Directive 98/8/EC concerning the placing biocidal products on the market

***Inclusion of active* substances in Annex I or I A to Directive 98/8/EC**

Assessment Report

Hydrogen cyanide
Product –type 14

25 May 2012

Annex I – the Czech Republic

1 STATEMENT OF SUBJECT MATTER AND PURPOSE

1.1 Procedure Followed

This assessment report has been established as a result of the evaluation of Hydrogen Cyanide as product-type 14 (wood preservatives), carried out in the context of the work programme for the review of existing active substances provided for in Article 16(2) of Directive 98/8/EC concerning the placing of biocidal products on the market, with a view to the possible inclusion of this substance into Annex I or IA to the Directive.

Hydrogen Cyanide (CAS no. 74-90-8) was notified as an existing active substance, by Lučební závody Draslovka a.s. Kolín, hereafter referred to as the applicant, in product-type 14. Commission Regulation (EC) No 1451/2007 of 4 December 2007 lays down the detailed rules for the evaluation of dossiers and for the decision-making process in order to include or not an existing active substance into Annex I or IA to the Directive. In accordance with the provisions of Article 7(1) of that Regulation, the Czech Republic was designated as Rapporteur Member State to carry out the assessment on the basis of the dossier submitted by the applicant. The deadline for submission of a complete dossier for Hydrogene Cyanide as an active substance in Product Type 14 was 1 March 2006, in accordance with Annex V of Regulation (EC) No 2032/2003.

In accordance with provision of Article 4a of Regulation (EC) No. 2032/2003 as amended by Regulation (EC) No. 1048/2005 the Czech Republic applied for essential use of the active substance Hydrogen Cyanide on 18.11.2005.

On 16.2.2006, the competent authority of the Czech Republic received a dossier from the applicant. The Rapporteur Member State accepted the dossier as complete for the purpose of the evaluation on 28.2.2006.

On 24.1.2008, the Rapporteur Member State submitted, in accordance with the provisions of Article 14(4) and (6) of Regulation (EC) No 1451/2007, to the Commission and the applicant a copy of the evaluation report, hereafter referred to as the competent authority report. The Commission made the report available to all Member States by electronic means on 19.2.2008. The competent authority report included a recommendation for the inclusion of Hydrogen Cyanide in Annex I to the Directive for product-type 14.

In accordance with Article 16 of Regulation (EC) No 1451/2007, the Commission made the competent authority report publicly available by electronic means on 25.2.2008. This report did not include any information that was to be treated as confidential in accordance with Article 19 of Directive 98/8/EC. In order to review the competent authority report and the comments received on it, consultations of technical experts from all Member States (peer review) were organised by the Commission. Revisions agreed upon were presented at technical and competent authority meetings and the competent authority report was amended accordingly.

On the basis of the final competent authority report, the Commission proposed the inclusion of Hydrogen Cyanide in Annex I to Directive 98/8/EC and consulted the Standing Committee on Biocidal Product on 25 May 2012.

In accordance with Article 15(4) of Regulation (EC) No 1451/2007, the present assessment report contains the conclusions of the Standing Committee on Biocidal Products, as finalised during its meeting held on 25 May 2012.

1.2 Purpose of the assesment report

This assessment report has been developed and finalised in support of the decision to include Hydrogen Cyanide in Annex I to Directive 98/8/EC for product-type 14. The aim of the assessment report is to facilitate the authorisation in Member States of individual biocidal products in product-type 14 that contain Hydrogen Cyanide . In their evaluation, Member States shall apply the provisions of Directive 98/8/EC, in particular the provisions of Article 5 as well as the common principles laid down in Annex VI.

For the implementation of the common principles of Annex VI, the content and conclusions of this assessment report, which is available at the Commission website[3], shall be taken into account.

However, where conclusions of this assessment report are based on data protected under the provisions of Directive 98/8/EC, such conclusions may not be used to the benefit of another applicant, unless access to these data has been granted.

1.3 Overall conclusion in the context of Directive 98/8/EC

It can be concluded from the evaluation that the proposed use of biocidal products based on hydrogen cyanide under the specified conditions fulfil the safety requirements laid down in Article 5 of Directive 98/8/EC. This conclusion is, thus, subject to

i. compliance with the particular requirements in the following sections of this

assessment report,

ii. the implementation of the provisions of Article 5(1) of Directive 98/8/EC, and

iii. the common principles laid down in Annex VI to Directive 98/8/EC.

Furthermore, these conclusions were reached within the framework of the uses that were proposed and supported by the applicant (see Appendix II). Extension of the use pattern betone those described will require an evaluation at product authorisation level in order to establish whether the proposed extensions of use will satisfy the requirements of Article 5(1) and of thecommon principles laid down in Annex VI to Directive 98/8/EC.

2 OVERALL SUMMARY AND COCLUSIONS

2.1 Presenatation of the Active Substance

2.1.1 Identity, Physico-Chemical Properties *& Methods of Analyssis*

CAS number	74-90-8
Einecs number	200-821-6
Other No.	CIPAC NO. 126
Chemical name, synonyms	Hydrogen cyanide, Hydrocyanic acid (water solution)
Molecular formula	HCN
Structural formula	H-C≡N
Molecular mass (g/mol)	27.03
Purity of the active substance as manufactured	Min. 97.6 % wt
Impurities	Water (1.18 -1.42 % wt)
Additives	Phosphoric acid (0.08-0.12 % wt) , sulphur dioxide (0.9 – 1.1 % wt)

Hydrogen cyanide is colourless liquid for temperatures between -13.4 and +25.7°C (acid), and colourless gas with almond-like odour for higher temperatures. It is miscible with water and soluble in ethanol and ether. Octanol/water partition coefficient of 5 (log Kow = 0.66) indicates slight preference of the hydrophobic compartments. High values of vapour pressure (84 kPa at 20°C, 35 kPa at 0°C) and of Henry's law constant signalize rapid evaporation and rapid leakage from water solution. Specific density of vapours is slightly below 1 (0.937 at 31°C) supports the assumption of an even distribution. The vapours are flammable and explosive in the range of concentrations in air of 5.6 to 40 v/v%.

The representative biocidal product named Uragan D2 (stabilized liquid hydrogen cyanide) is mixture of approx. 98 % of hydrogen cyanide (CAS No 74-90-8) with stabilizing additives. Uragan D2 is supplied completely soaked into a porous material in 1.5 kg gas-tight cans made of 0.45 mm steel. During fumigation it evaporates and brings about its effect as a gas.

Methods for analysis of the active substance as manufactured as well as methods for the determination of the additives and impurities have been described in sufficient detail. Methods for residue determinations in soil, water, air and blood have been validated and shown to be sufficiently specific, accurate, sensitive and to provide for appropriate LOQ with respect the toxicological and environmental endpoints of hydrogen cyanide.

Summary information on the identity and physico-chemical properties and analytical methods can be found in Appendix I to this document (List of Endpoints).

2.1.2 Intended Uses and Effficacy

Hydrogen cyanide is used as fumigant for professional use only to control pests (Main Group 03, PT 14 – rodenticide) in empty storehouses, depositories, transport facilities, containers, libraries , other buildings without any materials which are able to absorb hydrogen cyanide and which cannot be made strict gastight. Hydrogen cyanide can never be used in buildings inhabited by people.

Target organisms are rodents: *Rattus norvegicus, Rattus rattus, Mus musculus, Microtus arvalis.*

Universal efficacy against rodents follows from the well-known mechanism of toxic action. This is confirmed by long term experience as well as by acute toxicity studies. Experience shows that target organisms do not develop resistance.

2.1.3 Classification and labelling

Proposal of the classification and labelling of the active substance

	Classification and labelling in compliance with Annex VI Regulation (EC) No. 1272/2008
Hazard classification and Category Code(s)	Flam. Liq. 1; Acute Tox.1; Aquatic Acute 1; Aquatic Chronic 1

Hazard statement Code(s)	H224; H330; H400; H410
Labelling	
Pictogram and Signal word Code(s)	 Danger
Hazard statement Code(s)	H224: Extremely flammable liquid and vapour H330: Fatal if inhaled H410: Very toxic to aquatic life with long lasting effects

Precautionary statement Code(s)	P210 Keep away from heat/sparks/open flames/hot surfaces. — No smoking. P260 Do not breathe dust/fume/gas/mist/vapours/spray. P262 Do not get in eyes, on skin, or on clothing. P280/284 Wear protective gloves/protective clothing/eye protection/face protection/respiratory protection. P303+P361+P353 IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower. P304+P340 IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing. P310 Immediately call a POISON CENTER or doctor/physician. P273 Avoid release to the environment.

Proposal for classification of biocidal product Uragan D 2 is the same as that for the active substance.

2.2 Summary of the Risk Assessment

2.2.1 Human Health Risk Assessment

Human health risk assessment is based on data submitted by the applicant. Toxicology of hydrogen cyanide and generally of various sources of cyanide ion has long tradition: rich material has been accumulated on all relevant effects, and repeatedly analysed and discussed in peer-reviewed surveys. No new studies were therefore planned and performed by the applicant.

2.2.1.1 Hazard Identification

Dangerous properties as well as sub-cellular mechanisms of cyanide ion toxicity are thoroughly explored. Common mechanism of toxicity ,i.e. the toxic agent common to the below surrogates is CN^- , and known toxicokinetics, e.g. slow releases of CN obviate occurrence of acutely cyanide dangerous peaks, justifies

the use of toxicological data on inorganic cyanides and nitriles (aceton cyanhydrin, acetonitrile) as surrogates for missing or unreliable components of the toxicological profile of hydrogen cyanide. In addition to ample epidemiological and clinical evidence, literature provides a large quantity of experimental data; on the other hand most experimental studies collected did not meet requirements for a key study. The necessary validity and reliability is ensured by cross-comparison of results of many studies widely differing in the source of cyanide, routes of administration, endpoints, methods, species and interpretation approaches.

Toxicokinetics

Hydrogen cyanide is readily absorbed from orally administered water solutions or from fumigated food and oral absorption is 100 %. For respiratory route 100 % pulmonary retention is assumed. The rate of absorption of gaseous HCN by dry skin is by more than two orders of magnitude lower than absorption by inhalation.

Cyanides are readily distributed within the body by blood and up to 80 % of absorbed dose is metabolised to thiocyanate at a rate of 1 µg/kg body weight per minute. At absorption rate exceeding 1.2 µg/kg bw per minute the blood concentration of CN is expected to grow with duration of acute exposure in most subjects. Low affinity of HCN to lipids and relative rate of its metabolic transformation to thiocyanates indicate that cyanides do not accumulate in the organism.

Acute toxicity

Hydrogen cyanide is highly toxic on inhalation, its inhalation LC 50 ranging from 3778 mg/m^3 for exposure time of 10 seconds to 158 mg/m^3 for a 60 minute exposure. It is classified as very toxic (T+) with risk phrase R 26 (very toxic by inhalation) (CLP: acute tox.1; H330).

Due to low dermal uptake of gaseous hydrogen cyanide the acute toxicity via this route is low and no corresponding classification is required.

Hydrogen cyanide toxicity is due to the impairment of the tissue utilization of oxygen making the cells critically dependent on oxidative metabolism most vulnerable. Hence the effects on nervous and cardio vascular systems are the most critical ones.

None of the human or animal data meet requirements for labelling of hydrogen cyanide as a skin irritating substance, and hydrogen cyanide is not classified as irritant for eyes. Human data on respiratory irritation are mostly negative and do not justify classification either. Hydrogen cyanide does not present any

structural alert for skin sensitization and sensitization properties of cyanides or nitriles have not been suggested by the experience in humans over a period of many years of production and use.

Repeated toxicity

The toxic effects found in studies using repeated oral dosing of cyanides are interpreted as being due to cumulated injury from repeated acute poisonings resulting from acutely dangerous peaks of readily absorbed cyanides. Such peaks and hence the acute effects avoided, the inhibition of thyroid function is the only critical long term effect. This effect is ascribed to goitrogenic potency of thiocyanate, the main metabolite of cyanides. The NOAEL for this effect from which long term AEL was derived is 10 mg/kg.bw per day. This NOAEL primarily draws on two chronic (2 year) studies, inhalatory (acetonitrile in rats and mice) and oral (HCN in diet, rats), NOAEL in both being ≥ 10 mg CN/kg bw per day (top dose). This is further supported by several studies reporting daily doses of *4.7 to 26 mg cyanide/kg.bw* (top doses used) being without effect in 13-week to 26 week studies.

Genotoxicity

Genotoxicity was observed only in cells with seriously lowered viability. HCN has been shown to posses no intrinsic genotoxic potential. This based on negative outcome of various mutagenicity studies on bacteria, a relevant in vitro mutagenicity on mamalian cells, in vivo bone marrow chromosomal aberrations test in rats and test of inhibition of mouse testicular DNA synthesis.

Carcinogenicity

Carcinogenicity was explored in combined chronic toxicity – carcinogenicity study of acetonitrile in rats and mice and an extensive two-year inhalation studies with acetonitrile in rats and mice. Based on the data from these studies no carcinogenicity is expected at doses substantially below acutely toxic level. This is further confirmed by epidemiological studies in workers exposed for many years to hydrogen cyanide in concentrations exceeding 10 mg/m^3 where no data leading to suspicion of hydrogen cyanide carcinogenicity were reported.

