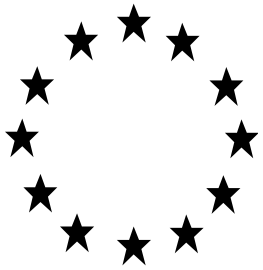
Regulation (EU) No 528/2012 concerning the making available on the market and use of biocidal products

Evaluation of active substances

Assessment Report



Hydrogen peroxide

Product-types 1-6

PT 1: Human hygiene biocidal products
PT 2: Private area and public health area disinfectants and other biocidal products
PT 3: Veterinary hygiene biocidal products
PT 4: Food and feed area disinfectants
PT 5: Drinking water disinfectants
PT 6: In-can preservatives

March 2015

Finland

CONTENTS

1. STATEMENT OF SUBJECT MATTER AND PURPOSE

1.1. Procedure followed

This assessment report has been established as a result of the evaluation of the active substance hydrogen peroxide as product-types 1-6 (PT1: Human hygiene biocidal products, PT2: Private area and public health area disinfectants and other biocidal products, PT3: Veterinary hygiene biocidal products, PT4: Food and feed area disinfectants, PT5: Drinking water disinfectants, PT6: In-can preservatives), carried out in the context of the work programme for the review of existing active substances provided for in Article 89 of Regulation (EU) No 528/2012, with a view to the possible approval of this substance.

Hydrogen peroxide (CAS no. 7722-84-1) was notified as an existing active substance, by the CEFIC Peroxygens Sector Group, Subgroup Hydrogen peroxide hereafter referred to as the applicant, in product-types 1-6.

Commission Regulation (EC) No 1451/2007 of 4 December 2007[1] lays down the detailed rules for the evaluation of dossiers and for the decision-making process.

In accordance with the provisions of Article 7(1) of that Regulation, Finland 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 hydrogen peroxide as an active substance in Product Types 1-6 was 31 July 2007 in accordance with Annex V of Regulation (EC) No 1451/2007.

On 26 July 2007, Finland competent authorities received a dossier from the applicant. The Rapporteur Member State accepted the dossier as complete for the purpose of the evaluation on 26 October 2007.

On 2 August 2013, the Rapporteur Member State submitted to the Commission and the applicant a copy of the evaluation report, hereafter referred to as the competent authority report.

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 Agency. Revisions agreed upon were presented at the Biocidal Products Committee and its Working Groups meetings and the competent authority report was amended accordingly.

1.2. Purpose of the assessment report

The aim of the assessment report is to support the opinion of the Biocidal Products Committee and a decision on the approval of hydrogen peroxide for product-types 1-6 and, should it be approved, to facilitate the authorisation of individual biocidal products. In the evaluation of applications for product-authorisation, the provisions of Regulation (EU) No 528/2012 shall be applied, in particular the provisions of Chapter IV, 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 from the Agency web-site shall be taken into account.

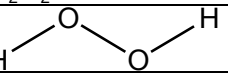
However, where conclusions of this assessment report are based on data protected under the provisions of Regulation (EU) No 528/2012, such conclusions may not be used to the benefit of another applicant, unless access to these data for that purpose has been granted to that applicant.

[1] Commission Regulation (EC) No 1451/2007 of 4 December 2007 on the second phase of the 10-year work programme referred to in Article 16(2) of Directive 98/8/EC of the European Parliament and of the Council concerning the placing of biocidal products on the market. OJ L 325, 11.12.2007, p. 3

2. OVERALL SUMMARY AND CONCLUSIONS

2.1. Presentation of the Active Substance

2.1.1. Identity, Physico-Chemical Properties & Methods of Analysis

CAS-No.	7722-84-1
EINECS-No.	231-765-0
Other No. (CIPAC, ELINCS)	None
IUPAC Name	Hydrogen peroxide
Common name, synonyma	Dihydrogen dioxide, hydrogen dioxide, hydrogen peroxide
Molecular formula	H_2O_2
Structural formula	
Molecular weight (g/mol)	34.01

The active substance as manufactured is an aqueous solution which contains 35-<70% (by weight) of hydrogen peroxide. The upper value (< 70%) is considered to cause no problems from the toxicological or ecotoxicological point of view. On a calculated dry weight basis the minimum purity of hydrogen peroxide is estimated close to 99.5% (by wt). The sum of hydrogen peroxide and water is close to 100 %.
For toxicology and ecotoxicology assessments, concentrations or amounts of hydrogen peroxide always refer to pure (100%) hydrogen peroxide unless stated otherwise. The assessment covers risks from use of biocidal products containing hydrogen peroxide up to 49.9 %.

Pure hydrogen peroxide does not exist commercially. Hydrogen peroxide is always directly produced as an aqueous solution and aqueous solutions of hydrogen peroxide are used as biocidal products. Furthermore, any attempt to produce pure hydrogen peroxide would be prevented by the explosion risks of such a compound.
For this reason the pure active substance hydrogen peroxide is never isolated. Furthermore, the pure active substance would be highly instable (explosion risk) at high concentrations when not dissolved in water. Detailed specifications of the content of hydrogen peroxide in the biocidal products and of impurity and additive profiles are given in Document IIB 6.1.

Impurities: There is no significant impurity (impurity at a concentration > 0.1%) in the substance. The sum of organic and inorganic impurities in aqueous solution is below 0.2 % (by wt), and the total amount of organic impurities in aqueous solutions is approximately 0.1 % (by wt). Calculated for the 35 % solution of hydrogen peroxide, the theoretical dry weight total impurity contents is below 0.5 % (by wt). For the following heavy metals (Pb, Hg, Cd, As) a maximum level of 1 mg/kg in aqueous solution was set in specification, for each of the four metals. For detailed specification of of hydrogen peroxide, see the Confidential A2. It also includes organic impurities, the list of stabilizers, such as phosphoric acid < 0.03%, Sodium phosphate < 0.05%, sodium stannate < 0.085%, ammonium sulfate < 0.03%, and the confidential stabilizers. Stabilizers need to be evaluated at product authorization.

Physico-Chemical properties
In this assessment mainly secondary and historical data sources for physico-chemical properties of H_2O_2 have been used. Most of the original studies referred here have been carried out decades ago, mainly in the 1950's and referred in secondary literature. Therefore detailed information on test methods, test conditions or source or purity of the tested material is not available. The dataset currently available and under the evaluation were not sufficient for the sole purpose of classification and labelling of H_2O_2 for physical chemical properties. The test results data could be compared against the criteria given in the classification and labelling rules according to Directive 67/548/EEC or to Regulation 1272/2008/EC but the scientific validity of the study results could not be evaluated in detail.

Hydrogen peroxide is a high production volume chemical manufactured and used in a wide variety of applications and there is a lot of practical experience on its properties and safe handling. A great deal of referenced relatively old physical-chemical test result data have also been published more recently in trusted peer rewieved handbooks. Taking these considerations into account the current dataset may be regarded sufficient and acceptable for the purpose of biocide risk assessment.

Data on 100% hydrogen peroxide

Hydrogen peroxide and water do not form azeotropic mixture and can be completely separated. Pure, 100% H_2O_2 may be produced on a laboratory scale and published physico-chemical data on it is available and reported also in this document as important additional information. However, pure 100 wt% hydrogen peroxide is of scientific interest only and is not produced on an industrial scale. Even small quantities of impurities may promote decomposition. Even at laboratory scale there are technical difficulties in testing accurately pure hydrogen peroxide since 100% H_2O_2 concentration may not be stable under all conditions and testing may sometimes be impossible. Extrapolations are needed to get estimated values for pure H_2O_2.

Summary of the key physico-chemical properties

Hydrogen peroxide is a clear colourless liquid with a freezing point of - 0.43°C, a boiling point of 150.2 °C and a density of 1.44 g/cm^3. Hydrogen peroxide is miscible with water in all proportions. H_2O_2 is a very weak acid and aquatic solutions are slightly acidic. H_2O_2 is not surface-active. The logarithmic octanol-water partition coefficient is < -1 indicating no potential for bioaccumulation. The low value of Henrys law constant indicates very poor volatilization of H_2O_2 from water to the air.

The vapour pressure of hydrogen peroxide is 214 Pa at 20 °C which is clearly lower compared to the vapour pressure of water. Hydrogen peroxide is a strong oxidizer and may decompose to water and oxygen. Depending on conditions H_2O_2 may also act as a reducing agent. Hydrogen peroxide is not flammable itself but it can cause spontaneous combustion of flammable materials and continued support of the combustion because H_2O_2 liberates oxygen as it decomposes. H_2O_2 decomposition is highly exothermic (98.5 kJ/mole) and therefore decomposition may be self-accelerated. Decomposition rate may be increased significantly in the presence of impurities. Highly concentrated H_2O_2 liquids and vapour can be made to explode. Hydrogen peroxide and highly concentrated aqueous solutions (> 65 wt-%) of H_2O_2 are soluble in a variety of organic solvents such as carboxylic esters (Shumb et al. 1989). Pure hydrogen peroxide should not be mixed with organic solvents (risk of violent reactions). For information on solubility in organic solvents in production, see Doc A2 and the Confidential A2. Once formed, the limited solubility in organic solvents makes hydrogen peroxide extractable in water.

Regulation (EU) No 98/2013 on the marketing and use of Explosive Precursors has to be considered for applications for authorisation for non-professional use. License is needed for private users to buy products containing above 12 % of hydrogen peroxide. It is forbidden to sell products containing hydrogen peroxide above 35 % to private users.

Table 2.1.1-1 Physico-chemical properties of hydrogen peroxide (pure)

Subsection	Method	Purity[a)]	Results	Reference
Melting point	Thermal analysis (freezing temperature)	100%	-0.40 – -0.43°C	Schumb et al., p. 212, 1955, referring to Giguère et al., 1954, Budavari (1989)
Boiling point	Extrapolation of H_2O_2/H_2O vapour pressure composition curves	100%	150.2°C at 101.3 kPa	Schumb et al., 1955
Bulk density/ relative density	Measurements	100%	1.44 g/cm^3 liquid at 25°C 1.71 g/cm^3 solid at −20°C	Schumb et al., 1955

Vapour pressure	Extrapolation from the measured H_2O_2/H_2O vapour pressure curve	100%	214 Pa, at 20°C (293 K) : 299 Pa, at 25°C (298 K) :	Weast et al., p. D-213, 1985 – 1986 Goor et al., p.446, 1989
Solubility in water		100%	miscible in water in all proportions	
Henry´s Law Constant H	Measurement; equilibrium gas-phase	100%	$7.5*10^{-4}$ Pa*m^3/mol at 20°C	Hwang H. and Dasgupta P.K., 1985
Dissociation constant	Measurement	100%	K_a= 2.4 * 10^{-12} at 25°C pKa: 11.62	Weast et al., p.D-163, 1985 – 1986
Surface tension	Capillary rise method	100%	result: 83.3 mN/m at 0°C result: 80.4 mN/m at 20°C	Schumb et al., p. 205 and 206, 1955 referring to Phibbes and Giguère, 1951
Partition coefficient n-octanol/water	Calculation	100%	log Kow = −1.57	Brachhold H., 2006 (not published)
Viscosity	Measured	100%	1.249 mPa*s at 20°C	Schumb et al., p. 202 and 203, 1955
Explosive properties	Tested, (liquid: BAM steel disk method)	variable	explosive limit: ≥ 40 % (wt) as vapour ≥ 86 % (wt) in aqueous liquid (limit may be lower if impurities present)	Goor et al., 1989 Schumb et al, 1955 CEFIC, 1998, Kratz, 1988
Oxidizing properties			see below and see A3	

[a)] in most cases the concentration value 100% represents extrapolated concentration, for technical reasons the actual testing has been carried out in lower than 100% concentration.

