categorisation under the *Australian Environmental Criteria for Persistent, Bioaccumulative and/or Toxic Chemicals* (DCCEEW, 2022).

Environmental risk characterisation

As the assessed chemical is inorganic, it is excluded from categorisation under the *Australian Environmental Criteria for Persistent, Bioaccumulative and/or Toxic Chemicals* (DCCEEW, 2022).

A Risk Quotient (PEC/PNEC) for the aquatic compartment was not calculated as the currently available information indicates the assessed chemical will not be released to the environment, untreated. Therefore, the risk from the assessed chemical can be managed.

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