Reproductive toxicity

Various studies on reproductive toxicity were evaluated. These include teratology study with aceton cyanohydrin with rats, 13 week study via oral route (NaCN in drinking water) including reproduction toxicity in rats and mice, in vivo DNA synthesis inhibition in mouse, 10 week male fertility study (inhalation route to acetone cyanohydrin) in rats, female fertility study (inhalation route to acetone cyanohydrin) in rats. The NOAELs for reproductive toxicity end points range from 1 to 26 mg CN/kg bw,

all the values being the top, or single, doses. All experimental studies permitting precise estimates of cyanide doses administered concur in a conclusion that decreased fertility, teratogenity, embryotoxicity or developmental toxicity is limited to doses severely toxic for the adults. This is further confirmed by epidemiological studies in workers exposed for many years to hydrogen cyanide in concentrations exceeding 10 mg/m^3 where no data leading to suspicion of hydrogen cyanide being toxic for reproduction. Hence, NOAEL of 10 mg/kg bw determined for repeated toxicity covers also reproductive toxicity endpoints.

Neurotoxicity

The central nervous system is the primary target of acute cyanide toxicity due to its mechanism of toxic action which impairs the tissue utilization of oxygen. Studies exploring neurotoxicity include 13 week study via oral route (NaCN in drinking water) in rats and mice, 13 – 14 week inhalation study with acetone cyanohydrin in rats, 2 year inhalation study with acetonitrile in rats and mice, 180 day inhalation of cyanogens in rhesus monkeys. The NOAELs of these studies ranged from 4.7 (monkeys) to 26 mg/kg .bw (mice). As all these NOAELs are top doses, it is concluded that the neurotoxic endpoints are covered by NOAEL of 10 mg/kg bw.

Toxicological reference doses

Two AELs have been defined for hydrogen cyanide covering the relevant exposure scenarios. Another condition that must be always fulfilled is that air concentrations of hydrogen cyanide never exceed AEC of 3 mg/m^3 so as to avoid acutely dangerous peaks of cyanide in blood.

The AEC and AEL have been derived from human toxicokinetic data showing that the rate of spontaneous detoxication of cyanides in humans is 1 ug/kg body weight per minute. This rate of elimination balances, even under the very conservative assumption of 100% pulmonary retention, inhalation of air concentrations up to 3 mg/m^3 with the concentration of CN in erythrocytes remaining on 24 hours exposure, safely below the concentration at which first subjective symptoms were reported. Thus 3mg /m^3 define AEC the purpose of which is to prevent occurrence of acutely dangerous peaks in blood. As accumulation of CN in blood depends also on the total amount of HCN absorbed applying AEC together with the relevant AEL, acute or chronic, prevents acutely dangerous CN peaks in blood from occurring in all the possible exposure scenarios. 24 hour exposure to 3mg/m^3 corresponds to systemic dose of 1.44 mg/kg . bw assuming 100% pulmonary retention, inhalation rate of 1.25 m^3/hour and body weight of 60 kg. The dose of 0.48 mg/kg bw is used as acute AEL which corresponds to 8 hour exposure under the above assumptions. The conservative assumptions used in the derivation of acute AEL of 0.48 mg/kg bw and the fact that the toxicokinetic data on which it is based come from studies on hospital patients treated for high blood pressure ensures protection of vulnerable groups as well as general

population. This value is further supported by the absence of acute complaints in workers exposed for 8 hours to airborne HCN concentration below 20 mg/m^3.

The derivation of long term AEL has been primarily based on two chronic studies, one for inhalatory and one for oral route of administration. In both the NOAEL was ≥ 10 mg CN/kg bw per day. Applying the standard assessment factor of 100 the long term AEL is 0.1 mg CN/kg bw per day. As the assessment factor accounts for both interspecies differences in toxicokinetics and toxicodynamics (coefficient < 10), and interindividual variability in heterogenous human population (coefficient >10) due to differences in thiocyanate elimination rate, thiosuphate and CN intake from diet, smoking etc. this AEL protects also the vulnerable groups as well as general population.

Exposure assessment and risk characterisation

Operator exposure during fumigation:

During fumigation the personnel is required to use the prescribed personal protective equipment which rules out exposure to HCN during fumigation and ventilation. Organizational measures ensuring that operators will not come to contact with high concentrations of HCN vapours must be followed throughout the whole fumigation procedure including the ventilation phase and post ventilation phase until handing over the properly ventilated and cleared structures to the client/owner. Operators not wearing adequate PPE can only be exposed to concentration of HCN not exceeding 3mg/m3 while their internal exposure must not exceed long term AEL of 0.1 mg/kg bw. (i.e., in a case when operator should not wear adequate

PPE for the whole shift of 8 hours they can only be exposed to concentrations not exceeding 0.6 mg/m3

This then results in respiratory intake of less than 0.1 mg/kg bw (8 hours x 1.25 m3/hour x 0.6 mg/m3/ 60kg).

Post fumigation exposure:

Re- entry of operators into treated structures/ areas for inspection without use of the prescribed PPE including self-contained breathing apparatus is allowed only when gas concentration dropped below AEC of 3mg/m^3. The structure is entered then for the purpose of being handed over to the client. The time required for this hand-over does not exceed 1 hour resulting in an intake via inhalation of cyanide not exceeding 0.063 mg/kg.bw ,which corresponds to 63% of long term AEL of 0.1mg/kg bw.

Exposure of professionals during ventilation phase

During ventilation phase exclusion zone is determined so that the airborne HCN concentration at its border is 3 mg/m^3 (AEC). An operator wearing prescribed PPE (i.e. a face mask with appropriate filter) is responsible for shifting the border if need be e.g. due to a change in weather conditions. This operator is not exposed to HCN while wearing the PPE but exposure can take place during the breaks when the operator takes off the face mask. As a worst case such breaks are assumed to take up to 4 hours/day and the operator is required to find a place for these breaks, where the concentration of HCN in the air does not exceed 1 mg/m^3. This then results in respiratory intake of HCN of 0.08 mg/kg bw when assuming body weight of 60 kg and inhalation rate 1.25 m^3/ h. This is 80% of long tem AEL of 0.1 mg/kg bw per day. If the operator is not to wear PPE for a major part of the 8 hour shift they must seek and stay in a place where the concentration of HCN does not exceed 0.6 mg/m^3. This then results in respiratory intake of less than 0.1 mg/kg bw (8 hours x 1.25 m^3/hour x 0.6 mg/m^3/ 60kg).

Exposure of other users:

Hydrogen cyanide is intended for use by adequately trained professionals only. Before delivery, the customer should declare the intended type of use and provide proof of his ability to handle the product safely. The manufacturer is, on the basis of delivery terms, entitled to carry out audits of the customer's premises.

Exposures of bystanders:

To avoid unacceptable exposure of by- standers and by- passers an exclusion zone is set around the fumigated structure which cannot be entered by any person except by adequately trained professionals

from the beginning of the fumigation till the handing over of the structure to the client. This zone is determined so as beyond its boundaries the concentration of airborne HCN never exceeds AEC of 3mg/m^3. As by–passers are assumed to be exposed to HCN only infrequently acute AEL is relevant to assess the risk they undergo. 8 hour exposure to 3mg/m^3 is needed before acute AEL is exceeded in adults which is more than passers-by can be reasonably assumed to spend near the frontier of the exclusion zone. Rather, reasonable assumption is that by-passers spent 30 minutes at the border of the exclusion zone. This corresponds to 0.03 mg /kg bw for adults when applying inhalation rate of 1.25 m^3/hour and body weight of 60 kg (0.5 hours * 1.25 m^3 *3 mg/m^3 / 60 kg) and to 0.15 mg/kg bw for infants when applying inhalation rate 1 m^3/hour and body weight of 10 kg. Thus, the systemic dose due to exposure of an adult passers- by corresponds to 6.3 % and that of an infant corresponds to 31% of acute AEL.

Exposure on the day following the hand over

On the day following its hand-over the fumigated structure is put to normal use. Eight hour exposure of persons entering it is assumed. HCN concentration in the air is bound to drop by several orders of

magnitude by the time of the beginning of exposure if the first order kinetics with the rate constant derived from decrease during ventilation phase is assumed (i.e., during ventilation the drop was from 10 g/m^3 to 3 mg/m^3 in 24 hours thus giving rate constant of 0.34 $hour^{-1}$for first order kinetics). In reality, the post ventilation drop will be even more drastic as all the seals will be removed from windows, doors etc. and thus more air will be exchanged per unit of time. In addition, during the first part of the normal use it is advisable to continue good ventilation of the object. Then during the ventilation the 8 hour exposure on the day following the hand over is calculated to be 0.008 mg /kg bw assuming inhalation rate of 1.25 m^3/hour, body weight of 60 kg, 12 hours between the hand over and the beginning of the exposure, no drop of HCN concentration during the 8 hour exposure. This dose is 8% of chronic AEL thus posing no risk to human health.

2.2.2 Physical-chemical hazard

The relevant physical and chemical properties of biocidal product Uragan D2 are the same as that of hydrogen cyanide. Hydrogen cyanide is at normal pressure an extremely flammable gas/liquid. HCN vapours form explosive mixtures with air with upper explosive limit 40 % vol. and lower explosive limit 5.6 % vol.: the maximum concentration used in fumigation is below 5 %, nevertheless the danger of fire and explosion of vapours is high with regard to local concentration inhomogeneity.

Risk characterisation for the physico-chemical properties

When used conformably to special "Manual for Organization of hydrogen cyanide sanitation procedures", physical and chemical properties of hydrogen cyanide do not present risk to users.

2.2.3 Environmental risk assessment

2.2.3.1 Fate and distribution in the environment

Environmental fate and behaviour of HCN, due to its low boiling point, high vapour pressure at temperature over 10 C and lower relative density compared to density of air, is different from the fate and behaviour of other cyanide compounds. The main compartment where the most significant part of HCN liberated into the environment is transferred is the atmosphere. The persistence half-time of HCN in the atmosphere is 1-3 years. The most important mechanism of its degradation in the atmosphere is a reaction with hydroxyl radicals brought to the atmosphere by air humidity

Hydrogen cyanide is completely miscible with water. However, its ability to cross from the atmosphere into aqueous media, characterized by the value of Henry's law constant 5.2 kPa. m^3.mol^{-1}, is low. Therefore, the part of hydrogen cyanide which is washed out from the atmosphere by precipitation is low as well. If hydrogen cyanide or cyanides enter aqueous media, equilibrium between the concentration of cyanide ions and undissociated hydrogen cyanide is established.

Biodegradation contributes to the elimination of cyanides from natural water. Cyanides occur in water most commonly in the form of hydrogen cyanide, cyanide ions and other cyanide compounds in a wide range.

In water, HCN and cyanide ion exist in equilibrium, their relative concentrations depend on pH and temperature. With pH lower than 8, more than 93 % free cyanides in water is in the form of undissociated hydrogen cyanide. HCN consequently hydrolyses to formamide which is further hydrolysed to ammonia and formate ion. However, the hydrolysis rate is slow and in the elimination of cyanide ion, it does not compete with evaporation and biodegradation.

Biodegradation of cyanides in surface water also depends on pH, cyanide concentration, temperature, availability of nutrients, and microbe adaptation. Cyanide ion is toxic for microorganisms at concentration 5-10 mg/l, but adaptation of microorganisms to this compound increases tolerance and microorganisms are able to decompose low cyanide concentrations.

In wastewater treatment plant conditions, adapted sludge is capable of decomposing cyanide concentrations lower than or equal to 100 mg/l.

Non-toxic concentrations of cyanides can be readily biodegraded, both aerobically and anaerobically. Aerobic degradation yields CO_2 and ammonia (that may be further converted to nitrate or nitrite); anaerobic biodegradation yields ammonia and methane.

In nature, degradation of free cyanide ions from aquatic environment occurs also due to these chemical processes: oxidation, hydrolysis, and photolysis, of which the last one plays only a negligible or very little role.

Hydrogen cyanide is very resistant to photolysis. The most important reaction of hydrogen cyanide in air is the reaction with photochemically generated hydroxyl radicals and subsequent rapid oxidation to carbon monoxide (CO) and nitric oxide (NO); photolysis and reaction with ozone are not important transformation processes, and reaction with singlet oxygen (O1D) is not a significant transformation process except at stratospheric altitudes where singlet oxygen is present in significant concentrations. The rate of hydroxyl radical reaction with hydrogen cyanide in the atmosphere depends on the altitude, and the rate of the reaction is at least one order of magnitude faster at lower tropospheric altitudes (0–8 km) than at upper tropospheric altitudes (10–12 km). Based on a reaction rate constant of $3x10^{-14}$ cm^3/(molecule.sec) at 25 °C

Photolysis in surface waters occurs, but is very low and its part in the degradation of cyanide ions from aquatic environment is insignificant.

Hydrogen cyanide hardly enters soil; its sorption ability to solid substances – sediment – is due to its high water solubility considered negligible.

Evaporation plays the biggest part in the dissipation of cyanides from water. In surface waters, this is a predominant fate of HCN.