Stabilizers: Commercial hydrogen peroxide grades are stabilized to prevent or slow down decomposition of H_2O_2. The stabilisers are of several types: mineral acids to keep the solution acidic (stability is at a maximum at pH 3.5-4.5), complexing/chelating agents to inhibit metal-catalysed decomposition or colloidal to neutralise small amounts of colloidal catalysts or adsorb/absorb impurities. For stabilisers, see also the EU Risk Assessment Report (2003).

Methods of Analysis

Analysis of active substance as manufactured. Hydrogen peroxide is always directly produced as an aqueous solution and aqueous solutions of hydrogen peroxide are used as biocidal products. The pure active substance hydrogen peroxide is never isolated. Consequently, no analytical method for technical-grade active substance was submitted. For analysis of hydrogen peroxide in aqueous solutions used as biocidal products, see formulation analysis.

Formulation analysis. The formulations of hydrogen peroxide are aqueous solutions of a concentration of 49.9% (w/w) and 35% (w/w). The hydrogen peroxide content in those aqueous solutions is determined by titration [redacted]

Residue analysis. A large body of publicly available literature exists regarding analytical methods for hydrogen peroxide in water. In addition to the published methods, ready-to-use testing systems are commercially available, e.g. Dräger© tubes for airborne hydrogen peroxide. All of these published or commercially available methods have been in use for many years, have undergone scientific review and also inter-laboratory validation studies were carried out

(Gunz et al., 1990). The choice of a particular method should be guided by the matrix to be analysed, the expected concentration range and potential interferences, which are discussed in the review literature (e.g. Gunz et al., 1990; Sturzenegger, 1998). Nevertheless, by way of example, a method was validated for analysis of airborne hydrogen peroxide after absorption into water. It may also be used for determination directly in water samples. Hydrogen peroxide is determined by UV-/VIS spectrometry with cobalt-bicarbonate as an indicator. The limit of quantification (LOQ) is 740 µg/L, in the aqueous solution. The LOQ in air depends on the sampling parameters. The method may also be automated by flow-injection analysis. Due to deficiencies in validation, new validated methods for air and water must be submitted before product authorisation. Alternatively, the existing methods must be validated according to available guidance on information requirements.
In **soil**, hydrogen peroxide is decomposed very rapidly, and therefore the validation of an analytical method is technically not feasible. Residual hydrogen peroxide in soil water may be analysed by the methods discussed for water samples.
An analytical method for hydrogen peroxide in **body fluids and tissues** of humans or animals is not required, since the substance is not classified as toxic or highly toxic.
In conclusion, suitable methods for the analysis of hydrogen peroxide in environmental media are available, as evaluated by needs for information at the time of submission of the dossier.

Disinfection by-products: Hydrogen peroxide is reactive and it degrades rapidly in contact with organic material. A significant proportion of hydrogen peroxide decomposes to water and oxygen. The antimicrobial action of hydrogen peroxide stems from its ability to form powerful oxidants such as the hydroxyl radical and singlet oxygen. These reactive oxygen species cause irreversible damage to cellular components such as enzymes, membrane constituents and DNA. The range of by-products is considered wide and not well characterized at detailed level. It would be very difficult to provide analytical methodology to cover the low level concentrations of the enormous variety of molecular structures including breakdown products. At a level of practical concentrations, no disinfection by-products (DBPs) with (eco)toxicological relevance have been identified. No methods for DBPs is required.

2.1.2. Intended Uses and Efficacy

For mode of action, see the previous subchapter. The assessment of the biocidal activity of the active substance demonstrates that it has a sufficient level of efficacy against the target organisms, i.e. bacteria, fungi, and viruses, and the evaluation of the summary data provided in support of the efficacy of the accompanying product, establishes that the products may be expected to be efficacious. In the risk assessment, concentrations used were with sufficient efficacy on at least one type of organism in each product type. Information on tests, test organisms, and effective concentrations relevant for each product type are in a tabled form in Doc IIB, Sec 7. For PT1 there is data on sufficient efficacy against at least one species (fungi) at lowest in-use concentration (4.9%) evaluated. In addition, risks from exposure to 7.4% have been assessed in PT 1. For products intended to be marketed for PT3 formulations, further evidence of virucidal activity is needed at product authorization. Virucidal activity has been demonstrated against Polio and Adenoviruses, but not against viruses of veterinary importance.

Uses evaluated include disinfection of human skin (PT 1) for professionals and non-professionals (PT1), surface disinfection by VHP process in private or public hygiene disinfection of rooms using the vaporised hydrogen peroxide (VHP) process (PT2), disinfection of animal housing by spraying (PT3), aseptic packaging: hydrogen peroxide is used to disinfect packaging for foodproducts by immersion (PT 4), surface disinfection by VHP process in food processing facilities (PT4), disinfection of distribution systems for drinking water (PT4), disinfection of drinking water for humans and animals (PT5), preservation of paper additives (PT6). In addition, in order to facilitate the work of Member States in granting or reviewing authorisations, the intended uses of the substance including their in use concentrations, as identified during the evaluation process, are listed in Appendix II.

Resistance: The lethal effects of oxidative molecular species generated from hydrogen peroxide can be avoided with any damage being repaired in microorganisms such as *Escherichia coli* and *Salmonella typhimurium. When E.coli and S.typhimurium* are exposed to low concentrations of H2O2, 3 µM and 60 µM respectively, cells produce enzymes and other proteins which are important for cellular defence and mitigate the toxic effects of the oxidative species. This adaptive response is triggered by nontoxic levels of the oxidative species to protect against and produce resistance to oxidative stress caused when challenged with higher concentrations, 10 mM (Dukan and Touati (1996), Christman et al (1985)). The resistance to oxidative stress that *E.coli* develops when exposed to H202, as reported in literature papers, demonstrates an adaptive response only. Hydrogen peroxide has been intensively used as a disinfectant and preservative for more than 3 decades and has not lead to the development of significant resistance levels among field populations. Genetically inherited resistance is not expected when the products are used as recommended. For resistance, see Doc IIA, Section 2.3.

2.1.3. Classification and Labelling

Hydrogen peroxide is included in Annex VI of the CLP Regulation (Index number 008-003-00-9). The classification, as presented in Annex VI Table 3.1, is the translation of the harmonised classification made for the substance under Directive 67/548/EEC.

In accordance with Regulation (EC) No 1272/2008, Annex VI Table 3.1, hydrogen peroxide is classified and labelled as follows:

Hazard Class and Category Codes	Ox. Liq. 1 H271 Acute Tox. 4 * H332 Acute Tox. 4 * H302 Skin Corr. 1A H314			
Hazard Statement Codes	H271 May cause fire or explosion; strong oxidiser. H332 Harmful if inhaled. H302 Harmful if swallowed. H314 Causes severe skin burns and eye damage.			
Supplemental Hazard Statement Codes	-			
Pictograms and Codes	GHS03 	GHS05 	GHS07 	
Signal Word (Code)	Danger (Dgr)			
Specific Concentration Limits M Factors	Ox. Liq.1; H271: C ≥ 70 %**** Ox. Liq. 2; H272: 50 % ≤ C < 70 %**** Skin Corr. 1A; H314: C ≥ 70 % Skin Corr. 1B; H314: 50 % ≤ C <70 % Skin Irrit. 2; H315: 35 % ≤ C <50 % Eye Dam. 1; H318: 8 % ≤ C <50 % Eye Irrit. 2; H319: 5 % ≤ C < 8 % STOT SE 3; H335: C ≥ 35 % *			
Notes	B			

The classification, as presented in the Table, is the translation of the harmonised classification made for the substance under Directive 67/548/EEC.

The RMS is of the opinion that based on the data evaluated in the present assessment report, Aquatic Chronic 3 (H412) classification should be applied according to the 2 ATP to CLP Regulation (Regulation (EC) No 286/2011). Regarding the acute toxicity classification the acute oral toxicity can be confirmed as category 4 based on the data presented in the present assessment report. For the acute inhalation toxicity category 4 some uncertainty remains, and therefore the minimum classification as category 4 cannot be confirmed.

2.2. Summary of the Risk Assessment

2.2.1. Human Health Risk Assessment

2.2.1.1. Hazard identification

Absorption, distribution, metabolism and excretion

Hydrogen peroxide is reactive and it degrades rapidly in contact with organic material. The rapid degradation upon contact with skin explains the absence of systemic effects from exposure to hydrogen peroxide. However, application of hydrogen peroxide solutions to damaged skin, or exceptional cases with excessive amounts of exogenous hydrogen peroxide on skin, may result in some systemic dose. Hydrogen peroxide is presumed to degrade rapidly into oxygen and water in contact with blood or other body fluids. Despite the fact that hydrogen peroxide is a normal metabolite in the cell metabolism and the knowledge of the hydrogen peroxide metabolism e.g. through catalase and glutathione peroxidase enzymes, data on the effects of exogenous hydrogen peroxide exposure in humans or animals is limited and mainly consists of case reports of oxygen embolisation following the degradation of hydrogen peroxide after exposure to high amounts of the substance. No standard dermal penetration studies with hydrogen peroxide have been successfully conducted. Based on the physico-chemical properties of hydrogen peroxide, 100% dermal penetration should be used in the absence of more accurate information. However, in the absence of clear systemic effects, no dermal penetration parameter was needed in order to conclude on human health risks from the presented uses of hydrogen peroxide. In rat blood diluted 1000 times, the half-life of hydrogen peroxide was less than 5 minutes for the low and intermediate concentrations of hydrogen peroxide 5 and 10 mg/L. For the high concentration (20 mg/mL) the half-life was more than 4 hrs. In the study, concentrations of hydrogen peroxide were far away from the range of a.s. in products or in-use concentrations. Even then the study demonstrates the high efficacy of the antioxidative system in blood. Furthermore, it supports the view that hydrogen peroxide if entering blood circulation is rapidly decomposed in blood and will not be systemically available. For this reason the distribution of hydrogen peroxide in the body is expected to be very limited after exposure to hydrogen peroxide solutions. In conclusion, it was acceptable to waive the dermal penetration study.

Acute toxicity

Acute toxicity of hydrogen peroxide depends on the concentration. The results of acute oral toxicity studies performed in rats with formulations containing hydrogen peroxide at concentrations from 35 % to 70 % demonstrated acute oral LD50 values in the range of 694-1270 mg/kg bw indicating that hydrogen peroxide, at the tested concentrations, is harmful by the oral route. When corrected to 100% hydrogen peroxide, the LD50 values were around 500 mg/kg bw. The acute dermal LD50 of formulations containing 70 % hydrogen peroxide was between at 6500 and 13000 mg/kg bw in the rabbit indicating that hydrogen peroxide, at the tested concentrations, is not acutely toxic by the dermal route. The inhalation LC50 value for the test substance containing 49.3% hydrogen peroxide was > 0.17 mg/l/4 h (highest attainable vapour concentration).

Irritation, corrosivity and sensitisation

Hydrogen peroxide causes burns. The irritating property of hydrogen peroxide to the skin and the eye varies dramatically with its concentration. No dermal irritation was noted after

application of 10 % hydrogen peroxide. The 35 % hydrogen peroxide caused slight to moderate reversible erythema and oedema in a skin irritation study. However, irreversible desquamation of skin triggers classification as Xi; R38 "Irritating to skin". 49.2 % hydrogen peroxide was severely irritating to skin in a study where ulceration and necrosis of skin were noticed in the histopathology. In addition, 50 % ≤ hydrogen peroxide < 70 % is classified with C; R34 "Causes burns". Under CLP, the 35 % hydrogen peroxide, causing irreversible desquamation skin, triggers classification of Skin irritation 2, H315: "Causes skin irritation", and 50 % ≤ hydrogen peroxide < 70 % is currently classified with Skin corrosion 1B, H314: "Causes severe skin burns and eye damage".