Evaporation is influenced by several parameters, e.g. temperature, pH, wind speed (in natural surface waters), and Henry's law constant.

At pH lower than 9.2, most of free cyanide in a solution exists in the form of HCN and volatile cyanides, and degradation (evaporation) proceeds faster. Evaporation is for HCN degradation from water more important than decomposition due to chemical reactions and biodegradation. This presumption applies to surface waters; elimination in ground waters shall take longer.

Most hydrogen cyanide from both natural and industrial sources reaches the atmosphere. HCN remains in the troposphere, only 2 % reaches the stratosphere.

In the atmosphere, HCN may be transported to long distances from the emission source.

HCN slowly degrades in air; its half-time is 1-3 years. In the atmosphere, it reacts with hydroxyl radicals brought there by air humidity, and through this reaction it decomposes. Although HCN is readily soluble in water, its elimination from the atmosphere through rain water is negligible.

HCN bioaccumulation in aquatic organisms is not expected. Bioconcentration factor for HCN was calculated - BCF 0.73. Neither HCN bioaccumulation in the food chain is expected.

Due to its usage as fumigant, using hydrogen cyanide for direct fumigation of food and feed is not expected. Since significant penetration of HCN into water or soil after treatment is not expected either, the risk of compartment-non-specific intoxication of people by the food chain may be considered negligible.

2.2.3.2 Effects assessment

Aquatic Compartment

The results of many experiments are published in the literature in which the toxicity of cyanides for fish, invertebrates and algae was investigated.

Acute toxicity for fish

Regarding fish toxicityin some species of juvenile fish the sensitivity is higher or the same at lower temperature, in other species the sensitivity to HCN is higher at higher temperatures. Generally, all measured values are within the classification highly toxic for aquatic organisms.

Observations from summary materials used are based on the article by Kovacs T. G., and G. Leduc. 1982. Acute toxicity of cyanide to rainbow trout (*Salmo gairdneri*) acclimated at different temperatures. Can . J. Fish. Aquat. Sci. 39: 1426-1429, in which dependency of temperature and HCN concentration effects on acute toxicity is documented. 96-hour mean LC50 values from the study conclusions:

LC50 = 0.028 ± 0.004 $mg.l^{-1}$ at 6 °C

LC50 = 0.042 ± 0.004 $mg.l^{-1}$ at 12 °C

LC50 = 0.068 ± 0.004 $mg.l^{-1}$ at 18 °C

Rainbow trout acclimated for the test temperature survived longer in lethal concentrations of cyanide. Toxicity curves clearly showed the temperature effect on the acute toxicity of cyanide is concentration dependent.

The LC50 = 0.042 $mg.L^{-1}$ value was selected for the risk assessment, with regard to temperatures at which acute toxicity test are performed according to current methods (Regulation (EC) 440/2008, EU method no. 203, temperature during the test 12-18 °C). Regarding the way of the substance use and the fact that the fumigation process is performed only at favourable climatic conditions, and regarding effects of other factors in the environment, no significant HCN concentration able to affect adversely aquatic organisms is expected to enter water. From this point of view the effect of temperature and concentration on the LC50 value is not important for the risk assessment.

Acute toxicity for invertebrates

A value from the test performed by the applicant was chosen as the key value, since this test was performed in the GLP system and according to the valid OECD methodology:

EC50 (Daphnia magna , 48 hours) = 1.07 mg.l-1

Growth inhibition on algae

A value from the test performed by the applicant in the GLP system and according to the valid OECD methodology was chosen as the key value:

EC50 (*Scenedesmus subspicatus*, 72 hours) = 0.040 $mg.l^{-1}$

The calculation was performed with EUSES program, using scenario for fumigation, with the following results:

PNEC Aqua: 4 x 10^{-5} mg/L

Although hydrogen cyanide is highly toxic for aquatic organisms, exposure of the aquatic environment during fumigation is negligible.

Significant exposure of aqueous environment is not expected.

Sediment

No sediment tests are available.

The calculation was performed with EUSES program, using a scenario for fumigation, with the following results:

PNEC for fresh-water sediment-dwelling organisms 3.81 x 10^{-5} mg/kg wwt

Direct exposure of sediment is not expected due to the use pattern and physico-chemical properties of hydrogen cyanide.

Inhibition of microbial activity

A value for inhibition of microbial activity 25 mg/l was found in literature sources.

Hydrogen cyanide is a gas and its use pattern as a fumigant with direct release to the environment, there is no likelihood that the active ingredient will enter aerobic microbial treatment plants/sewage plants/water treatments plants. Consequently, there is no likelihood of exposure for STP micro-organisms.

The calculation was performed with EUSES program, using a scenario for fumigation, with the following results:

$PNEC_{STP}$ = 2.5 x 10^{-1} mg/L

Terrestrial Compartment

The use is limited to closed spaces, hydrogen cyanide is used in the form of a gas for fumigation; the main environmental compartment it enters is air. Hydrogen cyanide tends to ascend to higher levels of the atmosphere. Direct release to the terrestrial compartment is not expected.

The calculation was performed with EUSES program, using a scenario for fumigation, with the following results:

$PNEC_{soil}$ = 1.02 x 10^{-5} mg/kgwwt

Significant exposure of terrestrial environment is not expected

Atmosphere

For the application of gaseous substances for fumigation, the general exposure scenario for the use of gaseous fumigants was proposed by working group of Environment Directorate OECD (OECD Series on Emission scenario Documents, Number 2, Emission Scenario Document for Wood Preservatives, Part 2, p. 93–96).

According to the above mentioned general scenario it is assumed that at most 2 % w/w of the total amount of the fumigant released into a closed object is retained in treated objects or materials and 0.1 % of the fumigant is decomposed. The extent of fumigant emissions to air is then expressed as an amount of the fumigant released into the treated object (decreased by the part retained in the treated object and by the part which underwent decomposition) recalculated on days in dependence on the ventilation time. If these general principles are applied to an individual case of fumigation with hydrogen cyanide, for the determination of the total amount of hydrogen cyanide emissions to air the calculation may be based on the volume of the treated object or working chamber in m^3 and on the hydrogen cyanide application concentration of 10 g/m^3.

For a extremely large object with the volume around 100,000 m^3, the consumption about 1,000 kg of hydrogen cyanide can be expected, for a large object with the volume around 10,000 m^3, the consumption about 100 kg of hydrogen cyanide can be expected, for a smaller object around 1,000 m^3 one tenth, i.e. 10 kg, can be expected, and for a small container around 100 m^3 approx. 1 kg. For a smaller container with the volume of 300 m^3 it is 3 kg.

Emission rates of active substance to atmosphere (Eatm, fumi) after fumigation acc. to OECD Series on Emission scenario Documents, Number 2, Emission Scenario Document for Wood Preservatives, Part 2, p. 93–96 for objects with volume of 100,000 m3, 10,000 m3, 1,000 m3, 300 m3 and 100 m3 are 979, 97.9, 9.79, 2.94 and 0.979 kg/d respectively during 24hr ventilation and 326, 32.6, 3.26, 0.98 and 0.326 kg/d for 72hr ventilation time.

If the decrease of the amount of ventilated hydrogen cyanide by its retention or decomposition is neglected, this amount should be ventilated in 24–72 hours. In the less favourable case, the whole applied amount of hydrogen cyanide should leave to air within 24 hours. The concentration of hydrogen cyanide

in gas leaving the ventilated object will decrease from the initial value higher than 10 g/m^3 practically to zero at the end of the ventilation phase.

2.2.3.3 PBT assessment

It can be reliably stated that hydrogen cyanide does not have properties of PBT or vPvB because of its preferential detention in free atmosphere, its low ability to bioaccumulate, characterised by BCF= 0.73 and low persistence from the point of view of definition values of those parameters.

Hydrogen cyanide does not fulfil the PBT or vPvB criteria.

2.2.3.4 Risk characterization

Risk for atmosphere

Hydrogen cyanide ventilated to air can cause damage by retaining in the air (and thus it could change the properties of atmosphere) and by indirect endangering human health and other parts of nature.

In air hydrogen cyanide behaves as small halogen-carbon compounds. It is capable of contributing to global warming, weakening the protective ozone layer, and increasing the ozone production in troposphere. However, the potential of those effects is small due to little penetration of hydrogen cyanide into stratosphere and due to a slow course of reactions by which ozone is formed in troposphere. The present conditions in atmosphere cannot be significantly changed by hydrogen cyanide entering the atmosphere after the end of fumigation, because the amount of hydrogen cyanide used for fumigation will always be only a negligible part of the amount of this substance formed spontaneously by natural processes or released into atmosphere from other anthropogenic sources.

The amount of hydrogen cyanide released to air during individual applications in medium and large objects can be of the order of tens or hundreds of kilograms. From the regional point of view, such a small amount cannot cause any measurable change of hydrogen cyanide concentration in the atmosphere.

From the local point of view, it is necessary to know the distribution of concentrations in the vicinity of a treated object during its ventilation. According to the already mentioned emission scenario for fumigation, it was proposed to assume that the total applied amount of the fumigant is equal to the total flux of the fumigant emissions to air for the time of its ventilation. The above given reasoning gave us the flux of emissions 1–1000 kg HCN/day for 24-hour venting time.

In 1970's, anthropogenic production of hydrogen cyanide into the atmosphere in the USA was estimated at approx. 20,000 t/y. Most of anthropogenic formed cyanides, around 90 %, were generated from motor vehicle exhaust fumes (7-9 mg/km for vehicles not equipped with a catalyst and approx. 0.6 mg/km for catalyst equipped vehicles). Further significant anthropogenic sources of hydrogen cyanide emissions to

the atmosphere include its production and production of other organic as well as inorganic cyanide compounds. In 2000, the total world production of HCN reached 1.4 mil. tons. Large amount of hydrogen cyanide is released to the atmosphere from processing industries such as metallurgy, surface treatment of metals, gold and silver mining from low-grade ores. Significant sources of HCN anthropogenic emissions include also landfills and sludge setting lagoons to which wastes containing cyanides, emissions from municipal and industrial waste incinerators, emissions from incinerating organic substances with high nitrogen content (polyurethane, acrylonitrile, polyamides etc.) are placed. A relatively small quantity comes from the usage of HCN for treatment of closed structures.

Overview on the calculated PEC in air (according to the EUSES calculation)

Concentration in air during emission episode

Product Type	**PEC_{air} [mg/m³]**
PT14	
PT14_1 - Fumigant applied in a container with volume 100 m^3, amount of HCN used: 1 kg.	2.72×10^{-4}
PT14_6 - Fumigant applied in a container with volume 300 m^3, amount of HCN used: 3 kg.	8.14×10^{-4}
PT14_2 – Fumigant applied in a small standard structure with volume 1,000 m^3, amount of HCN used: 10 kg.	2.72×10^{-3}
PT14_3 – Fumigant applied in a large standard structure with volume 10,000 m^3, amount of HCN used: 100 kg .	2.72×10^{-2}
PT14_4 – Fumigant applied in a large standard structure with volume 100,000 m^3, amount of HCN used: 1,000 kg.	2.72×10^{-1}

Concentration in air, 100 m from point source

Product Type	**PEC_{air} [mg/m³]**

Product Type	**PEC_{air} [mg/m³]**
PT14	
PT14_1 - Fumigant applied in a container with volume 100 m^3, amount of HCN used: 1 kg.	7.46 x 10^{-7}
PT14_6 - Fumigant applied in a container with volume 100 m^3, amount of HCN used: 1 kg.	2.24 x 10^{-6}
PT14_2 – Fumigant applied in a small standard structure with volume 1,000 m^3, amount of HCN used: 10 kg.	7.46 x 10^{-6}
PT14_3 – Fumigant applied in a large standard structure with volume 10,000 m^3, amount of HCN used: 100 kg.	7.46 x 10^{-5}
PT14_4 – Fumigant applied in a large standard structure with volume 100,000 m^3, amount of HCN used: 1,000 kg.	7.46 x 10^{-4}

Real values of concentration will depend on dispersion conditions (direction and velocity of wind, vertical temperature gradient, terrain configuration, surrounding buildings, etc.). At climatic situations favourable for dissipation of emissions, under which the fumigation and ventilation should be carried out, the real ground concentration of hydrogen cyanide should be significantly lower due to the tendency of hydrogen cyanide molecules, which are lighter than air, to move up to higher layers of atmosphere.

The values of hydrogen cyanide concentrations 0.272 mg/m^3, estimated as PEC_{local} for one-time application of 1,000 kg of HCN, are approximately 10 times lower than PEL or 40 times lower than the value of MAC for working atmosphere valid in a number of countries.

Risk for aquatic environment

The risk for water is not expected due to an insignificant potential exposure of aqueous environment during fumigation with hydrogen cyanide.

At fumigation, hydrogen cyanide is applied to hermetically closed spaces, which cannot in any way communicate with surface or underground waters. No water can be present in treated objects. Direct exposure of aquatic environment to hydrogen cyanide is thus completely excluded. Indirectly, aquatic environment could be exposed to hydrogen cyanide retained by precipitation or by descending fog. Fumigation and following ventilation should thus be carried out only under favourable temperature and

dissipation conditions. Therefore, there is low probability of direct contact of ventilated hydrogen cyanide with rain or fog.

If hydrogen cyanide comes into contact with atmospheric precipitations, its ability to be adsorbed in aqueous phase is low, as indicated by a relatively high value of Henry's constant. The highest nominal concentration of hydrogen cyanide in ventilated air at the beginning of ventilation should be close to applied concentration 10 g/m^3. In equilibrium with this concentration of hydrogen cyanide in air, the concentration of hydrogen cyanide dissolved in water should reach the theoretical value of approx. 200 µg HCN per litre. To reach this concentration, it would be necessary to keep constant initial concentration of hydrogen cyanide in air and sufficiently long time to establish equilibrium between aqueous and gaseous phases. In reality, even in the least favourable case, when an exposure of aquatic system would occur, the concentration of hydrogen cyanide in the contaminated water would reach micrograms or even lower values. After contact with ground, this concentration would further decrease by dilution with non-contaminated water, by re-volatilization of hydrogen cyanide, and by neutralization of its toxic effects by conversion into less toxicologically important compounds, eventually by hydrolysis supported by bacterial enzymes.

Risk of secondary intoxication

The risk of food chain intoxication is negligible because of insignificant penetration of hydrogen cyanide into this chain

2.2.4 List of endpoints

In order to facilitate the work of Member States in granting or reviewing authorisations, and to apply adequately the provisions of Article 5(1) of Directive 98/8/EC and the common principles laid down in Annex VI of that Directive, the most important endpoints, as identified during the evaluation process, are listed in Appendix I.

3 DECISION

3.1 Background to the proposed decision

Hydrogen cyanide is intended for use by adequately trained professionals as fumigant for control of pests in buildings and other closed spaces. After sufficient exposure, hydrogen cyanide immediately kills all development stages of pests. No signs of resistance development were reported. Hydrogen cyanide is classified as extremely flammable and very toxic by inhalation. Inclusion of hydrogen cyanide in Annex I is feasible for the human health aspect because several safe uses are identified. Adverse health effects to operators during fumigation are ruled out by obligatory usage of adequate PPE and other safety measures. cute toxic effects to persons re-entering the fumigated area after ventilation are prevented by following

the obligatory safety measures. The only effect of long-term operator exposures is inhibition of thyroid functions. This effect should be prevented by setting chronic AEL. However, it is recommended to check for a possible occurrence of this effect by appropriate functional testing. Adverse health effects of passers-by are prevented by setting an exclusion zone around the fumigated structure/area.

The environmental risk assesment has shown that the proposed usage of hydrogen cyanide presents no unacceptable risk to the environment and can thus be included in Annex I. Hydrogen cyanide entering the atmosphere after the end of fumigation forms only a negligible part of the amount of this substance formed spontaneously by natural processes or released into atmosphere from other anthropogenic sources. Due to its physico-chemical properties hydrogen cyanide used in fumigation does not contribute to increase in levels of local background HCN emission or its content in surface water, nor is it expected to bioaccumulate significantly in aquatic organisms. Accidental endangering of human and animal health by hydrogen cyanide being retained in the air on ventilation is minimized by following the strict measures proposed for fumigation procedure.

3.2 Proposed decision regarding the inclusion in Annex I

Hydrogen cyanide shall be included in Annex I to Directive 98/8/EC as an active substance for use in product-type 14 (rodenticides), subject to the following specific provisions:

The minimum purity of the active substance used for the evaluation was 976 g/kg.

Member states shall ensure that authorisations of products for use as a fumigant are subject to the following conditions:

Tab. 1. Product shall only be supplied to and used by professionals adequately trained to use them;

Tab. 2. Safe operational procedures during fumigation and venting shall be established for operators and bystanders;

Tab. 3. Products shall be used with adequate personal protective equipment including, where appropriate, self-contained breathing apparatus and gas-tight clothing;

Tab. 4. Re-entry into fumigated spaces shall be prohibited until the air concentration has reached safe levels for operators and bystanders by ventilation;

Tab. 5. Exposure during and after ventilation shall be prevented from exceeding safe levels for operators and bystanders by the establishment of a supervised exclusion zone;

Tab. 6. Prior to fumigation, any food and any porous material with a potential to absorb the active substance, except the wood intended to be preserved, shall either be removed from the space to be fumigated or protected from absorption by adequate means, and the space to be fumigated shall be protected against accidental ignition.

3.3 Elements to be taken into account by Member States when authorising products

Elements, which were not mentioned under the specific provisions of the decision but which need to be taken into account at product authorisation level:

- Studies proving efficacy including kinetics of HCN evaporation in a treated object shall be required at the product authorisation stage;
- Residential buildings fumigation is not recommended;
- Authorisation holders shall ensure that users of the product are provided with detailed instructions for use, specifying the safety measures to be observed to ensure a safe and efficient use of the product;
- An exclusion zone shall be determined at the border of which HCN concentration must not exceed 0.6 mg/m3 and shall be set according to assumed exposure duration so that long term AEL is not exceeded for operators. In the exclusion zone, the presence of bystanders shall be prohibited and operators shall wear appropriate personal protective equipment. The zone shall be supervised.
- After fumigation, fumigated spaces shall be ventilated until the air concentration is below the AEC of 0.6 mg/m^3 in order to protect operators shall they have to re-enter the fumigated spaces, and must in any case be below 3mg/m3 for the re-entry of bystanders. Fumigated spaces shall be returned to their normal use no earlier than 24 hours after this concentration has been reached.

3.4 Requirement for further information

It is considered that the evaluation has shown that sufficient data have been provided to verify the outcome and conclusions, and permit the proposal for the inclusion of hydrogen cyanide for use in product-type PT 08 (wood preservatives) in Annex I to Directive 98/8/EC.

3.5 Updating this Assessment Report

This assessment report may need to be updated periodically in order to take account of scientific developments and results from the examination of any of the information referred to in Articles 7, 10.4

and 14 of Directive 98/8/EC. Such adaptations will be examined and finalised in connection with any amendment of the conditions for the inclusion of spinosad in Annex I to the Directive.

APPENDIX I: LIST OF ENDPOINTS

Chapter 1: Identity, Physical and Chemical Properties, Details of Uses, Further Information, and Proposed Classification and Labelling

Active substance (ISO Common Name)	Hydrogen cyanide
Function (e.g. fungicide)	Rodenticide (Fumigant)

Rapporteur Member State	Czech Republic

Identity (Annex IIA, point II.)

Chemical name (IUPAC)	Hydrogen cyanide
Chemical name (CA)	Hydrocyanic-acid
CAS No	74-90-8
EC No	200-821-6
Other substance No.	Index no.: 006-006-00-X
Minimum purity of the active substance as manufactured (g/kg or g/l)	976 g/kg
Identity of relevant impurities and additives (substances of concern) in the active substance as manufactured (g/kg)	Sulphur dioxide 9 – 11 stabilizing additive preventing spontaneous polymerisation

	Phosphoric acid … 0.8-1.2 stabilizing additive preventing spontaneous polymerisation
Molecular formula	HCN
Molecular mass	27.03 g/mol
 al formula	

Physical and chemical properties (Annex IIA, point III., unless otherwise indicated)

Melting point (state purity)	-13.4°C (7.9°F)
Boiling point (state purity)	25.7°C (78.3°F) (acid)
Temperature of decomposition	Not required – No decomposition or sublimation occur at the melting or boiling temperature. It is gas.
Appearance (state purity)	HCN is produced as liquid which is sorbed on surface of inert material. Boiling temperature of HCN in liquid state is 25.7 °C (78.3 °F). Due to the large surface of sorbed inert material, the evaporation is very fast. Therefore the active substance as used is gas only. Gas/ colourless Smells of bitter almonds.Olfactory threshold: 0.17 ppm (wt/vol.) in water 0.58 ppm (vol./vol.) in air
Relative density (state purity)	Density 0.6884 g/cm3 (liquid at 20 °C/68 °F) Relative density / Specific gravity 0.687 (liquid at 20

	°C/68 °F) Specific density: vapours 0.937 at 31 °C/ 87.8 °F
Surface tension	Not relevant. Active substance hydrogen cyanide is gas. HCN is used in gas phase for fumigation as it evaporates from inert material to which it is sorbed.
Vapour pressure (in Pa, state temperature)	84kPa (at 20°C / 68 °F) 35 kPa (at 0°C / 32 °F)
Henry's law constant (Pa.m3.mol -1)	5.1 kPa.m3.mol-1
Solubility in water (g/l or mg/l, state temperature)	Substance is fully miscible with water.
Solubility in organic solvents (in g/l or mg/l, state temperature) (Annex IIIA, point III.1)	Soluble in ethanol, ether
Stability in organic solvents used in biocidal products including relevant breakdown products (IIIA, point III.2)	Not relevant. the active ingredient is actually the product. Hence, no organic solvents are used in the product
Partition coefficient (log POW) (state temperature)	Log Kow = +0.66 at 20 °C/ 68 °F
Hydrolytic stability (DT50) (state pH and temperature) (point VII.7.6.2.1)	Apparently at pH < 8.3 HCN is the dominant species, at pH < 7. 99% will be as HCN molecule, and at pH > 10 CN

Dissociation constant (not stated in Annex IIA or IIIA; additional data requirement from TNsG)	pKa of 9.2
UV/VIS absorption (max.) (if absorption > 290 nm state ε at wavelength)	Maximum ≤ 200 nm, no absorption above 290 nm
Photostability (DT50) (aqueous, sunlight, state pH) (point VII.7.6.2.2)	Airborne HCN undergoes slow photolysis. The overall atmospheric lifetime of HCN is 5 to 6 months.
Quantum yield of direct phototransformation in water at Σ > 290nm (point VII.7.6.2.2)	None
Flammability	-17.8°C (flashpoint, closed cup) 538 °C / 1,000 °F (ignition point)
Explosive properties	Forms explosive gaseous mixtures with air with these explosive limits: upper: 40% vol. lower: 5.6% vol. In alkali medium it may come under an autocatalytic polymerisation reaction running in an explosion speed.

List of intended uses[1]

Object and/or situation	Member State or Country	Product name	Organisms controlled	Formulation		Application			Applied amount per treatment			Remarks:
(a)			(c)	Type (d-f)	Conc. of as (i)	method kind (f-h)	number min/max (k)	interval between applications (min)	g as/L min/max	water L/m2 min/max	g as/m2 min / max	(m)
Control of rodent pests		URA-GAN	Mice, rats and other rodents..	Gas	97.6 ± 2.4	Fumigation	Single use for killing	Single use.			Dosage: 10g/m3, i.e. in operating conditions 1kg/100m3.	

1 Adapted from: EU (1998a): European Commission: Guidelines and criteria for the preparation of complete dossiers and of summary dossiers for the inclusion of active substances in Annex I of Directive 91/414/EC (Article 5.3 and 8,2). Document 1663/VI/94 Rev 8, 22 April 1998

damaging goods stored in storehouses, depositories – museums, temples, transport vehicles – airplanes, railway wagons, sea and river boats, containers, libraries etc.		D2			%		pests	Further applicatios only upon new occurrence of pests.			Packing: Uragan D2 (stabilised liquid hydrogen cyanide) is supplied fully soaked into porous matter in closed gas-tight cans of 1.5kg Uragan D2.	

Classification and proposed labelling (Annex IIA, point IX.)

with regard to physical/chemical data	F+	Extremely flammable.
	R 12	Extremely flammable.
with regard to toxicological data	T+	Very toxic
	R 26	Very toxic by inhalation.
with regard to fate and behaviour data	No classification	
with regard to ecotoxicological data	N	Dangerous for the environment
	R50/5	Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
	(S 1/2)	Keep locked up and out of reach of children.
	S 7/9	Keep container tightly closed and in a well-ventilated place.
	S 16	Keep away from sources of ignition – No smoking.
	S 36/37	Wear suitable protective clothing and gloves.
	S 38	In case of insufficient ventilation, wear suitable respiratory equipment.
	S 45	In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible).
	S60	This material and its container must be disposed of as hazardous waste.
	S 61	Avoid release to the environment. Refer to special instructions / Safety data sheets.

Classification and labelling in compliance with Annex VI Regulation (EC) No. 1272/2008

(Annex IIA, point IX.)

with regard to physical/chemical data	Flam. Liq. 1; H224: Extremely flammable liquid and vapour
with regard to toxicological data	Acute Tox.1; H330 Fatal if inhaled H330: Fatal if inhaled
with regard to fate and behaviour data	No classification
with regard to ecotoxicological data	Aquatic Acute 1; Aquatic Chronic H400 Very toxic to aquatic life H410 Very toxic to aquatic life with long lasting effects
	P210 Keep away from heat/sparks/open flames/hot surfaces. — No smoking. P260 Do not breathe dust / fume / gas / mist / vapours / spray. P262 Do not get in eyes, on skin, or on clothing. P280/284 Wear protective gloves/protective clothing/eye protection/face protection/respiratory protection. P303+P361+P353: IF ON SKIN (or hair) Remove/Take off immediately all contaminated clothing.Rinse skin with water/shower. P304+P340 IF INHALED: Remove victim to fresh air and keep at rest in position comfortable for breathing. P310 Immediately call a POISON CENTER or doctor/physician. P273 Avoid release to the environment.