Hydrogen peroxide causes concentration dependent eye lesions. At higher concentrations, severe and irreversible damage to the rabbit eye has been demonstrated. Dilute formulations exert only mild and completely reversible irritating effects. The 5 % hydrogen peroxide caused only mild eye irritation with effects that do not meet the classification criteria.

Both animal data and human experience indicate that hydrogen peroxide causes respiratory irritation. In a mice study an RD_{50} value of approx. 160 mg/m^3 (113 ppm) and an extrapolated (by the RMS) value RD_{10} value of 17.5 mg/m^3 (12 ppm) have been derived. According to the REACH guidance (Chapter R.8, APPENDIX R. 8-9), the RD_{10} in mice is proposed as a starting point to derive a threshold for a biologically significant sensory irritation in humans, and a candidate AEC (ca. 1.5 ppm; 2.2 mg/m^3) can be derived from RD_{10}. Such value is potentially useful in future refinements of the risk assessment, but was not used as the reference value in the risk assessment in the CAR.

A modified Magnusson-Kligman test from 1953 with a negative result from the EU Risk Assessment Report was cited by the Applicant. However, this test does not meet the current requirements for proper testing. The available human clinical data reveals two reported cases of positive patch tests with hydrogen peroxide. Taking into account the widespread occupational and consumer use over many decades, sensitization to hydrogen peroxide seems to be rare. Hence, it is unlikely that a new skin sensitization test would give results warranting classification either. Hydrogen peroxide is not considered to be a potential skin sensitiser.

Repeated dose toxicity

Oral toxicity. Repeated dose toxicity of hydrogen peroxide has been studied via oral and inhalation route. In an oral toxicity study, 35% hydrogen peroxide was applied to a sensitive, catalase-deficient strain of mice via drinking water for 90 days. The local effects included minimal to mild mucosal hyperplasia of the duodenum. **The no observed adverse effect level (NOAEL) was 100 ppm (males: 26 mg/kg bw/day; females: 37 mg/kg bw/day).** The NOAEL of 100 ppm of hydrogen peroxide in water corresponds to a concentration of 100 mg/litre. Use of the units of concentration of hydrogen peroxide in drinking water is regarded appropriate for a substance with predominantly local effects.

Inhalation toxicity. In a 28-day inhalation study in the rat clinical signs of systemic toxicity (salivation, urinary incontinence and piloerection) and respiratory irritation were observed at ≥ 25 ppm. At 60/30 ppm irregular breathing, chromodacryorrhoea, hunched posture, increased response to touch and thin appearance were also observed in some animals. Treatment related local effects due to irritation of the respiratory tract with necrosis and inflammation in the nose appeared at ≥10 ppm (14.6 mg/m^3). Although rhinitis, epithelial erosion in larynx and increased incidence of perivascular neutrofil infiltration in lung occurred in one or in a few animals only (mostly at 25 ppm), these findings may also be treatment related. Signs of inflammation (minimal decreases in plasma total protein and albumin), decreased glucose in females, minimal decreases in red blood cell parameters in males and females and slightly decreased body weight and food consumption in males were observed at 25 ppm. Exposure at levels of 60/30 ppm was overtly toxic and animals of this group were sacrificed at day 13 for animal welfare reason. The no observed adverse effect level (NOAEC) was 2 ppm (2.9 mg/m^3). The experiment via inhalation route does not provide additional information with regard to the toxicity profile of hydrogen peroxide following repeated exposure. The Applicant submitted in 2014 a 90-day study which was considered necessary to cover the long term exposures. In this study the NOAEC of 7 ppm (ca 10 mg/m^3) was the highest concentration tested in rats in

nose only exposure. By use of the overall assessment factor of 8, the **$AEC_{inhalation}$ of 1.25 mg/m^3** was derived. The slight effect on body weight development in males only was not considered as adverse, as the body weights at 90 days was regarded as being within historical controls and not associated with clear biologically significant effects such as observed in the 28-day study. The AEC in the 90-day study was agreed to cover all durations of exposure. No clear systemic effects were observed and the NOAEC was less critical than the NOAEC in the 28-day study. Spacing of doses and the design of exposure were not identical in these two studies; 28-day and 90-day.

As the local effects are the primary effects of hydrogen peroxide exposure and these effects are considered to be caused by the direct chemical reactivity of the substance and the effects are regarded to remain constant from species to species, there is no need for a study with the second species.

The Applicant has submitted no dermal repeated dose study with specific justification (please refer to Doc III, section A6.4.2) and further argumentation was provided by the Applicant during the evaluation of the dossier. Based on the available data, it can be assumed that dermal exposure to hydrogen peroxide mainly causes local effects in the skin, systemic effects being of much less significance. However, it is not possible to estimate the severity of local effects caused by repeated dermal exposure. A guideline 90-day study may not be appropriate for a substance like hydrogen peroxide. Furthermore, as the effects on the skin may vary from formulation to formulation, information on effects on skin may be needed at product authorisation. Initiating a human volunteer study was not considered acceptable by the MSs. The RMS has been informed on few earlier reports on uses of skin disinfection products containing hydrogen peroxide. Unfortunately, the reporting of them is not of good quality, and the concentrations are lower than in those in the use described for skin treatment in DOC II B. The WG V 2014 agreed that a study on repeated dermal exposure is not required at product authorisation stage. A qualitative risk assessment should be performed for the local dermal effects taking into account the classification of the product.

There are no data on chronic toxicity available. Systemic effects of hydrogen peroxide in a subchronic oral study were rather weak and limited, and the effects could not be confirmed as primary effects. However, based on the available data it is not possible to estimate the effects of long-term exposure at lower dose levels, considering both the systemic and local effects.

Reproductive toxicity

The Applicant submitted no reproductive toxicity study**.** In the published literature, no studies employing appropriate protocols were available. Hydrogen peroxide is readily passing through biological membranes and may reach blood circulation. The applicant has submitted a study on rapid degradation of hydrogen peroxide in diluted rat blood. Due to the rapid degradation of hydrogen peroxide, it is doubtful whether hydrogen peroxide could reach inner organs such as ovaries and testes as well as foetuses to cause reproductive toxicity. A toxicologically meaningful systemic availability of hydrogen peroxide and transportation of the substance via blood circulation therefore is unlikely. This view is supported by the available repeated dose toxicity studies, which did not result in primary systemic effects. It can be concluded that a data gap with regard to studies of reproductive and developmental toxicity does not exist.

Genotoxicity

Hydrogen peroxide was mutagenic *in vitro* in a bacterial gene mutation test both in the presence and absence of metabolic activation, in a mammalian gene mutation test without metabolic activation and in a chromosomal aberration test in the presence and absence of metabolic activation.

The positive results of the *in vitro* genotoxicity tests were not confirmed *in vivo* in a bone marrow micronucleus test and a UDS test in liver cells. However, there is no evidence that hydrogen peroxide has reached the target organ in the *in vivo* UDS test. Likewise, in micronucleus test with exposure via drinking water hydrogen peroxide has most probably not reached the bone marrow. Due to the uncertainty with regard to availability of the test

substance in the target organ, the result of this study is equivocal. In a micronucleus test with i.p. application, no increase in the frequency of micronucleated polychromatic erythrocytes was observed. Effects in the bone marrow (decreased ratio of polychromatic erythrocytes to normochromatic erythrocytes) were noticed indicating the suitability of test conditions.

The results of the genotoxicity studies do not meet the current classification criteria for mutagenicity.

Local genotoxicity: (Doc IIA, Section 3.6.) Hydrogen peroxide is capable of undergoing or initiating numerous reactions, including molecular additions, substitutions, oxidations and reductions. It is a strong oxidant and can form free radicals by homolytic cleavage. Hydroxyl radicals can damage vital cellular components.
The effects found in the *in vitro* and *in vivo* -genotoxicity tests are most probably due to the oxidising properties of hydrogen peroxide. The oxidative damage to DNA may lead to different toxic outcomes if exposure becomes chronic, but the local genotoxicity of hydrogen peroxide cannot be characterised. This leads to the uncertainty of local genotoxicity of hydrogen peroxide.
Hydrogen peroxide can be used as a positive control in Comet tests but relevance of assays like Comet to humans remains unclear.

According to one of the reports listed in the Appendix III to DocIIA, a study was conducted to explore target tissue *in vivo* genotoxicity and mutagenicity as a pre-screen for carcinogenicity. Hydrogen peroxide 0.2-3.2% solutions were applied to the skin of mice twice weekly for 4 weeks. There was no indication of induced DNA damage (increased 8-OHdG), c-Ha-ras mutations, epidermal hyperplasia and dermal cellularity changes in this tissue model.
In conclusion, the available studies are not in support of a significant genotoxicity/ mutagenicity for hydrogen peroxide under *in vivo* conditions. A wider database of genotoxicity and mutagenicity observations on other relevant target tissues in direct contact with hydrogen peroxide would be desirable.

Due to the irritating properties of hydrogen peroxide, the risk mitigation measures include the use of personal protective equipment. The RMS considers the protections sufficient, and does not consider the local genotoxicity as a relevant endpoint for a risk assessment.

Chronic toxicity/ carcinogenicity

The Applicant submitted no guideline-conform chronic toxicity/carcinogenicity studies. All carcinogenicity studies and publications described in the EU Risk Assessment Report (2003) are summarised in the dossier under Doc IIA, Appendix 4 as copied from the EU Risk Assessment Report. In summary, some tumorigenic activity is observed, the incidence of tumours being clearly dependent on the efficiency of detoxification ability of the tested strains. In the light of the data, genotoxic mechanisms cannot be ruled out in the carcinogenicity of hydrogen peroxide. However, the endogenous defence mechanisms against reactive oxygen species may suggest a threshold for carcinogenicity of hydrogen peroxide. No NOAEL for carcinogenicity could be determined in the referred studies. The concentrations of hydrogen peroxide in drinking water in the carcinogenicity studies were about one tenth of the concentration of hydrogen peroxide with irritation property on eyes. The effects are proposed to be secondary to local irritation.

In the context of the existing risk assessment (EU RAR 2003) it was concluded that…."should not be classified as a carcinogen since carcinogenic effects can be seen secondary to local effects at high concentrations". This is also the current interpretation of classification criteria under CLP, see Guidance on the Application of the CLP, Section 3.6.2.3.1. Criteria, (2009).

Overall, the available repeated dose studies support the conclusion that the toxicity of hydrogen peroxide is mediated mainly by local irritation at the site of first contact although the possibility of systemic effects cannot be completely ruled out.

Neurotoxicity

Hydrogen peroxide does not belong to a class of compounds for which a neurotoxic potential can be expected. In addition, the available toxicity studies gave no indication of a relevant neurotoxic potential of the compound. There is no need to conduct specific neurotoxicity tests.

Human data

The primary human health hazard associated with exposure to hydrogen peroxide is irritation (and corrosion) of the skin, eyes and respiratory tract (i.e. sites of first contact). Local effects may arise both after short-term and repeated / long-term exposure.