Chapter 2: Methods of Analysis

Analytical methods for the active substance

Technical active substance (principle of method) (Annex IIA, point 4.1)	Assessment of the hydrogen cyanide content during its production is carried out by argentometric titration of cyanides by silver nitrate following chemisorption of hydrogen cyanide into sodium hydroxide solution. Method principle Titration of cyanide with nitrate in an alkaline medium leads first to dissolution of silver cyanide in NaCN excess. As soon as all cyanide ions are used for forming a complex anion, the first excessive drop of AgNO3 will make a silver cyanide precipitate.
Impurities in technical active substance (principle of method) (Annex IIA, point 4.1)	There are no impurities.

Analytical methods for residues

Soil (principle of method and LOQ) (Annex IIA, point 4.2)	Modification of Standard Methods for the Examination of Water and Wastewater, American Public Health Association, Washington, Method No. 413:Cyanide Method principle All cyanides are isolated from acided sample by distillation with help of the inert gas, allowing for 5 to 10 fold enrichment, and after that are determined photometrically. Cyanides react with chloramine T to produce chlorcyan, which yields in combination with pyridine and barbiture acid at pH 4 – 5 in red-purple colouring. Its intensity is measured at a wavelength of 578nm.

	The LOQ is 0.005 mg/l for enrichment factor 5.
Air (principle of method and LOQ) (Annex IIA, point 4.2)	1) The determination of cyanides content in workplace and storehouse atmospheres, and at combustion gases inlets from waste gas incinerators is done with COMPUR 4120 STATOX analyser operating with infrared detectors. Measuring range 0–50ppm (0–56mg.m-3). Manufacturer: Compur Monitors GmbH & Co. KG, Weissenseestrasse 101, D-81539 Munich, Germany. And there is another possibility: Using detection tubes designed for hydrogen cyanide determination, type: hydrogen cyanide 2/a, No. CH 25701, Detection tubes manufacturer: Dräger Safety, AG&Co.KGaA, Lubeck, Germany. measuring range for 5 pump strokes is: 2 – 30 ppm. The measuring range of the method depends on the number of strokes, e.g. for 40 strokes it is 0.25 - 3.75 ppm.
Water (principle of method and LOQ) (Annex IIA, point 4.2)	Modification of Standard Methods for the Examination of Water and Wastewater, American Public Health Association, Washington, Method No. 413:Cyanide Method principle All cyanides are isolated from acided sample by distillation with help of the inert gas allowing for 5 to 10 fold enrichment,and after that are determined photometrically. Cyanides react with chloramine T to produce chlorcyan, which yields in combination with pyridine and barbiture acid at pH 4 – 5 in red-

	purple colouring. Its intensity is measured at a wavelength of 578nm. The LOQ is 0.005 mg/l for enrichment factor 5.
Body fluids and tissues (principle of method and LOQ) (Annex IIA, point 4.2)	Modification of Standard Methods for the Examination of Water and Wastewater, American Public Health Association, Washington, Method No. 413:Cyanide Method principle All cyanides are isolated from acided sample by distillation with help of the inert gas allowing for 5 to 10 fold enrichment and after that are determined photometrically. Cyanides react with chloramine T to produce chlorcyan, which yields in combination with pyridine and barbiture acid at pH 4 – 5 in red-purple colouring. Its intensity is measured at a wavelength of 578nm. The LOQ is 0.005 mg/l for enrichment factor 5.
Food/feed of plant origin (principle of method and LOQ for methods for monitoring purposes) (Annex IIIA, point IV.1)	In its use, HCN does not come in contact with food or feed
Food/feed of animal origin (principle of method and LOQ for methods for monitoring purposes) (Annex IIIA, point IV.1)	In its use, HCN does not come in contact with food or feed

Chapter 3: Impact on human health

Absorption, distribution, metabolism and excretion in mammals (Annex IIA, point 6.2)

Rate and extent of oral absorption:	HCN is a gas at body temperature. HCN and cyanates are readily absorbed from water solutions.

	Rate of oral absorption is considered 100 %.
Rate and extent of dermal absorption:	Gaseous hydrogen cyanide may be absorbed by skin; ratio of inhalatory/dermal absorption is estimated to be 300/1.
Rate and extent of absorption on inhalation	HCN is readily absorbed on inhalation. Initial lung retention is 100 %.
Distribution:	HCN is after absorption quickly, within seconds, distributed by blood into all tissues.
Potential for accumulation:	Hydrogen cyanide does not accumulate in organism. Thiocyanate concentration in blood may increase as a result of repeated exposure to HCN.
Rate and extent of excretion:	CN is excreted as thiocyanate, renal clearance: half-time of thiocyanate = 4 h – 2 d.
Toxicologically significant metabolite	Cyanide ion transformed to Thiocyanate

Acute toxicity (Annex IIA, point 6.1)

Rat LD50 oral	3.1 mg/kg bw as cyanide(i.e., 5.7 mg/kg .bw NaCN)
Rat LD50 dermal	6.7 mg/kg bw (rabbit, water solution of HCN)
Rat LC50 inhalation	493 mg/m3 (5 minutes) 173 mg/m3 (30 minutes) 158 mg/m3 (60 minutes) Little change expected at longer exposures.
Skin irritation	No primary data on skin irritation are available due to the inherent difficulty of performing such studies for gases in general. Apart from this, high toxicity

	of CN- makes it impossible to perform such studies using liquid HCN or solutions of cyanides as this would lead to immediate death of the animal following dermal absorption. .
Eye irritation	No primary data on eye irritation are available due to the inherent difficulty of performing such studies for gases in general. Apart from this, high toxicity of CN- makes it impossible to perform such studies using liquid HCN or solutions of cyanides as this would lead to immediate death of the animal following dermal absorption. Mild irritation reported in men.
Skin sensitization (test method used and result)	No primary data on skin senstization are available due to the inherent difficulty of performing such studies for gases in general. Apart from this, high toxicity of CN- makes it impossible to perform such studies using liquid HCN or solutions of cyanides as this would lead to immediate death of the animal following dermal absorption. Mild irritation is reported in men..

Repeated dose toxicity (Annex IIA, point 6.3)

Species/ target / critical effect	
Lowest relevant oral NOAEL / LOAEL	NOAEL: 10mg/kg/day , 2-year dietary study in rats (summary in DOC IIIA 6.5b) ,(top dose)
Lowest relevant dermal NOAEL / LOAEL	Not available.
Lowest relevant inhalation NOAEL / LOAEL	180 day ,rats and monkeys LOAEL: 25 ppm cyanogens (corresponding to 25 ppm CN or 30mg HCN /m3), lower body weight , transient change in behaviour

	NOAEL: 11 ppm cyanogens (corresponding to 11 ppm CN or 13.2 mg/ m3) (Summary in DOC IIIA, section 6.4.3a).

Genotoxicity (Annex IIA, point 6.6)	No genotoxic risk (Discussion in DOC IIA, section 3.6)

Carcinogenicity (Annex IIA, point 6.4) Non-carcinogenic

Species/type of tumour	No tumours have been observed at combined chronicity – carcinogenictity study in rats and mice
lowest dose with tumours	No tumours have been observed.

Reproductive toxicity (Annex IIA, point 6.8).

Species/ Reproduction target / critical effect	No relevant effects on reproduction were observed.
Lowest relevant reproductive NOAEL / LOAEL	NOAELs ranged from 1-26 mg/kg, rats and mice, always top doses
Species/Developmental target / critical effect	No effect observed
Lowest relevant developmental NOAEL / LOAEL	rat, NOAEL 3.3 mg CN/kg bw (top dose)

Neurotoxicity / Delayed neurotoxicity (Annex IIIA, point VI.1)

Species/ target/critical effect	Increased mortality rate and serious neurological disorders (tremor, ataxia, cerebral cells kill) were observed in laboratory animals at concentrations 50mg/m3 HCN).
Lowest relevantl NOAEL / LOAEL.	NOAEL s ranged from 4.7 to 25 mg/kg.bw, rats monkeys and mice. Duration of studiesranged from 13 weeks to 2 years.

Other toxicological studies (Annex IIIA, VI/XI)

Goitrogenic effects found in exposed animals and humans.	Thyrotropic effects in rats at a dose in water 3mg/kg bw of KCN. (Summary in DOC IIIA.6.8.1b; discussion also in DOC IIA.3.9.2.)

Medical data (Annex IIA, point 6.9)

	Inhalation of hydrogen cyanide in concentrations >120mg/m3 may be fatal. Chronic occupational exposure to HCN concentrations approximately 17 mg/m3 revealed a high prevalence of neurological, cardiovascular and gastrointestinal symptoms at concentrations about 17 mg/m3, mild symptoms at concentrations in the rage 5 to 13 mg/m3. Thyroid enlargement has been observed in workers exposed still lower concentrations in air for two years, but no symptoms and toxic effects at concentrations <3.6 mg/m3.

Summary (Annex IIA, point 6.10)	Value	Study	Safety factor

ADI *			
AOEC (Operator/Worker Exposure	3 mg/m^3	Toxicokinetic studies in human adults (Schulz et al., 1982,1984)	
AEC (non professionals)	3 mg/m^3	Toxicokinetic studies in human adults (Schulz et al., 1982,1984)	
AOEL (Operator/Worker Exposure) (acute)	0.48 mg/kg bw per day***.	Toxicokinetic studies in human adults (Schulz et al., 1982,1984)	1
AEL (non professionals, by-standers) (acute)	0.48 mg/kg bw per day***.	Toxicokinetic studies in human adults (Schulz et al., 1982,1984)	1
AOEL/AEL (Operator/Worker Exposure) (chronic)	0.1 mg/kg bw per day***	2-year studies in rats (inhalation – NTP 1994, oral – Howard, Hanzal, 1955)	100
AOEL/AEL (Operator/Worker Exposure) (medium term)	0.1 mg/kg bw per day***	2-year studies in rats (inhalation – NTP 1994, oral –	100

		Howard, Hanzal, 1955)	
Drinking water limit	0.05mg/l **		
ARfD (acute reference dose)	0.48 mg/kg bw***		

* no residues in food or feed; AEL (chronic) may serve as estimate for ADI, DOC IIA 3.11

** Czech Republic

*** equal to AEL (acute), DOC IIA 3.11

Acceptable exposure scenarios (including method of calculation)

Production	Concentration of HCN in the production hall is continuously monitored and each surpassing of OEL is signalised. Workers are approx. 90% of working hours in the control room, isolated from the production hall.
Professional users	Recommended HCN occupational concentration in treated structures is 10,000mg/m3 (= 9,000 ppm). Professional exposure of persons carrying out fumigation of closed spaces with hydrogen cyanide is for safety reasons reduced by using whole body gas-tight protective clothing (ČSN EN 464), special breathing apparatuses with filter-ventilation units (ČSN EN 132 and ČSN EN 133), rubber gloves (ČSN EN 374-1) and rubber boots (ČSN EN 346). Exposure of wood in special hermetised chambers reduces substantially the potential exposure of operators.
Non-professional users	Non-professional usage is not permitted.

	.
Indirect exposure as a result of use	Structures (or subjects) treated by fumigation may be opened and used only after being thoroughly ventilated to 3mg/m3. Exposure of bystanders and re-entering persons is discussed in DOC IIB 8.2.3.

Chapter 4: Fate and Behaviour in the Environment

Route and rate of degradation in water (Annex IIA, point 7.6, IIIA, point XII.2.1, 2.2)

Hydrolysis of active substance and relevant metabolites (DT50) (state pH and temperature)	pH_____: -
	pH_____: -
	pH_____: -
Photolytic / photo-oxidative degradation of active substance and resulting relevant metabolites	Direct photolysis of HCN does not practically occur.
Readily biodegradable (yes/no)	No
Biodegradation in seawater	Hydrogen cyanide does not spread into sea water.
Non-extractable residues	-
Distribution in water / sediment systems (active substance)	Hydrogen cyanide does not spread into surface waters, groundwater and sediments.
Distribution in water / sediment systems (metabolites)	Hydrogen cyanide does not spread into surface waters, groundwater and sediments.

Route and rate of degradation in soil (Annex IIIA, point VII.4, XII.1.1, XII.1.4; Annex VI, para. 85)

Mineralization (aerobic)	Not applicable
Laboratory studies (range or median, with number of measurements, with regression coefficient)	Not applicable

Field studies (state location, range or median with number of measurements)	Not applicable
	Not applicable
Anaerobic degradation	Not applicable
Soil photolysis	Not applicable
Non-extractable residues	Not applicable
Relevant metabolites - name and/or code, % of applied a.i. (range and maximum)	Not applicable
Soil accumulation and plateau concentration	Not applicable

Adsorption/desorption (Annex IIA, point XII.7.7; Annex IIIA, point XII.1.2)

Ka , Kd Ka_{oc} , Kd_{oc} pH dependence (yes / no) (if yes type of dependence)	Not applicable

Fate and behaviour in air (Annex IIIA, point VII.3, VII.5)

Direct photolysis in air	Direct photolysis of HCN does not practically occur.
Quantum yield of direct photolysis	Not applicable
Photo-oxidative degradation in air	Not applicable
Volatilization	Not applicable

Monitoring data, if available (Annex VI, para. 44)

Soil (indicate location and type of study)	No
Surface water (indicate location and type of study)	No
Ground water (indicate location and type of study)	No
Air (indicate location and type of study)	No

Chapter 5: Effects on Non-target Species

Toxicity data for aquatic species (most sensitive species of each group)
(Annex IIA, point 8.2, Annex IIIA, point 10.2)

Species	Time-scale	Endpoint	Toxicity
Fish			
Fish Salmo gairdnei	96 hrs.	LC50	0.042 mg/l
Invertebrates			
Daphnia Daphnia magna	48 hrs.	EC50	1.07 mg/l
Algae			
Scenedesmus subspicatus	72 hrs.	EC50	0.04mg/l
Microorganisms			
Data not found.			

Effects on earthworms or other soil non-target organisms

Acute toxicity to …….. (Annex IIIA, point XIII.3.2)	Not applicable for intended usage of the substance.
Reproductive toxicity to	Not applicable for intended usage of the substance.

…… (Annex IIIA, point XIII.3.2)	

Effects on soil micro-organisms (Annex IIA, point 7.4)

Nitrogen mineralization	Not applicable for intended usage of the substance.
Carbon mineralization	Not applicable for intended usage of the substance.

Effects on terrestrial vertebrates

Acute toxicity to mammals (Annex IIIA, point XIII.3.3)	Not applicable for intended usage of the substance.
Acute toxicity to birds (Annex IIIA, point XIII.1.1)	Not applicable for intended usage of the substance.
Dietary toxicity to birds (Annex IIIA, point XIII.1.2)	Not applicable for intended usage of the substance.
Reproductive toxicity to birds (Annex IIIA, point XIII.1.3)	Not applicable for intended usage of the substance.

RMS: Czech Republic	Hydrogen cyanide PT 18	

Effects on honeybees (Annex IIIA, point XIII.3.1)

Acute oral toxicity	Not applicable for intended usage of the substance.
Acute contact toxicity	Not applicable for intended usage of the substance.

Effects on other beneficial arthropods (Annex IIIA, point XIII.3.1)

Acute oral toxicity	Not applicable for intended usage of the substance.
Acute contact toxicity	Not applicable for intended usage of the substance.
Acute toxicity to ……..	Not applicable for intended usage of the substance.

Bioconcentration (Annex IIA, point 7.5)

Bioconcentration factor (BCF)	BCF = 0.73 Hydrogen cyanide has low bioaccumulation potential.
Depration time (DT50) (DT90)	Not applicable
Level of metabolites (%) in organisms accounting for > 10% of residues	Not applicable

APPENDIX II: LIST OF INTENDED USES

Hydrogen cyanide has been evaluated for its use in fumigantion to kill rodents (Product Type 14 of the Biocidal Products Directive). It is applied as gas gradually evaporating from an inert sorbent and can be used only by adequately trained profesional users.

The product URAGAN D 2 was submitted by the applicant for evaluation. It is the active substance as manufactured sorbed onto an inert sorbent. The prescribed concentration of hydrogen cyanide vapors in fumigated structures is 10g/m^3.

The structures to be fumigated include storehouses, depositories, transport facilities, containers, libraries and other buildings.

APPENDIX III: LIST OF STUDIES

Data protection is claimed by the applicant in accordance with Article 12.1(c) (i) and (ii) of Council directive 98/8/EC for all study reports marked "Y" in the "Data Protection Claimed" column of the table below. For studies marked Yes(i) data protection is claimed under Article 12.1(c) (i) , for studies marked Yes(ii) data protection is claimed under Article 12.1(c) (ii). These claims are based on information from the applicant. It is assumed that the relevant studies are not already protected in any other Member State of the European Union under existing national rules relating to biocidal products. It was however not possible to confirm the accuracy of this information.

References listed by reference number in DOC IV A and IVB:

Supplementary literature listed by DOC III A or B section number:

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A1 A3		2006	Hazardous Substance Data Bank (HSDB), National Library of Medicine´s TOXNET system (state in February 2006): Hydrogen cyanide *Peer reviewed*	N	n/a
DOC IV A2 A6.2, A6.7, A6.8.1, A6.9, A6.10,A 7.1.4		2004	ATSDR 1997 Toxicological Profile for Cyanide, U.S. Department of Health and Human Services, September 2004.	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A3	Rambeau M.	2001	Delphine Benitez, S. Dupuis* and P. Ducom HYDROGEN CYANIDE AS AN IMMEDIATE ALTERNATIVE TO METHYL BROMIDE FOR STRUCTURAL FUMIGATIONS Ministry of Agriculture, Fisheries and Food. National Laboratory of Plant Protection, Research Unit on Fumigation and Stored Products Protection, Chemin d'Artigues, 33150 Bordeaux-Cenon, France [*e-mail: lnds@easynet.fr]	N	n/a
DOC IV A4 A3.5, A.6			Data From SRC PhysProp Database	N	n/a
DOC IV A5a, A5b A7.4.1.1._1		1980	US EPA (1980) Ambient Water Quality Criteria for Cyanides. 440/5-80-037 (published).	N	n/a
DOC IV A6 A6.1.1, A6.1.2	Smyth H.F.	1969	Carpenter CP, Weil CS, et. Al. 1969. Range-finding toxicity data: List VII. Am Ind Hyg Assoc J 30: 470-476	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A7 A6.1.1	Ferguson H.C.	1962	Dilution of dose and acute oral toxicity. Toxicol Appl Pharmacol 4: 759-762.	N	n/a
DOC IV A8 A6.1.1, A6.1.1.1a, A6.1.2, A6.1.2a, A6.1.2b, A6.1.2d, A6.1.4.2a, A6.3.2 A6.9, A6.12	Balantyne Bryan	1988	Toxicology and Hazard Evaluation of Cyanide Fumigation Powders, Applied Toxicology Department, Union Carbide Corporation, Danbury, Connecticut 06817, Clinical Toxicology, 26 (5&6), 325-335	N	n/a
DOC IV A9 A6.3.2	B. Ballantyne	1983 b	Acute systemic toxicity of cyanides by topical application to the eye. J Toxicol, Cutan, Ocular Toxicol 2: 119-129 (DOC IVA /)	N	n/a
DOC IV A10 A6.1.4.2, A6.2 A6.1.2, A6.1.2c A.1.3, A6.3.2	Ballantyne B.	1983 a	. The influence of exposure route and species on the acute lethal toxicity and tissue concentrations of cyanide. In: Hayes AW, Schnell RC, Miya TS, eds. Developments in the science and practice of toxicology. New York, NY: Elsevier Science Publishers, 583-586	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A11 A6.1.3	Matijak-Schaper M Alarie Y.	1982	Toxicity of carbon monoxide, hydrogen cyanide and low oxygen. J Combust Toxicol 9:21-61.	N	n/a
DOC IV A12 A6.1.3a, A6.2, A6.4, A6.4a	J.M.McNerney, M.P.H., H.H.Schrenk, PhD.,	1960	The Acute Toxicity of Cyanogen, Industrial Hygiene Foundation, 4400 Fifth Avenue, Pittsburg 13, Pennsylvania, Industrial Hygiene Journal, 121 – 124	N	n/a
DOC IV A13 A6.1.4.2	Blac P, Hoan M, Mallin K	1985	Cyanide intoxication among silver-reclaiming workers. J Am Med Assoc 253: 367-371	N	n/a
DOC IV A14 A6.1.4.1, A6.1.4.2, A6.10	El Ghawabi SH, Gaafar MA, El-Saharti AA, et al.	1975	Chronic cyanide exposure: A clinical, radioisotope, and laboratory study. Br J Ind Med 32:215-219.	N	n/a
DOC IV A15 A6.1.4.1, A6.2 A6.4, A.4c, A6.9	Fairley A, Linton EC, Wild FE.	1934	The absorption of hydrocyanic acid vapour through the skin with notes on other matters relating to acute cyanide poisoning. J Hyg 34: 283-294	N	n/a
DOC IV A16 A6.1.4.2, A6.12	Bonsall JL.	1984	Survival without sequelae following exposure to 500 mg/m3 hydrogen cyanide. Hum Toxicol 3:57-60	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A17 A6.1.4.2, A6.2, A6.12	Chandra H, Gupta BN, Bhargava SK, Clerk SH, Mahendre PN	1980	Chronic cyanide exposure: a biochemical and industrial hygiene study. Journal of Analytical Toxicology, 3:161–165.	N	n/a
DOC IV A18 A6.2	Yamamoto K, Yamamoto Y, Hattori H, et al.	1982 .	Effects of routes of administration on the cyanide concentration distribution in the various organs of cyanide-intoxicated rats. Tohoku J Exp Med 137: 73-78	N	n/a
DOC IV A19 A6.2	Walton D.C., Witherspoon MG	1926	. Skin absorption of certain gases. J Pharmacol Exp Ther 26: 315-324	N	n/a
DOC IV A20 A6.2, A6.7, A6.10, A6.12		2004	IPCS (WHO, CICAD 61: Hydrogen cyanide and cyanides: human health aspects). CICAD 61	N	n/a
DOC IV A21 A6.2, A6.12	Schultz V	1984	Clinical pharmacokinetics of nitroprusside, cyanide, thiosulfate and thiocyanate. Clinical Pharmacokinetics, 9:239–251.	N	n/a
DOC IV A22 A6.3.1	Sousa A.B., Soto-Blanco B, Guerra JL, Kimura ET, Gorniak S	2002	Does prolonged oral exposure to cyanide promote hepatotoxicity and nephrotoxicity? Toxicology, 174:87–95.	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A23 A6.3.3	Valade M.P.	1952	Central nervous systém lesions in chronic experimental poisoning with gaseous hydrocyanic acid. Bull Acad Natl Med (Paris) 136: 280-285. (in French) (DOC IVA /)	N	n/a
DOC IV A24 6.4.1	Tewe O.O., Maner JH	1981	Performance and pathophysiological changes in pregnant pigs fed cassava diets containing different levels of cyanide. Research in Veterinary Science,30:147–151	N	n/a
DOC IV A25 A6.4.1, A6.7, A6.7a	Howard J. W., R. F. Hanzal	1955	Chronic Toxicity for Rats of Food Treated with Hydrogen Gyanide, Hazleton Laboratories, Falls Church, Va., Agricultural and Food Chemistry, Volume 3 No.4	N	n/a
DOC IV A26 A6.4.1, A6.9, A6.10	Philbrick D.J., Hopkins JB, Hill DC, et al.	1979	Effects of prolonged cyanide and thiocyanate feeding in rats. J Toxicol Environ Health 5:579-592.	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A27 A6.4.1a, A6.6.1, A6.6.1a, A6.8.2	NTP.	1993	Technical Report on toxicity studies of sodium cyanide (CAS No. 143-33-9) administered in drinking water to F344/N rats and B6C3Fl mice. Research Triangle Park, NC: National Toxicology Program, U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication 94-3386. NTP TOX 37. .	N	n/a
DOC IV A28 A6.4.1		1993	US EPA ydrogen cyanide (CASRN 74-90-8). US Environmental Protection Agency, Integrated Risk Information System.	N	n/a
DOC IV A29 A6.4.3, A6.4.3a	Lewis T.R., Anger WK, Te Vault RK	1984 .	Toxicity evaluation of sub-chronic exposures to cyanogen in monkeys and rats. J Environ Pathol Toxicol Oncol 5:151-163.	N	n/a
DOC IV A30 A6.2, A6.12	Ansell & Lewis	1970	Ansell M, Lewis FAS, A review of cyanide concentrations found in human organs: A survey of literature concerning cyanide metabolism, „normal", non-fatal and fatal bydy cyanide levels. Journal of Forensic Medicine, 17: 148-155	N	n/a

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DOC IV A31 A6.6.1, A6.6.1b	Kushi A., Matsumoto T, Yoshida D.	1983	Mutagen from the gaseous phase of protein pyrolyzate. Agric Biol Chem 47: 1979-1982	N	n/a
DOC IV A32 A6.6.1	De Flora S., Camoirano A, Zanacchi P, et al	1984	Mutagenicity testing with TA97 and TA102 of 30 DNA-damaging compouds, negative with other Salmonella strains. Mutat Res 134:159-165.	N	n/a
DOC IV A33 A6.6.1	Friedman M.A., Staub J.	1976 .	Inhibition of mouse testicular DNA synthesis by mutagens and carcinogens as a potential simple mammalian assay for mutagenesis. Mutat Res 37: 67-76	N	n/a
DOC IV A34 A6.6.1	Kubo T, Urano K, Utsumi H	2002	Mutagenicity characteristics of 255 environmental chemicals. J Health Sci 48(6):545-554.	N	n/a
DOC IV A35 A6.6.1	Bhattacharya R., Laskshmana Rao PV.	1997	Cyanide induced DNA fragmentation in mammalian cell cultures. Toxicology 123:207-215	N	n/a
DOC IV A36 A6.6.1	Henderson L., Wolfreys A, Fedyk J, et al.	1998	The ability of the Comet assay to discriminate between genotoxins and cytotoxins. Mutagenesis 13:89-94	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A37 A6.6.1, A6.6.4	Yamamoto H., Mohanan PV	2002.	Melatonin attenuates brain mitochondria DNA damage induced by potassium cyanide in vivo and in vitro. Toxicology 179:29-36.	N	n/a
DOC IV A38 A6.6.4	Friedman M.A., Staub J.	1976	Inhibition of mouse testicular DNA synthesis by mutagens and carcinogens as a potential simple mammalian assay for mutagenesis. Mutat Res 37: 67-76	N	n/a
DOC IV A39 A6.9, A7.1.1.2.1	Fechter L.D., Chen G, Johnson DL.	2002	Potentiation of noise-induced hearing loss by low concentrations of hydrogen cyanide in rats. Toxicol Sci 66(1):131-138.	N	n/a
DOC IV A40 A6.12	Vladimír Pitschmann	2004	Vojenská chemie kyanovodíku HCN, , Brno 2004, str. 28,Borowitz J. L., Isom G.E. Baskin S.I. v knize Somani S.M. Romano J.A. (Eds.): Chemical Warfare Agents: Toxicity at Low Levels. CRC Press, Boca Raton 2001	N	n/a
DOC IV A41	Manyonda, I.T.	1986	Shaw, D.E, Foulkes, A., Osborn, D.E Industrial exposure to hydrogen cyanide: implications for treatment British Medical Journal, Volume 293, 1986	N	n/a