Symptoms of respiratory irritation have been reported among manufacturing plant personnel exposed to hydrogen peroxide. A single case of long-term inhalation exposure to hydrogen peroxide with progressive dyspnoea and bilateral diffuse nodular infiltrates of the lungs was also seen. The patient improved progressively without treatment after withdrawal from the occupational exposure (Kaelin et al., 1988). A survey of the health surveillance data on the production workers encompassed 110 workers of whom 80 had been involved in the production for more than 10 years. Collection of exposure data over the 1990's was targeted on loading and filling operations, packing drums, containers, trucks, railway cars with semi-automatic equipment, addition of stabilisers, and preparation of hydrogen peroxide solutions of various concentrations. The mean levels of hydrogen peroxide over the shift had been below the OEL of 1.4 mg/ m^3 whereas short-term concentrations were up to about 5 mg/m^3, and about 10 mg/ m^3 in an accidental situation. The health examination data included some measurements of lung function, a symptom inquiry and other observations. No remarkable findings were reported in the lung function (Degussa-Hüls, 1999). The Finnish Institute of Occupational Health coordinated a worker health surveillance study in one company which concerned a small group of workers (6 persons) exposed to hydrogen peroxide vapours in aseptic packaging (Riihimäki et al., 2002). All the workers were engaged with hydrogen peroxide for 3 years or less. Complaints among operators/ maintenance workers appeared concerning irritation in the eyes and airways, headaches, temporary loss of olfaction, symptoms and signs in the skin, and blanching of hair. Peak exposures of up to 11 mg/m^3 (8-hour TWA 2-3 mg/m^3) of hydrogen peroxide in air were measured in the breathing zone of the individuals. From the studies of Riihimäki et al. (2002 and 2004) a level of no symptoms 0.5 to 0.7 mg/m^3 (0.36–0.5 ml/m^3; 8-hour mean values, no higher peak exposures) could be determined. The NOAEC of 0.7 mg/m^3 was used in deriving the national German MAK value (2005) of 0.7 mg/m^3 (0.5 ml/m^3).

There are a number of reported incidents of human poisoning by oral ingestion of aqueous hydrogen peroxide solutions. A case was reported in which a 54-year old male suffered transient shock following wound irrigation with a 3% aqueous hydrogen peroxide solution (Bassan et al., 1982). Evidence of distinct, but transient neurologic and cardiac dysfunctions was available. It was hypothesised by the authors reporting the case that widespread systemic embolisation of oxygen micro-bubbles occurred. This may have been the result of significant local absorption of hydrogen peroxide and/or oxygen formed in the treated wound and in contact with blood. These effects were due to rapid decomposition of hydrogen peroxide after entering human blood circulation.

Several reports showing skin and eye irritation effects are available. The severity of effects is closely connected to the applied hydrogen peroxide concentration.

A retrospective review of all exposures reported to the Utah Poison Control Centre over a 36-month period found that 325 cases (0.34%) were due to hydrogen peroxide. Ingestion was the most common route of exposure accounting for 83% of all exposures. The next most common routes of exposure were ocular and dermal accounting for 8.0% and 7.7% of cases, respectively (Dickson & Caravati, 1994).

Several reports on patch testing of hairdressers and other patients with negative result are available. Only two cases on positive patch tests to hydrogen peroxide are published (Aguirre et al., 1994). In spite of these two positive patch tests, and on recognition of the widespread

occupational and consumer use of hydrogen peroxide over many decades, it may be confidently stated that the potential of hydrogen peroxide to cause skin sensitization is extremely low. The substance does not meet the criteria for classification.

In a human volunteer study (Ernstgård *et al*., 2012) subtle acute respiratory effects were found after 2 hours of inhalation exposure to 2.2 ppm levels. The non-effect level was 0.5 ppm. The large inter-individual variation of the endpoints measured and the statistical significance of the findings in this study are questionable and therefore the study has not been used for setting the AECinhalation.

2.2.1.2. Effects assessment

The adverse effects of hydrogen peroxide in humans are limited to local effects at the site of first contact with the body and to embolism in some cases. No clear systemic effects were observed which is plausible in the light of the mode of action, i.e. direct chemical reactivity leading to rapid degradation. Corrosion and/or irritation of the skin and mucous membranes are the most prominent observations in the variety of animal studies. These effects are concentration dependent with no or only minor dependence from exposure duration.
Besides the direct chemical reactivity underlying the irritation and corrosion related lesions, hydrogen peroxide causes sensory irritation. This phenomenon is also concentration dependent and the symptoms manifest soon after start of exposure. There is no human data available specifically on sensory irritation. In a mouse test of sensory irritation, an RD_{50} value of 113 ppm has been determined. The data allows extrapolation of an RD_{10} of approximately 12 ppm. In order to extrapolate the animal data to humans an assessment factor of 2.5 and an intraspecies assessment factor of 3.2 for the remaining uncertainty is considered sufficient to derive an AEC value (ca. 1.5 ppm). This value was not used for risk characterization, but could be used in later refinements.

Indicative dermal reference value
No dermal irritation was noted after application of 10% hydrogen peroxide. The 35 % hydrogen peroxide caused slight to moderate reversible erythema and edema in a skin irritation study. However, irreversible desquamation of skin triggers classification of Skin irritation 2, H315: "Causes skin irritation". (Xi; R38 "Irritating to skin"). In view of the absence of systemic effects after exposure to hydrogen peroxide, only external exposure limits are relevant to account for the potential local effects of hydrogen peroxide. Since in the intended use(s) the in-use concentration of hydrogen peroxide is below a skin irritating threshold (concentration limit for classification as skin irritating is 35%), only the inhalation route of exposure has been identified to be relevant in the quantitative exposure and risk assessment. In the absence of more accurate data, potential exposure in the different use scenarios should be compared to the thresholds set for classification. In mixing and loading exposure to undiluted products may occur.

Serious eye damage/eye irritation: Hydrogen peroxide causes concentration dependent eye lesions. At higher concentrations, severe and irreversible damage to the rabbit eye has been demonstrated. The results support the current classification with Eye irritation 2, H319: "Causes serious eye irritation" for 5 % ≤ hydrogen peroxide < 8 % (Xi; R36 "Irritating to eyes), and for for 8% ≤ hydrogen peroxide < 50% classification with Eye damage 1, H318 (Xi; R41 "Risk of serious damage to eyes").

Inhalation reference values
The following **AEC for inhalation exposure** is proposed for hydrogen peroxide: For acute, medium-term and long-term exposure: 1.25. mg/m^3 based on the NOAEC in 90-day inhalation rat study with the overall assessment factor of 8. This value is reasonably well in line with human data, where a level of no symptoms at 0.5 to 0.7 mg/m^3 (0.36–0.5 ml/m^3; 8-hour mean values, no higher peak exposures) could be determined.

2.2.1.3. Exposure assessment

Description of uses

Hydrogen peroxide is used in different product types mainly for professional use. Only for disinfection of human skin (PT 1) hydrogen peroxide is provided for professionals and non-professionals. For exposure assessment dermal and inhalation exposure is addressed. Oral exposure is very unlikely for PT 1, 2, 3 and 6 and could only appear using hydrogen peroxide for aseptic packaging and disinfection of distribution systems for drinking water (PT 4) and for drinking water disinfection (PT 5). Based on the assessment, oral route is relevant only in secondary exposure in PT 5.

PT 1: *Human skin disinfection* with 7.4/4.9% (w/w) hydrogen peroxide by private and professional users.

PT 2.01: *Surface disinfection by VHP process* in private or public hygiene*.* Disinfection of rooms using the vaporised (250 to 400 ppm in air) hydrogen peroxide (VHP) process. Vaporised hydrogen peroxide is decomposed by the VHP machine after disinfection.

PT 3: *Disinfection of animal housing* by spraying aqueous solutions of 7.4% (w/w) of hydrogen peroxide.

PT 4a: *Aseptic packaging:* Hydrogen peroxide is used to disinfect packaging for food products by immersion into 35% (w/w) aqueous solutions.

PT 4b: *Surface disinfection by VHP process* in food processing facilities. The VHP process used in food processing areas is the same as the process assessed under PT 2.01.

PT 4c: *Disinfection of distribution systems for drinking water:* The likely use concentration is 4% (w/w)

PT 5: *Disinfection of drinking water* for humans and animals.

PT 6.02: *Preservation of paper additives* with up to 1.0% (w/w) hydrogen peroxide.

For dermal and inhalation exposure values in different scenarios, please refer to the Table below, and the Appendix IV, p.85.

In the absence of clear systemic adverse effects the risk characterisation of hydrogen peroxide is focused on local effects and no systemic doses are estimated. For the inhalation route the airborne exposure concentration is compared with the AEC for inhalation (1.25 mg/m^3). For dermal exposure a comparison with the skin irritation limit (35%) in the current classification has been considered to account for the potential local effects of hydrogen peroxide.

Biocidal use patterns and the typical in-use concentrations are described in the table (below) under the supported product types. Information on the use conditions of hydrogen peroxide was collected from a questionnaire by the CEFIC Peroxygens Sector Group, Brussels.

Table 2.2.1.3-1 Summary of professional exposure

Intended use (PT)	Exposure scenario (H_2O_2 concentration)	Method for exposure assessment	Inhalation exposure, concentration in air (mg/m^3)	Dermal exposure (external)	PPE

Intended use (PT)	Exposure scenario (H_2O_2 concentration)	Method for exposure assessment	Inhalation exposure, concentration in air (mg/m^3)	Dermal exposure (external)	PPE
PT 1 Disinfection of human skin	mixing and loading (professionals, once a month) (35%/**49.9%)**	a) TNsG m&l model 7 for inhalation; m&l model 4 for dermal b) ART	a) 0.47 b) 0.27	a) 29.8 mg/day (with PPE, 10% penetration)	gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	skin application (regular use 25x1min day) (a) **7.4%** b) **4.9%**)	ConsExpo (constant rate release)	a) 1.04 (as 8-h TWA) 1.83 (event) b) 0.69 (as 8-h TWA) 1.21 (event)	a) 222 mg/application, 0.27 mg/cm^2/ application b) 147 mg/application, 0.18 mg/cm^2/ application	-
PT 2.01 Surface disinfection by VHP process	evaporation (max 3x/day for 200days/y) (**35%** (350-560 mg/m^3 in the sealed space)	- (no exposure, sealed space)	1.4 mg/m^3 is used as a worst case estimate.	none	RPE in re-entry if the concentration over AEC
PT 3 Disinfection of animal housing	mixing and loading (1 or 0.5 h; professional operators: max 200days/y; trained farmers: max 10 operations/y) (35%/**49.9%**)	a) US-EPA PHED surrogate exposure guide for inhalation; TNsG Loading DEGBE model in BEAT for dermal b) ART	a) 0.26-0.5 b) 3.9	a) 13868 mg/day (with PPE, 10% penetration)	gloves, coverall, goggles/face shield, RPE
	spraying (professional operators: max 200days/y, 120–400 min/operation; trained farmers 6-10 operations/y, 400min/operation) (**7.4%**)	a) TNsG spraying model 2 b) ConsExpo (exposure to spray) c) ART	a) 5.6 b) 0.4 c) 4.0	a) 559 mg/day (with gloves and 5% clothing penetration) b) 798 (with PPE, 5% penetration)	gloves, coverall, goggles/face shield, RPE
PT 4 Aseptic packaging	loading (**35%**)	-	incidental	incidental	gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV

Intended use (PT)	Exposure scenario (H_2O_2 concentration)	Method for exposure assessment	Inhalation exposure, concentration in air (mg/m^3)	Dermal exposure (external)	PPE
	application (around machines)	measured	0.14-0.7	no data	-
	maintenance work	measured	0.7-1.4	incidental	gloves, coverall, goggles/face shield, RPE
PT 4 Surface disinfection by VHP process (same as in PT 2)	evaporation (max 3x/day for 200days/y) (**35%** (350-560 mg/m^3 in the sealed space)	- (no exposure, sealed space)	1.4 mg/m^3 is used as a worst case estimate	none	RPE in re-entry if the concentration over AEC
PT 4 Disinfection of distribution systems for drinking water	mixing and loading (20 min on 1 to 2 days/wk) (35%/**49.9%**)	a) TNsG m&l model 7 b) ART	a) 0.47 b) 0.43	incidental	gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	application (0.5-2h/day, max 200days/year) (**4%**)	a) TNsG spraying model 2 b) ART	a) 3.04 b) 7.1	90.7 mg/day (with gloves and 5% clothing penetration)	gloves, coverall, goggles/face shield, RPE
PT 5 Disinfection of water	loading (20 minutes, 10-100 days/y) (35%/**49.9%**)	a) TNsG m&l model 7 b) ART	a) 0.47 b) 0.19	incidental	gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	application (25 mg/l)	automated process	none/very low	-	-
PT 6.02 In-can preservatives of paper additives	mixing and loading (35%/**49.9%**)	ART	0.23	incidental	gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	application (evaporation, exposure times short in automated continuous processes) (1%)	measured	max. 0.7	none	gloves, coverall, goggles/face shield, RPE in maintenance work

Non-professional exposure:
The application is comparable to the scenario for professionals as a worst case. Exposure estimates for non-professionals do not include mixing and loading, since non-professionals purchase a ready to use 7.4/4.9 % solution.