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DOC IV A42 A6.12	Gettler A.O., Baine JO	1938	The toxicity of cyanide. American Journal of Medical Science, 195:182–198.	N	n/a
DOC IV A43 A7.1.1.1.1	Krieble V. E	1930	McNally, J. G.: The Hydrolysis of Hydrogen Cyanide by Acids II, J. Am. Chem, Soc., 1929, 51, 3368.	No	n/a
DOC IV A44 A7.1.1.1.1	Krieble V. E	1929	McNally, J. G.: The Hydrolysis of Hydrogen Cyanide by Acids I, J. Am. Chem, Soc., 1929, 51, 3368.	No	n/a
DOC IV A45			Kirk-Othmer Encyclopedia of Chemical Technology (4th Edition)	No	n/a
DOC IV A46 A7.1.1.2.1	Klecka G.M., Landi LP, Bodner KM.	1985	Evaluation of the OECD activated sludge, respiration inhibition test. Chemosphere 14:1239-1251.	N	n/a
DOC IV A47 A7.1.1.11, A7.1.1.1.2, A7.1.1.2.1, A7.1.3, A7.1.4, A7.2, A7.3.1			JACC No 53, Cyanides of Hydrogen, Sodium and Potasium, and acetone Cyanohydrin (CAS No. 74-90-8, 143-33-9, 151-50-8 and 75-86-5), ECETOC JACC REPORT No. 53 European Centre for Ecotoxicology and Toxicology of Chemicals Volume I	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A48 A7.1.1.11, A7.1.1.1.2, A7.1.1.2.1, A7.1.3, A7.1.4, A7.2, A7.3.1			JACC No 53, Cyanides of Hydrogen, Sodium and Potasium, and acetone Cyanohydrin (CAS No. 74-90-8, 143-33-9, 151-50-8 and 75-86-5), ECETOC JACC REPORT No. 53 European Centre for Ecotoxicology and Toxicology of Chemicals, Volume II	N	n/a
DOC IV A50 A7.4.1.1	Smith L.L., Broderius S.J., Osied D.M., Kimbal G.L., Koenst W.M.,		Acute Toxicity of Hydrogen Cyanide to Freshwater Fishes, Paper No. 9954, under Grant No. R802914	N	n/a
DOC IV A51		2007	Crop Research Institute (CRI) Evaluation of URAGAN (HCN) Field Efficacy – CRI - 2007	Y	
DOC IV A52	Rambeau M.	1999	Hydrogen cyanide as an immediate alternative to methyl bromide for structural fumigations D. BENITEZ, S. DUPUIS, P. DUCOM	Y	

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A53 A4.2		2002	Modification of Standard Methods for the Examination of Water and Wastewater, American Public Health Association, Washington, Method No. 413:Cyanide	Y	
DOC IV A54	Walton D.C	1925	Witherspoon MG. 1926. Skin absorption of certain gases. J Pharmacol Exp Ther 26: 315-324	N	n/a
DOC IV A55			Compur Statox 4120	N	n/a
DOC IV A56 A6.8.1a	Benito Soto-Blanco, Silvana L. Go´rniak	2004	Prenatal toxicity of cyanide in goats—a model for teratological studies in ruminants. Theriogenology 62: 1012–1026	N	n/a
DOC IV A57 A6.8.1b	Altamir Benedito de Sousa, Paulo C´esar Maiorka, Ivair Donizete Goncalves, L´ılian Rose Marques de S´a, Silvana Lima G´orniak	2007	Evaluation of effects of prenatal exposure to the cyanide and thiocyanate in Wistar rats. Reproductive Toxicology 23: 568–577	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A58		2002	European Union Risk Assessment Report Acetonitrile European Commission Joint Research Centre Priority List Volume 18	N	n/a
DOC IV A59 A6.2		2005	Acetone Cyanohydrin. Acute Exposure Guideline Levels August	N	n/a
DOC IV A60 A6.2	Schultz V., Gross R, Pasch T, Busse J, Loescheke G	1982	Cyanide toxicity of sodium nitroprusside in therapeutic use with and without sodium thiosulfate. Klinische Wochenschrift,60:1393–1400.	N	n/a
DOC IV A61 A6.10	Way J.L.	1984	Cyanide intoxication and its mechanism of antagonism. Annual Review of Pharmacology and Toxicology, 24:451–481.	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A62	Banerjee et al	1997	Kishore K. Banerjee, PhD, A. Bishayee, PhD, P. Marimuthu, MSc Evaluation of Cyanide Exposure and Its Effect on Thyroid Function of Wokers in a Cable Industry JOEM, Volume 39, Number 3, March 1997	N	n/a
DOC IV A63 A6.4.1	Tewe O.O., Maner JH	1985	Cyanide, protein and iodine interactions in the performance and metabolism of rats. Journal of Environmental Pathology and Toxicology, 6:69–77.	N	n/a
DOC IV A64 A6.12	Jackson L.C., Bloch EF, Jackson RT, Chandler JP, Kim YL, Malveaux F	1985	Influence of dietary cyanide on immunoglobulin and thiocyanate levels in the serum of Liberianadults. Journal of the National Medical Association, 77:777–782.	N	n/a
DOC IV A65 A6.2	Schultz et al.	1982	Detoxification of cyanide in a newborn child Klinische Wochenschrift,60,527-528	N	n/a
DOC IV A66 A6.2	Schultz V., Bonn R, Kindler J	1979	[Kinetics of elimination of thiocyanate in 7 healthy subjects and 8 subjects with renal failure.] Klinische Wochenschrift, 57:243–247 (in German).	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A67 A6.2	Schultz et al.	1983	Resorption of hydrocyanic acid from linseed, Leber Magen Darm 13: 10-14.	N	n/a
DOC IV A68 A6.2	Schultz et al.	1979	Counteraction of cyanide poisoning by thiosulphate when administering sodium nitroprusside as hypotensive treatment, Klinische Wochenschrift 57, 905-907 .	N	n/a
DOC IV A69	Schulz V.,	1978	Thiozyanat-Vergiftung bei der antihypertensiven Therapie mit Natriumnitroprussid Medizinische Universitätsklinik Köln und Department für Innere Medizin der Medizinischen Hochschule Hannover	N	n/a
DOC IV A70	Olumide O.,Tewe and Jerome H. Manert	1980	Long-Term and Carry-Over Effect of Dietary Inorganic Cyanide (KCN) in the Life Cycle Performance and Metabolism of Rats Department of Animal Science, University of Ibadan, Ibadan, Nigeria. Centro International De Agricultura Tropical, Colombia, South America	N	n/a

Reference No DOC IV "A" Section No DOC III A	Author(s)	Year	Title. Source (where different from company) Company, Report No. GLP (where relevant) / (Un)Unpublished	Data Protection Claimed (Yes/No)	Owner
DOC IV A71 A6.9	Jackson L.C	1988	Behavioural effects of chronic sublethal dietary cyanide in an animal model: Implications for humans consuming cassava (Manihot esculenta). Human Biology, 60:597–614.	N	n/a
DOC IV A72 A6.4.3, A6.5a, A6.6.1a, A6.7			National Toxicology Program (NTP)(1994). Toxicology and carcinogenesis of acetonitrile (CAS Nº 75-05-8) in F344/N rats and B6C3F1 mice (Inhalation studies). TR 447. NIH Publication Nº 94-3363. US Department of Health and Human Services, Public Health Service, National Institutes of Health.	N	n/a
DOC IV A73	R.C. Brandys, G.M. Brandys	2006	Global occupational exposure limits for over 5,000 specific chemicals Occupational & Environmental Health Consulting Services, Hinsdale, Ill.	N	n/a
DOC IV A74		2002	Technical Guidance Document on Risk Assessment Part II, BCF	N	n/a
DOC IV A75		2006	EN 335-1 Durability of wood and wood-based product, Definition of use classes- Part 1: General	N	n/a

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DOC IV A76 A6.6.4	Monsanto Co.	1984	CHO/HGPRT mammalian cell forward mutation assay, acetone cyanohydrin. St. Louis, MO, Monsanto Co. (Report PR-82-204).	N	n/a
DOC IV A77 A6.4.3, A6.8.2	Monsanto Co.	1985	Male fertility study of Sprague-Dawley rats exposed by the inhalation route to acetone cyanohydrin. St. Louis, MO, Monsanto Co. (Report ML-82-144; US EPA/OPTS Public Files No. 878216404).	N	n/a
DOC IV A78 A6.8.2	Monsanto Co.	1985	Female fertility study of Sprague Dawley rats exposed by the inhalation route to acetone cyanohydrin. St. Louis, MO, Monsanto Co. (Report ML-82 145; US EPA/OPTS Public Files No. 878216396).	N	n/a
DOC IV A79 A6.4.3	Monsanto Co.	1984	Three-month inhalation toxicity of acetone cyanohydrin in male and female Sprague-Dawley rats. St. Louis, MO, Monsanto Co. (Report ML-82-143; US EPA/OPTS Public Files No. 878216397).	N	n/a

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DOC IV A80 A7.4.2	By E. A. . PARKIN , M.Sc., PH.D., D.I.C.	1937	THE TOXICITY OF HYDROGEN CYANIDE TO CERTAIN WOOD-BORING INSECTS Entomology Section, Forest products Research Laboratory, Princes Risborough, Bucks	N	n/a
DOC IV A81 A7.1.1.1.2	D. J. Lary,	2004	Atmospheric pseudohalogen chemistry, Atmos. Chem. Phys. Discuss., 4, 5381–5405, 2004 www.atmos-chem-phys.org/acpd/4/5381/ SRef-ID: 1680-7375/acpd/2004-4-5381 © European Geosciences Union	N	n/a
DOC IV A82 A7.1.1.2.1	Dumestre Alain	1997	, THERESE CHONE, JEAN-MARIE PORTAL, MYLENE GERARD, AND JACQUES BERTHELIN Cyanide Degradation under Alkaline Conditions by a Strain of Fusarium solani Isolated from Contaminated Soils, APPLIED AND ENVIRONMENTAL MICROBIOLOGY, 0099-2240/97/$04.0010 July 1997, p. 2729–2734	N	n/a

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DOC IV A83 A7.1.1.2.1			Cyanide Degradation under Alkaline Conditions by a Strain of Fusarium solani Isolated from Contaminated Soils,APPLIED AND ENVIRONMENTAL MICROBIOLOGY, 1997, p. 2729–2734 ALAIN DUMESTRE,† THERESE CHONE, JEAN-MARIE PORTAL, MYLENE GERARD, AND JACQUES BERTHELIN*	N	n/a
DOC IV A84 A7.1.4.2			Hydrogen cyanide: An acute toxicity study with the daphnia Daphnia magna Strauss, Research Institute of Organic syntheses, Centre for ekotoxicology, toxicology an analytics, Pardubice – Rybitví, Czech Republic,Report No. 1514/L (unpublished), 2002-02-18	N	n/a
DOC IV A85 A7.1.4.3			Growth Inhibition of Green Algae (Scenedesmus subspicatus Brinkmann 1953/SAG 86.81) by hydrogen cyanide liquid stabilized, Research Institute of Organic syntheses, Centre for ekotoxicology, toxicology an analytics, Pardubice – Rybitví, Czech Republic,Report No. 1522/L (unpublished), 2002-03-20	N	n/a

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A3.1, A3.3.1, A3.3.2, A3.3.3	Budavari S., ed	1989	Merck index: An encyclopedia of chemicals, drugs, and biologicals. 11 th ed. Rahway, NJ: Merck &Co., Inc.	N	n/a
A3.1.1, A3.11, A3.12, A3.15	Jenks W.R.	1979	Cyanides. In: Grayson, M, ed. Kirk-Othmer encyclopedia of chemical technology. New York, NY: John Wiley and Sons, Inc., 307-334	N	n/a
A3.11, A3.15	Quincy	1997	Fire Protection Guide to Hazardous Materials. 12 ed, MA: National Fire Protection Association, 1997	N	n/a
A3.2, A7.1.3	Daubert T.E., Danner RP	1989	Physical and Thermodynamic Properties of Pure Chemicals Data Compilation Washington, DC: Taylor and Francis	N	n/a
A3.9, A7.4.2	Hansch C., Leo, A., D. Hoekman.	1995	Exploring QSAR - Hydrophobic, Electronic, and Steric Constants. Washington, DC: American Chemical Society., p. 3	N	n/a