Table 2.2.1.3-2 Summary of non-professional exposure

Intended use (PT)	Exposure scenario	Method for exposure assessment	Inhalation exposure, concentration in air	Dermal exposure (dermal load)
PT 1 Disinfection of human skin	skin application (25/day as a worst case) (a) 7.4%, b) 4.9%)	ConsExpo (constant rate release)	a) 1.04 (as 8-h TWA) 1.83 (event) *b) 0.69 (as 8-h TWA)* *1.21 (event)*	a) 222 mg/application, 0.27 mg/cm^2/ application b) 147 mg/application, 0.18 mg/cm^2/ application

Indirect exposure:

Secondary oral and dermal exposure of consumers to residual hydrogen peroxide in food and drinking water is in theory possible under PT 4 (aseptic packaging, disinfection of distribution systems for drinking water) and PT 5 (disinfection of drinking water). However, hydrogen peroxide used for aseptic packaging evaporates while the wrapping material is heated before filled with food and no residues in food are expected. Furthermore, hydrogen peroxide, if present, would rapidly decompose in contact with any type of food.

Secondary oral exposure of consumers to hydrogen peroxide is possible via disinfection of distribution systems for drinking water as well as via disinfected drinking water. Pipes and containers disinfected with hydrogen peroxide are flushed before refilled with drinking water and relevant residual hydrogen peroxide is regarded as negligible under disinfection of distribution systems for drinking water (PT 4).

In PT 5 exposure of humans is possible via oral route. In consideration of the Drinking Water Directive (Directive 98/83/EC), the limit value of 0.1 µg/l for pesticides and their relevant metabolites is provided for drinking water. In the intended uses of hydrogen peroxide as a disinfectant of drinking water, below 0.1 mg/L at the consumer tap, this limit value is exceeded. However, this limit value is not considered applicable for hydrogen peroxide used intentionally for disinfection of water. In the absence of a chronic oral study, it is not possible to set an ADI (Acceptable Daily Intake) which is needed as a reference value for a risk assessment of consumers possibly exposed for a life-time to hydrogen peroxide residues in drinking water. An ADI may not be necessary or practical as a reference value in a risk characterization in this type of case.

Dermal exposure: Secondary dermal exposure of humans via shower and washing is considered possible, yet negligible.

Indirect exposure - inhalation: Exposure of by-standers to unacceptable concentrations of hydrogen peroxide in air following disinfection is possible but can be prevented if appropriate waiting periods are applied in PT 3 and in some applications in PT 4.

Secondary exposure - animals. Products based on hydrogen peroxide may be used in certain EU member states for disinfection of drinking water intended for animal use, for example in the breeding of broilers. The aim is to maintain a constant concentration of 5 mg/L of

hydrogen peroxide at the final delivery point in order to maintain the quality of drinking water.

2.2.1.4. Risk characterisation

In the absence of primary systemic adverse effects the risk characterisation is focused on local effects. The systemic effects, e.g. salivation, urinary incontinence and piloerection, changes in serum proteins, and changes in body weight gain, and with higher concentrations even mortalities, are considered to be secondary to the local irritation/corrosion. Although some NOAEL/LOAEL values have been set based on the study results, there is, however, no need to compare these internal values to any external dose descriptors in order to decide on the most critical effects. Hydrogen peroxide is highly reactive and will degrade rapidly at the site of first contact with organic material, and if entering blood, will be rapidly degraded.

Tables of risk characterisation for human health are presented in Appendix IV, p. 85.

Conclusion for professional uses

The exposure and accompanying risk assessments performed for professional uses of hydrogen peroxide as a disinfectant (PT 1-5) or a preservative (PT 6) demonstrated that eye protection and protective clothing against dermal exposure are needed for acceptable use when handling concentrated products, and, in some uses, diluted formulations. Protection is necessary in pre-application steps in Disinfection of animal housing by spraying (PT 3), Aseptic packaging (PT 4), Disinfection of distribution systems for drinking water (PT 4), Disinfection of drinking water for humans and animals (PT 5), and in Hand disinfection (PT 1).

In hand disinfection (PT 1), the application of 4.9 % solutions results in inhalation concentrations below the AEC (1.25 mg/m^3) both at the short periods of application and when calculated as 8-h TWA. In hand disinfection, the dermal concentration of 4.9% is ca. one seventh of the concentration irritating to skin. However, there is no sufficient information to confirm that this concentration in repeated use can be regarded as safe. The properties of specific formulations can be taken into consideration at product authorisation stage. The WG V 2014 agreed that a study on repeated dermal exposure is not required at product authorisation stage. A qualitative risk assessment should be performed for the local dermal effects taking into account the classification of the product.

Respiratory protection equipment (RPE) is needed for inhalation exposure to be below the AEC in pre-application steps in PT 1, PT 3, PT 4, PT 5, and PT 6, especially if there is insufficient ventilation or no LEV. RPE is also needed in applications (PT 3, PT 4) or post-application steps (PT 2, PT 4 and PT 6) in the maintenance work or if re-entry is allowed too soon.

Conclusion for non-professional uses:
Non-professional use of hydrogen peroxide is only intended under PT 1 (disinfection of human skin), see also the professional use, above. In the absence of a dermal repeated dose toxicity study it is not possible to assess risks caused by local effects to skin when hydrogen peroxide is used for disinfection of the skin. Hence, no safe use level of hydrogen peroxide as skin disinfectant has been set. The use of 4.9% hydrogen peroxide for disinfection of human skin (PT 1) does not represent a risk through inhalation when applied to the skin repeatedly. The WG V 2014 agreed that a study on repeated dermal exposure is not required at product authorisation stage. A qualitative risk assessment should be performed for the local dermal effects taking into account the classification of the product.

Secondary exposure

Product type 4. Pipes and containers disinfected with hydrogen peroxide are flushed before refilled with drinking water and relevant residual hydrogen peroxide is regarded as negligible under disinfection of distribution systems for drinking water (PT 4). Hydrogen peroxide used under PT 4 (aseptic packaging) evaporates while the wrapping material is heated before filling with food and no residues in food are expected.

Product type 5. The main use under this PT is in disinfection of drinking water intended for animals, see below. In addition, in some geographical sites secondary oral or dermal human exposure to residual hydrogen peroxide in food and drinking water is possible as a result of disinfection of drinking water intended for human use. As far as there is no data on the effects of chronic oral exposure, it is not possible to determine a normal reference value (Acceptable Daily Intake, ADI) for a safe level of hydrogen peroxide in drinking water. As described in Document IIB section 8.2.2.8, the final concentration of hydrogen peroxide in drinking water intended for human use is anticipated to be below 0.1 mg/L at the consumer tap. The limit value of 0.1 µg/L (as in Drinking Water Directive) for active substances and their relevant metabolites in drinking water is exceeded. However, the margin of safety is at least 1000 when the concentration in drinking water is compared to the NOAEC (NOAEL) of 100 mg/L observed in the 90-day drinking water study in a sensitive, catalase-deficient strain of mice, suggesting that potential human health risks in the general public are adequately controlled under the conditions of hydrogen peroxide use in disinfection of human drinking water. For comparison, in a risk assessment by the NSF International, dose-response data on duodenal hyperplasia incidence from the same 90-day study was used to calculate a benchmark dose, with a conclusion that a total allowable concentration of hydrogen peroxide following drinking water disinfection is 8 mg/L in drinking water, also taking into account of other sources of ingested hydrogen peroxide (oral hygiene products, food).

In the absence of a chronic oral study the 90-day oral study on catalase deficient animals resulting in a margin of safety (NOAEL/Exposure ratio) of 1000 is considered sufficient to demonstrate that potential human health risks in the general public are adequately controlled following disinfection of human drinking water. For normal animals the NOAEL can be expected to be higher than 100 ppm (100 mg/L), and consequently the NOAEL/Exposure ratio would be higher than based on the study with the catalase deficient animals. Also situations (even if currently not frequent) of life-long oral exposure are considered covered. In those considerations it was taken into account that the carcinogenicity studies with experimental deficiencies have demonstrated local effects at considerably higher concentrations, ca. 1 000 mg/L or higher. **Final concentration of hydrogen peroxide in drinking water intended for human use anticipated to be below 0.1 mg/L is considered acceptable.** It should also be noted, see below, that oral exposure to hydrogen peroxide from other sources is a common phenomenon.

Secondary oral exposure to hydrogen peroxide in food, following use of water disinfected with hydrogen peroxide, is considered of minor importance and has not been assessed in the CAR.

Secondary dermal and inhalation exposure of humans to hydrogen peroxide in water via shower and washing is considered negligible.

Risks from different sources, including water and dental care products.

Due to the lack of guidance, the RMS has not assessed the combined exposure. However, a daily oral consumer exposure level from various non-biocidal sources is estimated to be up to milligram(s). This amount includes exposure via drinking water, tooth and mouth care products as well as dietary intake by natural hydrogen peroxide in food sources. Council Directive 2011/84/EU allows the use of hydrogen peroxide, present or released, in oral products sold to consumers up to a maximum concentration of 0.1%, i.e. 1 mg/ mL (i.e. 1 g/L).

For human drinking water, additional sources of hydrogen peroxide in drinking water include hydrogen peroxide used in other types of chemical water treatments, as well as generation of hydrogen peroxide from other chemicals and UV radiation used in disinfection of water.

Relevant exposure of professionals occurs through inhalation and/or dermal route. Exposure via these routes in acceptable PTs does not substantially increase the level of exposure via drinking water, tooth and mouth care products and food.

Risk characterization - animals

Product type 5. The target concentration of hydrogen peroxide in drinking water for animals is much higher compared to the concentration in drinking water intended for human consumption. Drinking water for animals is consumed especially by chicken, broilers, which have a short lifespan, typically about one month. Due to a relatively short life-span, long term assessment is not necessary. A comparison of the final concentration of hydrogen peroxide in drinking water, 5 mg/L, to the NOAEC of 100 mg/L from the 90 day drinking water study gives a ratio of 1:20. The ratio is considered as a sufficient margin of safety for a substance with local effects at the site of contact.

If drinking water will be intended for animals with a long lifespan, such as cows, a refinement in the risk assessment would be necessary.

2.2.2. Environmental Risk Assessment

2.2.3. Fate and distribution in the environment

Hydrogen peroxide decomposes rapidly in different environmental compartments. The following processes are involved in the decomposition/degradation of hydrogen peroxide in the environment:

- Biotic degradation catalysed by microbial catalase and peroxidase enzymes
- Abiotic degradation by:
    - transition metal (Fe, Mn, Cu) and heavy metal catalysed decomposition
    - oxidation or reduction reactions with organic compounds or formation of addition compounds with organic or inorganic substances

Hydrogen peroxide decomposes into water and oxygen ($2H_2O_2 \rightarrow 2H_2O + O_2$). The rate of this reaction depends on the contact with catalytic materials and other factors such as heat and sunlight.

Hydrogen peroxide shows a very rapid biodegradation in sewage sludge with a DT_{50} of 2 minutes (at 20°C). Ready biodegradability has not been unequivocally demonstrated as the standard ready biodegradability tests are not suitable for inorganic substances. Rapid degradation of hydrogen peroxide has also been observed in surface water and soil compartments. This degradation has been proposed to be mainly microbially derived based on the difference in degradation rates between the natural and filtered/sterilized samples.

The biotic and abiotic decomposition reactions proceed in parallel with the formation reactions and the equilibrium of these reactions depends on the environmental conditions. The measured/estimated representative half-lives for different environmental compartments are shown in the following Table 2.3.1-1.

2.2.2-1 Measured/estimated half-lives of hydrogen peroxide in different environment compartments

Compartment	DT_{50} of hydrogen peroxide in tests (measured / estimated)
Sewage sludge	2 min (measured at 20°C)
Surface water	5 days (estimated*)
Soil	12 hours (estimated**)
Air	24 hours (estimated**)

* An extreme worst case DT_{50} estimate to take account for unfavourable conditions, i.e. oligotrophic cold waters with low microbial density and low transition metal concentrations.
** Worst case DT_{50} estimate based on the literature sources.

The low measured value of Henry's law constant of H = 7.5 × 10^{-4} Pa × m^3/mol indicates very low volatilisation of hydrogen peroxide from water. As hydrogen peroxide is miscible with water in all proportions and taking into account that the calculated log K_{OC} is 0.2036 mL/g, it is expected that hydrogen peroxide has a low potential for adsorption to soil and for partitioning to suspended matter or sediment.

The estimated log K_{ow} of −1.57 indicates negligible potential of bioconcentration of hydrogen peroxide in biota. The BCFs calculated according to TGD for fish and earthworm are 1.4 and 0.84, respectively. Therefore, no accumulation of hydrogen peroxide in the food chain is expected either.

2.2.4. Effects assessment

Hydrogen peroxide is toxic or moderately toxic to aquatic organisms; the LC_{50} values in the tests with fish range from 16.4 to 37.4 mg/L, the 48-h EC_{50} for invertebrates is 2.34mg/L and the E_bC_{50} for the marine diatom *Skeletonema costatum* is 2.39 mg/L. The long-term NOEC value for the reproduction of *Daphnia magna* is 0.63 mg/L representing the lowest chronic NOEC for the aquatic invertebrates and the NOEC value for *S. costatum* was 1.69 mg/L. $PNEC_{aquatic}$ is 12.6 µg/L based on the NOEC of 0.63 mg/L for *Daphnia magna*.

The PNEC for sewage treatment plant micro-organisms is 4.66 mg/L. No data for sediment-dwelling and soil organisms is available and due to the intrinsic properties of hydrogen peroxide data is not considered necessary. $PNEC_{soil}$ was calculated to be 0.0018 mg/kg using the equilibrium partitioning method. Birds and mammals are not anticipated to be directly exposed to hydrogen peroxide, thus a risk assessment for bird and mammals is not considered necessary.

The derivation of PNECs are explained in the DocIIA,Chapter 4.3.

2.2.5. PBT and POP assessment

Hydrogen peroxide shows a very rapid biodegradation in sewage sludge and rapid degradation has also been observed in surface water and soil compartments. Therefore, hydrogen peroxide does not fulfil the criteria for a persistent compound. The estimated log Kow is −1.57 and calculated BCFs for fish and earthworm are 1.4 and 0.84, respectively. Thus the bioaccumulation criterion is not fulfilled. The long-term NOEC value for *Daphnia magna* is 0.63 mg/L. Furthermore hydrogen peroxide is not classified for CMR properties and it is also not classified for specific target organ toxicity after repeated exposure (STOT RE). Therefore, the toxic criterion is not fulfilled. As hydrogen peroxide does not fulfill any of the 3 criteria, it is not a PBT substance.

Hydrogen peroxide does not fulfil criteria for being persistent organic pollutant (POP), since it is not an organic substance. In addition, hydrogen peroxide does not have potential for long-range transboundary atmospheric transport. The vapour pressure of hydrogen peroxide is below 1000 Pa (214 Pa, 20 °C), but the estimated atmospheric half-life (24 hours) is less than two days given for persistent organic pollutants (POP) as defined in the Annex D of the Stockholm Convention (2001).

2.2.6. Exposure assessment

Production

Hydrogen peroxide is always directly produced as an aqueous solution and the aqueous solutions of hydrogen peroxide are used as biocidal products. For this reason the pure active substance hydrogen peroxide is never isolated. Biocidal products are manufactured in the same processes as the a.s. not intended for biocidal use. Only a minor fraction of total

hydrogen peroxide manufactured in the EU is used as biocidal product. Therefore, emissions from the manufacturing processes are covered by the EU Risk Assessment Report (2003), and no specific environmental risk assessment for production of biocidal products was carried out.

Intended uses and emission routes in different PTs

PT 1: *Human skin disinfection* with 7.4 % (w/w) hydrogen peroxide by private and professional users. Emission to sewage is considered to be the only relevant route to the environment for this use.

PT 2.01: *Surface disinfection by VHP process* in private or public hygiene*.* Emissions were estimated for disinfection of rooms using the vaporised hydrogen peroxide (VHP) process. Air emissions are minor, since vaporised hydrogen peroxide is decomposed by the VHP machine after disinfection. Likewise, only very minor amounts of hydrogen peroxide might be discharged to sewage. Emission estimates were obtained from the tailor-made scenarios by the applicant based on information from the manufacturer of VHP machines.

PT 3: *Disinfection of animal housing* by spraying aqueous solutions of 7.4 % (w/w) of hydrogen peroxide. No direct emissions to the environment occur since all wastewaters are collected in the manure storage pit. Only negligible amounts of hydrogen peroxide may be present in manure when it is spread to soil because of degradation (fraction remaining in the manure after degradation was calculated to be 7.5×10^{-37}). Thus, it is not meaningful to calculate any environmental concentrations.

PT 4a: *Aseptic packaging:* Hydrogen peroxide is used to disinfect packaging forfood products by immersion into 35% (w/w) aqueous solutions. Emissions of spent disinfection solutions to sewage were estimated in the tailor-made emission scenario considering very large-scale creameries.

PT 4b: *Surface disinfection by VHP process* in food processing facilities. No official emission scenario document exists for this process. The VHP process used in food processing areas is the same as the process assessed under PT 2.01. Therefore, the emission calculations provided for PT 2.01 are also valid for the use of the VHP process under PT 4.

PT 4c: *Disinfection of distribution systems for drinking water:* The available emission scenario was adapted based on information from the applicant.

PT 5: *Disinfection of drinking water* for humans and animals. The available emission scenario was adapted based on information from the applicant.

PT 6.02: *Preservation of paper additives* with up to 1 % (w/w) hydrogen peroxide. Emissions may occur when the additives are used in a paper mill. Emissions were estimated on the basis of two officially adopted emission scenarios. Emission estimates were calculated for the production of printing and writing paper, tissue paper and newsprint. Only the result for the paper type with the highest emissions (newsprint) was used in the risk assessment.

PECs in STP and surface water

Assessment of potential routes of entry into the environment shows that emission to sewage is the only relevant route for the intended biocidal uses. Direct emissions of hydrogen peroxide to surface water do not occur in any of the biocidal uses evaluated. Predicted environmental concentrations (PEC) in STP effluents ranged from 0.0031 μg/L for PT 2 and PT 4b (Surface disinfection by VHP process) to 31 μg/L for PT4 (disinfection of distribution systems for drinking water). After degradation in the STP, residual hydrogen peroxide may reach surface water. Consequently, PECs in surface water (river) receiving STP effluents were in the range of 0.00031 - 3.1 μg/L assuming 10-fold dilution of the STP effluents when entering the river. Hydrogen peroxide in surface water does not partition to suspended matter or sediment to any relevant extent. Calculated PECs in sediment ranged from 2.6×10^{-7} to 0.0013 mg/kg.

Hydrogen peroxide is ubiquitous in natural waters, with typical background concentrations in surface water of 1 - 30 µg/L. The PEC values in river water estimated from potential emissions from the intended uses are generally well below compared natural background concentrations. Thus, the emissions from the intended biocidal uses are not expected to contribute significantly to the background concentrations of hydrogen peroxide in natural waters.

PEC in air

Emissions to air from biocidal uses are negligible and do not alter existing background concentrations in the troposphere to any relevant degree. Therefore, an assessment of PECs in air and rainwater from emissions due to use of biocidal products is not relevant.

PEC in soil

No direct emissions to soil are expected following the biocidal uses of hydrogen peroxide in PT 1, 2.01, 3, 4, 5 and 6.02. Indirect emissions are in principle possible from the disinfection of animal housing (PT 3), where residual hydrogen peroxide might be spread to soil with manure. However, only negligible amounts of hydrogen peroxide are expected to remain in manure when it is spread to soil, considering the very rapid degradation of hydrogen peroxide in matrices with high density microbial populations and high organic-matter content and the long storage times of manure before spreading to soil. Indirect emissions to soil are also possible from the application of sewage sludge from an STP; calculated PECs in soil ranged from 1.1 x 10^{-8} to 1.2 x 10^{-4} mg/kg.

2.2.7. Risk characterisation

STP

The PEC/PNEC ratios for STP micro-organisms are below 1 indicating that hydrogen peroxide does not cause unacceptable risk to biological processes at the sewage treatment plant concerning the evaluated uses in PT1-6.

Aquatic compartments (including sediment)

The PEC/PNEC ratios for water (sediment) are below 1 indicating that hydrogen peroxide does not cause unacceptable risk to aquatic organisms and sediment-dwelling organisms concerning the evaluated uses in PT1- 6.

Groundwater

Possible movement from soil to groundwater is calculated by EUSES according to the TGD (2003) by equations 67 and 68, where the predicted concentration in porewater of agricultural soil is taken as an indication for potential groundwater levels. Exposure of hydrogen peroxide to the soil via sludge application is possible. Accordingly calculated groundwater concentrations (presented detailed in Doc II-B, Section 8.3.3.1) from the uses in PTs 1, 2, 4abc, 5 and 6 are 0.0008, 0.000013, 0.01, 0.000013, 0.13, 0.003 and 0.02 µg/L, respectively. The maximum permissible concentration by directive 2006/18/EC of 0.1 µg/L is exceeded only slightly in one use and taking into account the very worst-case assumptions (i.e. no transformation in deeper soil layers), the risk to unacceptable groundwater contamination can be regarded as low and insignificant.

Terrestrial compartment

Direct exposure of soil in any of the uses is assessed to be negligible. It is possible that soil may become exposed following the spreading of sewage sludge from a sewage treatment plant that has been exposed to hydrogen peroxide from uses in PTs 1, 2, 4, 5 and 6. From the uses in PT3 exposure to soil via manure application is assessed to be negligible due to the expected rapid degradation of hydrogen peroxide in manure. All the estimated PEC/PNEC ratios for the

soil compartment are below 1 indicating no unacceptable risk to soil organisms. Other terrestrial organisms are not regarded to be exposed with the proposed use patterns.

Potential for secondary poisoning

The estimated log K_{ow} of hydrogen peroxide is −1.57 indicating a negligible potential for bioconcentration in biota. Therefore, accumulation of hydrogen peroxide in the food chain is not expected, and the risk of secondary poisoning in aquatic and terrestrial predators is considered negligible.

Aggregated (environmental) exposure assessment

According to Article 10(1) of BPD a cumulative risk assessment shall be performed where relevant. For hydrogen peroxide it was agreed at the WG V 2014 that aggregated risk assessment is not regarded relevant due to the high reactivity of the substance.