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A6.1.3		1959	Toxicology and Applied Pharmacology. (Academic Press, Inc., 1 E. First St., Duluth, MN 55802) V.1- 1959- v. 42, p. 417, (TXAPA9);	N	n/a
A6.1.3		1987	Fundamental and Applied Toxicology. (Academic Press, Inc., 1 E. First St., Duluth, MN 55802) V.1-40, 1981-97. For publisher information, see TOSCF2 v. 9, p. 236, (FAATDF)	N	n/a
A6.1.3	AMRL	1971	The acute toxicity of brief exposures to hydrogen fluoride, hydrogen chloride, nitrogendioxide, and hydrogen cyanide singly and in combination with carbon monoxide. Wright-Patterson Air Force Base, OH: Aerospace Medical Research Laboratory. AD751442	N	n/a
A6.1.3	Hume A.S., Mozigo JR, McIntyre B, et al.	1995	Antidotal efficacy of alpha-ketoglutaric acid and sodium thiosulfate in cyanide poisoning. Clin Toxicol 33(6):721-724.	N	n/a
A6.1.3	Monsanto Co.Report	1985	One-month inhalation toxicity of acetone cyanohydrin in male and female Sprague-Dawley rats. St Louis, Monsato Co. Report ML-81-178/810068 (US EPA/OPTS Public Files No. 878216393).	N	n/a

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A6.1.3 A6.9	Purser DA, Grimshaw P, Berrill KR.	1984	Intoxication by cyanide in fires: A study in monkeys using polyacrylonitrile. Arch Environ Health 39:394-400. IPCS, ATSDR	N	n/a
A6.10	Maduh E.U., Johnson JD, Ardelt BK, Borowitz JL, Isom GE	1988	Cyanide-induced neurotoxicity: Mechanisms of attenuation by chlorpromazine. Toxicology and Applied Pharmacology, 96:60–67.	N	n/a
A6.10	Pettersen JC, Cohen SD	1993	The effects of cyanide on brain mitochondrial cytochrome oxidase and respiratory activities. Journal of Applied Toxicology, 13:9–14.	N	n/a
A6.10, A6.12	Hardy H.L., Jeffries WM, Wasserman MM, Waddell WR	1950	Thiocyanate effect following industrial cyanide exposure. New England Journal of Medicine, 242:968–972	N	n/a
A6.10, A6.12	US EPA	1990	Summary review of health effects associated with hydrogen cyanide. Health issue assessment. Research Triangle Park, NC, US Environmental Protection Agency, Office of Research and Development (EPA/600/8-90/002F).	N	n/a

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A6.12	Anderson R.A., Harland WA	1982	Fire deaths in the Glasgow area. III. The role of hydrogen cyanide. Medicine, Science and the Law, 22:35–40	N	n/a
A6.12		1986	American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, OH: American Conference of Governmental Industrial Hygienists,p. 314]	N	n/a
A6.12	Abuye C., Kelbessa U, Wolde-Gebriel S	1998	Health effects of cassava consumption in south Ethiopia. East African Medical Journal, 75:166–170	N	n/a
A6.12	Alarie Y.	2002	Toxicity of fire smoke. Crit Rev Toxicol 32(4):259-289	N	n/a
A6.12	ATSDR	1991	Case studies in environmental medicine. Atlanta, GA, US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.	N	n/a

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A6.12	Birky M.M., Clarke FB	1981	Inhalation of toxic products from fires. Bulletin of the New York Academy of Medicine, 57:997–1013.	N	n/a
A6.12	Boivin M.J.	1997	An ecological paradigm for a health behavior analysis of “Konzo,” a paralytic disease of Zaire from toxic cassava. Social Science and Medicine, 45:1853–1862.	N	n/a
A6.12	Chen K.K., Rose CL.	1952	1952. Nitrite and thiosulfate therapy in cyanide poisoning. J Am Med Assoc 149:113-119.	N	n/a
A6.12	Cherian M.A., Richmond I.	2000	Fatal methane and cyanide poisoning as a result of handling industrial fish: A case report and review of the literature. J Clin Pathol 53:794-795	N	n/a
A6.12	Cliff J., Coutinho J	1995	Acute intoxication from newlyintroduced cassava during drought in Mozambique. Tropical Doctor, 25:193.	N	n/a

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A6.12	Cooles P.	1988	Diabetes and cassava in Dominica. Tropical and Geographical Medicine, 40:272–273.	N	n/a
A6.12	Delange F., Hershman JM, Ermans AM	1971	Relationship between the serum thyrotropin level, the prevalence of goiter and the pattern of iodine metabolism in Idjwi Island. Journal of Clinical Endocrinology and Metabolism, 33:261–268.	N	n/a
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A6.12	DOA	1976	Estimates of the toxicity of hydrocyanic acid vapours in man. Aberdeen Proving Ground, MD: Department of the Army. EBTR76023. ADA02850	N	n/a
A6.12	Drinker P.	1932	Hydrocyanic acid gas poisoning by absorption through the skin. J Ind Hyg 14:1-2	N	n/a
A6.12	Dudley H.C., Sweeney TR, Miller JW.	1942	Toxicology of acrylonitrile (vinyl cyanide). II: Studies of effects of daily inhalation. J Ind Hyg Toxicol 24:255-258	N	n/a

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A6.12	Ernesto M., Cardoso AP, Nicala D, Mirione E, Massaza F, Cliff J, Haque MR, Bradbury JH	2002	Persistent konzo and cyanogen toxicity from cassava in northern Mozambique. Acta Tropica, 82:357–362.	N	n/a
A6.12	Gill G.	1996	Tropical diabetes? Tropical Doctor, 26:1–3.	N	n/a
A6.12	Gosselin, R.E., R.P. Smith, H.C. Hodge	1984	Clinical Toxicology of Commercial Products. 5th ed. Baltimore: Williams and Wilkins,., p. III-126]	N	n/a
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A6.12	Hugh-Jones P.	1955	Diabetes in Jamaica. Lancet, 2:891–897. Concise International Chemical Assessment Document 61 36	N	n/a
A6.12	JECFA	1993	Cyanogenic glycosides. In: Toxicological evaluation of certain food additives and naturally occurring toxicants. Geneva, World Health Organization, 39th Meeting of the Joint FAO/WHO Expert Committee on Food Additives (WHO Food Additives Series 30)	N	n/a
A6.12	Kumar P., Das M, Kumar A.	1992	Health status of workers engaged in heat treatment (case hardening) plant and electroplating at cyanide bath. Indian J Environ Prot 12(3):179-183	N	n/a
A6.12	Lantum H.	1998	Spastic paraparesis–konzo in the Garoua Boulai Health District, East Province–Cameroon: A hidden Hydrogen cyanide and cyanides: Human health aspects 37	N	n/a
A6.12	Lasch E.E., El Shawa R.	1981	Multiple cases of cyanide poisoning by apricot kernels in children from Gaza. Pediatrics 68:5-7.	N	n/a

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A6.12	Lundquist P., Rammer L, Sorbo B	1989	The role of hydrogen cyanide and carbon monoxide in fire casualties: a prospective study. Forensic Science International, 43:9–14.	N	n/a
A6.12	Makene W.J., Wilson J.	1972	Biochemical studies in Tanzanian patients with ataxic tropical neuropathy. J Neurol Neurosurg Psychiatry 35:31-33	N	n/a
A6.12	McGlashan N.D.	1967	Geographical evidence on medical hypotheses. Tropical and Geographical Medicine, 19:333–344.	N	n/a
A6.12	Ministry of Health, Mozambique.	1984	Mantakassa: An epidemic of spastic paraparesis associated with chronic cyanide intoxication in a cassava staple area of Mozambique. 1. Epidemiology and clinical and laboratory findings in patients. Bull WHO 62:477-484	N	n/a

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A6.12	Mittal, S.; Gupta, K. S. and Gupta, Y. K	1981	Kinetics of Carboxylic Acid Catalysed Hydrolysis of Formamide: Evidence for Specific Hydronium Ion Catalysis, Indian J. Chem, 20A, 1220–1221.	N	n/a
A6.12	Miyakawa, S.; Cleaves, H. J.	2001	: Implications Based on the Hydrolytic Stabilities of Hydrogen Cyanide and Formamide, Journal of Applied Chemistry, 196	N	n/a
A6.12	Nahrstedt A.F.	1993	Cyanogenesis and food plants. In: van Beek TA, Breteler H, eds. Proceedings of the International Symposium on Phytochemistry and Agriculture, 22–24 April 1992, Wageningen. Oxford, Oxford University Press, pp. 107– 129.	N	n/a
A6.12	NIOSH	1976	Health hazard evaluation report. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Center for Disease Control, National Institute for Occupational Safety and Health. No. 74-129-268	N	n/a

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A6.12	Oluwole OSA, Onabolu AO, Cotgreave IA, Rosling H, Persson A, Link H	2002	Low prevalence of ataxic polyneuropathy in a community with high exposure to cyanide from cassava foods. Journal of Neurology, 249:1034–1040.	N	n/a
A6.12	Osuntokun BO	1981	Cassava diet, chronic cyanide intoxication and neuropathy in the Nigerian Africans. World Review of Nutrition and Dietetics, 36:141–173	N	n/a
A6.12	Peden NR, Taha A, McSorley PD, et al.	1986	Industrial exposure to hydrogen cyanide: Implications for treatment. Br Med J 293:538.	N	n/a
A6.12	Pentore R, Venneri A, Nichelli P	1996	Accidental choke cherry poisoning: early symptoms and neurological sequelae of an unusual case of cyanide intoxication. Italian Journal of Neurological Science, 17:233–235.	N	n/a
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A6.12	Rieders F	1971	Noxious gases and vapors. I: Carbon monoxide, cyanides, methemoglobin, and sulfhemoglobin. In: De	N	n/a
A6.12	Sayre JW, Kaymakcalan S	1964	Cyanide poisoning from apricot seeds among children in Central Turkey. New England Journal of Medicine, 270:.	N	n/a
A6.12	Singh BM, Coles N, Lewis P, et al.	1989	The metabolic effects of fatal cyanide poisoning. Postgrad Med J 65:923-925	N	n/a
A6.12	Suchard JR, Wallace KL, Gerkin RD	1998	Acute cyanide toxicity caused by apricot kernal ingestion. Ann Emerg Med 32(6):742-744.	N	n/a
A6.12	Sullivan, J.B. Jr., G.R. Krieger	1992	(eds.). Hazardous Materials Toxicology-Clinical Principles of Environmental Health. Baltimore, MD: Williams and Wilkins, p. 704	N	n/a

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A6.12	Tylleskar T, Banea M, Bikangi N, et al.	1992	Cassava cyanogens and konzo, an upper motoneuron disease found in Africa [erratum in Lancet 1992 Feb 15;339(8790):440]. Lancet 339(8787):208-211.	N	n/a
A6.12	Tylleskar T, Legue FD, Peterson S, et al.	1994	Konzo in the Central African Republic. Neurology 44(5):959-61	N	n/a
A6.12	VanderLaan WP, Bissell A.	1946	Effects of propylthiouracil and of potassium thiocyanate on the uptake of iodine by the thyroid gland of the rat. Endocrinology 39:157-160.	N	n/a
A6.12	Wexler J, Whittenberger JL, Dumke PR.	1947	The effect of cyanide on the electrocardiogram of man. Am Heart J 34:163-173	N	n/a

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A6.12	Zuidema PJ	1959	Cirrhosis and disseminated calcification of the pancreas in patients with malnutrition. Tropical and Geographical Medicine, 11:70–74.	N	n/a
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A6.2	Ansell & Lewis,	1970	A review of cyanide concentrations found in human organs: A survey of literature concerning cyanide metabolism, „normal“, non-fatal and fatal bydy cyanide levels. Journal of Forensic Medicine, 17: 148-155	N	n/a
A6.2,	Williams, 1959 – Williams RT	1959	Detoxification mechanisms, 2nd ed. London, Chapman and Hall, p. 393	N	n/a
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A6.3.2	Hugod C.	1981	Myocardial morphology in rabbits exposed to various gas-phase constituents of tobacco smoke: an ultrastructural study. Atherosclerosis 40: 181 - 190.	N	n/a
A6.7, A6.8.1	Doherty P.A., Ferm VH, Smith RP.	1982	Congenital malformations induced by infusion of sodium cyanide in the Golden hamster. Toxicol Appl Pharmacol 64:456-464.	N	n/a
A6.8.1	Frakes R.A., Sharma RP, Willhite CC, Gomez G	1986	Effect of cyanogenic glycosides and protein content in cassava diets on hamster prenatal development. Fundamental and Applied Toxicology, 7:191–198	N	n/a
A6.9	Hertting G.O., Kraupp E, Schnetz E, Wuketich ST	1960	Investigation about the consequences of a chronic administration of acutely toxic doses of sodium cyanide to dogs. Acta Pharmacologica et Toxicologica, 17:27–43	N	n/a

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