2.2.8. Assessment of endocrine disruptor properties

Hydrogen peroxide is not included in the Commission staff working document on implementation of the Community Strategy for Endocrine Disrupters - a range of substances suspected of interfering with the hormone systems of humans and wildlife (COM (1999) 706)). There is no evidence of any endocrine disruption potential in the human health or ecotoxicological studies presented in the dossier.

2.3. Overall conclusions

The outcome of the assessment for hydrogen peroxide in product-types 1-6 is specified in the BPC opinions following discussions at the 9 meeting of the Biocidal Products Committee (BPC). The BPC opinions are available from the ECHA website.

2.4. List of endpoints

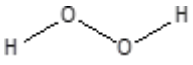
The most important endpoints, as identified during the evaluation process, are listed in Appendix I.

Appendix I: List of endpoints

Chapter 1:Identity, Physical and Chemical Properties, Classification and Labelling

Active substance (ISO Name)	Hydrogen peroxide
Product-type	Bactericide, Fungicide, Sporicide, Virucide. PT1: Human hygiene biocidal products, PT2: Private area and public health area disinfectants and other biocidal products, PT3: Veterinary hygiene biocidal products, PT4: Food and feed area disinfectants, PT5: Drinking water disinfectants, PT6: In-can preservatives

Identity

Chemical name (IUPAC)	Hydrogen peroxide
Chemical name (CA)	Hydrogen peroxide
CAS No	7722-84-1
EC No	231-765-0
Other substance No.	-
Minimum purity of the active substance as manufactured (g/kg or g/l)	Hydrogen peroxide is always directly produced as an aqueous solution and these aqueous solutions of hydrogen peroxide range from 35 % to <70 % (by wt). Min purity on a calculated dry weight basis is ca 99.5% Detailed specification of the content of hydrogen peroxide in the Confidential Doc A2.Detailed specifications of the content of hydrogen peroxide in the biocidal products are given in Document IIB 6.1
Identity of relevant impurities and additives (substances of concern) in the active substance as manufactured (g/kg)	There is no single impurity in the substance at concentration > 0.1%. The sum of organic and inorganic impurities in aqueous solution is below 0.2 % (by wt). Calculated from the 35 % aqueous solution of HP, the theoretical dry weight total impurity contents is below 0.5 % (by wt). Max level set for heavy metals, Pb, Hg, Cd, As, max 1g/kg in aqueous solution, for each. Stabilizers in hydrogen peroxide, including confidential stabilizers are listed in the Confidential A2
Molecular formula	H_2O_2
Molecular mass	34.01
Structural formula	

Physical and chemical properties

Melting point (state purity)	Freezing point : - 0.43°C, (extrapolated for theoretical hydrogen peroxide content of 100%)
Boiling point (state purity)	150.2°C at 101.3 kPa, (extrapolated for theoretical hydrogen peroxide content of 100%)
Thermal stability / Temperature of decomposition	Not tested due to explosion risk
Appearance (state purity)	Clear, colourless liquid (purity not stated)
Relative density (state purity)	1.4425 g/cm^3 at 25°C, (extrapolated for theoretical hydrogen peroxide content of 100%)
Surface tension (state temperature and concentration of the test solution)	80.4 mN/m at 20°C, (extrapolated for theoretical hydrogen peroxide content of 100%)
Vapour pressure (in Pa, state temperature)	at 20°C (293 K) : 214 Pa at 25°C (298 K) : 299 Pa (both values extrapolated for theoretical hydrogen peroxide content of 100%)
Henry's law constant (Pa m^3 mol $^{-1}$)	7.5*10^{-4} Pa*m^3/mol at 20°C (293K), (purity not stated)
Solubility in water (g/l or mg/l, state temperature)	pH 5 at ___ ºC: Miscible with water in all proportions pH 9 at ___ ºC: pH *[X]* at ___ ºC:
Solubility in organic solvents (in g/l or mg/l, state temperature)	Hydrogen peroxide and highly concentrated aqueous solutions (> 65 wt-%) of H_2O_2 are soluble in a variety of organic solvents such as carboxylic esters. Pure hydrogen peroxide should not be mixed with organic solvents (risk of violent reactions). For information on solubility in organic solvents in production, see Doc A2 and the Confidential A2. Once formed, the limited solubility in organic solvents makes hydrogen peroxide extractable in water.
Stability in organic solvents used in biocidal products including relevant breakdown products	Hydrogen peroxide as manufactured does not include an organic solvent.
Partition coefficient (log P_{OW}) (state temperature)	pH 5 at ___ ºC: pH 9 at ___ ºC: pH *[X]* at ___ ºC: log Kow : −1.57 (calculated)
Dissociation constant	2.4 * 10^{-12} at 25°C, pKa: 11.62
UV/VIS absorption (max.) (if absorption > 290 nm state ε at wavelength)	E = 19.6±0.3 litres/mol*cm at 253.7 nm, no relevant absorption at wavelenghts > 290 nm

Flammability or flash point	Hydrogen peroxide itself is not flammable but it can cause spontaneous combustion of flammable materials. Explosive vapour phases can only be formed of aqueous hydrogen peroxide solutions with concentrations higher than 70% (w/w) at temperatures above 110°C
Explosive properties	Hydrogen peroxide itself is not flammable but it can cause spontaneous combustion of flammable materials. Explosive vapour phases can only be formed of aqueous hydrogen peroxide solutions with concentrations higher than 70% (w/w) at temperatures above 110°C
Oxidising properties	Hydrogen peroxide is a strong oxidizer.
Auto-ignition or relative self ignition temperature	-

Classification and proposed labelling

with regard to physical hazards	Ox. Liq. 1; H271 May cause fire of explosion; strong oxidiser Specific Concentration Limits: Ox. Liq.1; H271 C ≥ 70 %**** Ox. Liq. 2; H272:50 % ≤ C < 70 %**** Pictogram: GSH03
with regard to human health hazards	Acute Tox. 4 *; H332 Harmful if inhaled Acute Tox. 4 *; H302 Harmful if swallowed. Skin Corr. 1A; H314 Causes severe skin burns and eye damage Specific Concentration Limits: Skin Corr. 1A; H314: C ≥ 70 % Skin Corr. 1B;H314: 50 % ≤ C <70 % Skin Irrit. 2;H315: 35 % ≤ C <50 % Eye Dam. 1;H318: 8 % ≤ C <50 % Eye Irrit. 2; H319:5 % ≤ C < 8 % STOT SE 3;H335; C ≥ 35 % * Pictograms: GHS03, GHS05 and GHS07 Signal Word Code: Danger
with regard to environmental hazards	H412 Harmful to aquatic life with long lasting effects
	Generic Concentration limits: Aquatic Chronic 3; H412: C ≥ 25 % should be applied according to the 2 ATP to CLP Regulation

	Notes. B

Chapter 2:Methods of Analysis

Analytical methods for the active substance

Technical active substance (principle of method)	Titrimetric determination [redacted]
Impurities in technical active substance (principle of method)	Not applicable, no relevant impurities

Analytical methods for residues

Soil (principle of method and LOQ)	Not applicable, because hydrogen peroxide is rapidly decomposed in soil and does not adsorb to soil matrix. Trace amounts of hydrogen peroxide in soil water may be analysed by the method for water.
Air (principle of method and LOQ)	Spectrometric determination [redacted].New method must be submitted before product authorisation, or the existing validated.
Water (principle of method and LOQ)	Spectrometric determination [redacted] New method must be submitted before product authorisation, or the existing validated.
Body fluids and tissues (principle of method and LOQ)	Not required as the substance is not acutely toxic (T) or very toxic (T+)
Food/feed of plant origin (principle of method and LOQ for methods for monitoring purposes)	Not required as not expected in food/feed of plant origin
Food/feed of animal origin (principle of method and LOQ for methods for monitoring purposes)	Not required as not expected in food/feed of animal origin

Chapter 3:Impact on Human Health

Absorption, distribution, metabolism and excretion in mammals

Rate and extent of oral absorption:	No significant absorption, local effects
Rate and extent of dermal absorption*:	Not determined
Distribution:	None
Potential for accumulation:	None
Rate and extent of excretion:	None
Toxicologically significant metabolite(s)	None

* the dermal absorption value is applicable for the active substance and might not be usable in product authorization

Acute toxicity

Rat LD_{50} oral	805 mg/kg bw (70 %, combined). (486 mg/kg bw; as 100%, females) 1232 mg/kg bw (35 %, combined) (420 mg/kg bw; as 100%, males)
Rat LD_{50} dermal	>2000 mg/kg bw
Rat LC_{50} inhalation	>170 mg/m^3 (vapour, highest attainable vapour concentration)

Skin corrosion/irritation	35%≤ c <50% H_2O_2: irritant ≥50% H_2O_2: corrosive
Eye irritation	5%≤ c <8% H_2O_2: irritant ≥8% H_2O_2: severe irritant
Respiratory tract irritation	Is an irritating substance to respiratory tract
Skin sensitisation (test method used and result)	Not sensitizing (modified Magnusson-Kligman and human data)
Respiratory sensitisation (test method used and result)	-

Repeated dose toxicity

Short term

Species / target / critical effect	Rodent
Relevant oral NOAEL / LOAEL	Not established, not systemically available
Relevant dermal NOAEL / LOAEL	Not established
Relevant inhalation NOAEC / LOAEC	2 ppm; 2.9 mg/m^3 (28-day rat)

Subchronic

Species/ target / critical effect	Rodent
Relevant oral NOAEL / LOAEL	NOAEL: 26 mg/kg bw/day (100 ppm, 90-day mouse, catalase-deficient strain)
Relevant dermal NOAEL / LOAEL	Not established
Relevant inhalation NOAEL / LOAEL	NOAEC: 10 mg/m^3 (7 ppm, 90-day rat)

Long term

Species/ target / critical effect	
Relevant oral NOAEL / LOAEL	not assessed

Relevant dermal NOAEL / LOAEL	not assessed
Relevant inhalation NOAEL / LOAEL	not assessed

Genotoxicity

Mutagenic *in vitro* in a bacterial gene mutation test both in the presence and absence of metabolic activation, in a mammalian gene mutation test without metabolic activation and in a chromosomal aberration test in the presence and absence of metabolic activation.

The positive results of the *in vitro* genotoxicity tests were not confirmed *in vivo* in a bone marrow micronucleus test and a UDS test in liver cells. However, there is no evidence that hydrogen peroxide has reached the target organ in the *in vivo* UDS test. Likewise, in micronucleus test with exposure via drinking water hydrogen peroxide has most probably not reached the bone marrow. Due to the uncertainty with regard to availability of the test substance in the target organ, the result of this study is equivocal. In a micronucleus test with i.p. application, no increase in the frequency of micronucleated polychromatic erythrocytes was observed. Effects in the bone marrow (decreased ratio of polychromatic erythrocytes to normochromatic erythrocytes) were noticed indicating the suitability of test conditions.

The results of the genotoxicity studies do not meet the current classification criteria for mutagenicity.

Information on local genotoxicity in literature was added in the CAR. Due to the irritating properties of hydrogen peroxide, the risk mitigation measures include the use of personal protective equipment. The RMS considers the protections sufficient, and does not consider the local genotoxicity as a relevant endpoint for a risk assessment.

Carcinogenicity

Species/type of tumour	Not considered carcinogenic. Primary local irritation (corrosion) at the site of contact. Not considered genotoxic in vivo. Genotoxic mechanisms cannot be ruled out in the carcinogenicity of hydrogen peroxide. However, the endogenous defence mechanisms against reactive oxygen species may suggest a threshold for carcinogenicity of hydrogen peroxide
Relevant NOAEL/LOAEL	

Reproductive toxicity

Developmental toxicity

Species/ Developmental target / critical effect	Not reprotoxic, no systemic availability
Relevant maternal NOAEL	
Relevant developmental NOAEL	

Fertility

Species/critical effect	Not reprotoxic, no systemic availability
Relevant parental NOAEL	
Relevant offspring NOAEL	
Relevant fertility NOAEL	

Neurotoxicity

Species/ target/critical effect	Available studies give no indication of a neurotoxic potential

Developmental Neurotoxicity

Species/ target/critical effect	Available studies give no indication of a neurotoxic potential

Immunotoxicity

Species/ target/critical effect	

Developmental Immunotoxicity

Species/ target/critical effect	

Other toxicological studies

Medical data

Information from EU Risk Assessment Report (2003)
and publications:

Reports of respiratory irritation symptoms when exposed to hydrogen peroxide vapour with progressive dyspnoea and bilateral diffuse nodular infiltration of lung. Improvement after withdrawal from exposure.

Reports of irritation in the eyes and airways, headaches, temporary loss of olfaction, symptoms and signs in the skin, and blanching of hair.

Human poisoning by oral ingestion. Oxygen embolism has been observed.

Summary

	Value	Study	Safety factor
AEC inhalation$_{long\text{-}term}$	1.25 mg/m^3	NOAEC in 90-day inhalation study (rat)	8

AEC inhalation$_{medium-term}$	1.25 mg/m^3	NOAEC in 90-day inhalation study (rat)	8
AEC inhalation$_{acute}$	1.25 mg/m^3	NOAEC in 90-day inhalation study (rat)	8
ADI[2]	ADI not established, the substance is not systemically available. The agreed acceptable max concentration is 0.1 mg/L in human drinking water. In the main use in PT 5, drinking water of chicken, max concentration: 5 mg/L		
ARfD	not established		

MRLs

Relevant commodities	-

Reference value for groundwater

According to BPR Annex VI, point 68	-

Dermal absorption

Study (*in vitro/vivo*), species tested	not feasible, not assessed
Formulation (formulation type and including concentration(s) tested, vehicle)	-
Dermal absorption values used in risk assessment	100 % as default

Acceptable exposure scenarios (for method of calculation see App IV)

Formulation of biocidal product	No risk characterization is made
Intended uses	
Industrial users	

[2] If residues in food or feed.

Professional users	**PT1** *Disinfection of human skin***:** Dilution of product to in-use concentration of 7.4/4.9% hydrogen peroxide and application. PPE in M/L: gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV. Application with in-use concentration of 4.9% is acceptable. **PT2.01** S*urface disinfection by VHP process*: no exposure (35% hydrogen peroxide) . RPE in re-entry if the concentration higher than $AEC_{inhalation}$. **PT3** *Disinfection of animal housings*: Dilution of product to in-use concentration of 7.4% hydrogen peroxide and application. PPE: gloves, coverall, goggles/face shield, RPE **PT4** *Aseptic packaging*: loading and use in aseptic packaging machine (measured data) (35% hydrogen peroxide). PPE: gloves, coverall, goggles/face shield, RPE in loading and maintenance work. *Surface disinfection by VHP process*: no exposure (35% hydrogen peroxide). RPE in re-entry if the concentration higher than $AEC_{inhalation}$ *Disinfection of distribution systems for drinking water*: Dilution of product to in-use concentration of 4% hydrogen peroxide and application. PPE: gloves, coverall, goggles/face shield, RPE **PT5** *Drinking water disinfection*: Loading and application (no exposure expected). PPE in M/L: gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV **PT6.02** *Preservative for paper additives*: Loading and application. PPE: gloves, coverall, goggles/face shield, RPE in loading and maintenance work
Non professional users	In PT1, Disinfection of human skin with a ready-to-use product, as in professionals
General public	Secondary exposure of animals or consumer by residual hydrogen peroxide in drinking water after disinfection (PT5)
Exposure via residue in food	No exposure expected

Chapter 4: Fate and Behaviour in the Environment

Route and rate of degradation in water

Hydrolysis of active substance and relevant metabolites (DT_{50}) (state pH and temperature)	Not applicable. No hydrolysis because of the chemical nature of the compound. However, decomposition of hydrogen peroxide is catalysed abiotically by transition metal ions. The half-life in surface water includes abiotic catalysis, next to biotic degradation.
pH 5	
pH 9	
Other pH: *[indicate the value]*	
Photolytic / photo-oxidative degradation of active substance and resulting relevant metabolites	Not applicable. Absorption spectrum shows no propensity of the molecule to be decomposed by UV/VIS light.
Readily biodegradable (yes/no)	Yes Half-life in activated sludge stage of sewage treatment plants: 2 minutes
Inherent biodegradable (yes/no)	No
Biodegradation in freshwater	Half-life in freshwater: 5 days
Biodegradation in seawater	Not relevant
Non-extractable residues	None
Distribution in water / sediment systems (active substance)	Hydrogen peroxide does not partition from the water phase, adsorption to sediment is negligible
Distribution in water / sediment systems (metabolites)	Not relevant, no relevant metabolites

Route and rate of degradation in soil

Mineralization (aerobic)	Rapidly decomposed in soil to water and oxygen. Worst-case half-life of 12 hours.
Laboratory studies (range or median, with number of measurements, with regression coefficient)	
DT_{50lab} (20°C, aerobic):	Not applicable
DT_{90lab} (20°C, aerobic):	Not applicable
DT_{50lab} (10°C, aerobic):	Not applicable
DT_{50lab} (20°C, anaerobic):	Not applicable
degradation in the saturated zone:	Not applicable
Field studies (state location, range or median with number of measurements)	
DT_{50f}:	Not applicable

DT_{90f}:	Not applicable
Anaerobic degradation	Not applicable
Soil photolysis	Not applicable. Absorption spectrum shows no propensity of the molecule to be decomposed by UV/VIS light.
Non-extractable residues	None
Relevant metabolites - name and/or code, % of applied a.i. (range and maximum)	None
Soil accumulation and plateau concentration	Not applicable

Adsorption/desorption

Ka , Kd Ka_{oc} , Kd_{oc} pH dependence (yes / no) (if yes type of dependence)	Log K_{OC} = 0.2036 estimated by QSAR Miscible with water at all proportions, negligible adsorption expected.

Fate and behaviour in air

Direct photolysis in air	Hydrogen peroxide is part of a complex equilibrium system of photooxidants in the troposphere. For risk assessment purposes, a half-life of 24 hours was derived
Quantum yield of direct photolysis	Not applicable
Photo-oxidative degradation in air	Latitude: ..... Season: ..... DT_{50} .....
Volatilization	Volatilisation of hydrogen peroxide from aqueous solutions is negligible.

Reference value for groundwater

According to BPR Annex VI, point 68	0.1 µg/l

Monitoring data, if available

Soil (indicate location and type of study)	None
Surface water (indicate location and type of study)	Typical natural background concentrations 1 - 30 µg/L, maximum > 100 µg/L (Various locations and studies)
Ground water (indicate location and type of study)	Typical natural background concentration 0.7 µg/L, maximum 2.3 µg/L (Various locations and studies)

Air (indicate location and type of study)	Typical natural background concentrations 0.14-1.4 µg/m3 (0.1-1 ppb), maximum 10 µg/m3 (7 ppb) (Various locations and studies)

Chapter 5: Effects on Non-target Species

Toxicity data for aquatic species (most sensitive species of each group)

Species	Time-scale	Endpoint	Toxicity
Fish			
Pimephales promelas	96 h, semi-static	mortality, LC50	16.4
Invertebrates			
Daphnia pulex (crustaceans)	48 h, semi-static	immobility, EC_{50}	2.34
Daphnia magna (crustaceans)	21 d, flow-through	reproduction, NOEC	0.63
Algae			
Skeletonema costatum (marine diatom)	72 h, static	growth rate, NOEC	~~0.63~~ 1.69
Microorganisms			
Activated sludge from sewage treatment plant	0.5 h & 3 h , static	respiration inhibition, EC50	466

Effects on earthworms or other soil non-target organisms

Acute toxicity to ……..	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required
Reproductive toxicity to ……	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required

Effects on soil micro-organisms

Nitrogen mineralization	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required
Carbon mineralization	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required

Effects on terrestrial vertebrates

Acute toxicity to mammals	70% H2O2: LD50 (rat) = 694 & 1026 mg/kg bw (females & males); 35% H2O2: LD50 (rat) = 1270 & 1193 mg/kg bw (females & males)

Acute toxicity to birds	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required
Dietary toxicity to birds	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required
Reproductive toxicity to birds	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required

Effects on honeybees

Acute oral toxicity	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required
Acute contact toxicity	

Effects on other beneficial arthropods

Acute oral toxicity	no exposure, since no direct or indirect release to the terrestrial environment. Therefore no test required
Acute contact toxicity	
Acute toxicity to ……..	

Bioconcentration

Bioconcentration factor (BCF)	no test required due to log Kow of appr. −1.5 calculated BCFs according to TGD: fish 1.4 earthworm 0.84
Depration time (DT_{50})	Not applicable
Depration time (DT_{90})	Not applicable
Level of metabolites (%) in organisms accounting for > 10 % of residues	Not applicable

Chapter 6: Other End Points

Appendix II: List of Intended Uses

Object and/or situation	Member State or Country	Product name	Organisms controlled	Formulation		Application			Applied amount per treatment			Remarks:
				Type (a)	Conc. of a.s. (b)	method kind (c, d)	number min max (e)	interval between applications (min)	In- use concen-tration min max	water L/m^2 min max	g as/m^2 min max	(f)
PT1: Dis-infection of human skin	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Direct appli-cation onto skin	10 appli-cations daily	As required by user	7.4/4.9 % (w/w) 49 - 74 g/kg	n.a.	n.a.	For private or professional users, e.g. disinfection of hands and forearms by workers in food processing industries.
PT2: Surface dis-infection by VHP process	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Vapori-sation with VHP machine	As required by user	As required by user	Vapour concen-tration 250 - 400 ppm 350 - 560 mg/m^3	n.a.	n.a.	Vaporisation of closed rooms (e.g., in hospitals, emergency vehicles, biological laboratories). Professional users
PT3: Dis-infection of animal housing	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Overall, airless spray	Depends on life-cycle of animals	Depends on life-cycle of animals	7.4% (w/w) 74 g/kg	Spray until run-off	n.a.	Application by professional users to cleaned housing after animals were removed.

Object and/or situation	Member State or Country	Product name	Organisms controlled	Formulation		Application			Applied amount per treatment			Remarks:
				Type (a)	Conc. of a.s. (b)	method kind (c, d)	number min max (e)	interval between applications (min)	In- use concen-tration min max	water L/m^2 min max	g as/m^2 min max	(f)
PT4: Aseptic packaging	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Immersion	1	n.a.	35% (w/w) 350 g/kg	n.a.	n.a.	Packaging material is immersed briefly into a bath containing heated hydrogen peroxide solution. Professional users
PT4: Surface disinfection by VHP process	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Vapori-sation with VHP machine	As required by user	As required by user	Vapour concen-tration 250 - 400 ppm 350 - 560 mg/m^3	n.a.	n.a.	Vaporisation of closed rooms in food processing facilities. Professional users
PT4 : Disinfection of distribu-tion systems for drinking water	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Pipes: flooding Tanks: spraying	1	n.a.	2% (w/w) 20 g/kg	Pipes: as needed for flooding Tanks: spray until run-off	n.a.	Professional users

Object and/or situation	Member State or Country	Product name	Organisms controlled	Formulation		Application			Applied amount per treatment			Remarks:
				Type (a)	Conc. of a.s. (b)	method kind (c, d)	number min max (e)	interval between applications (min)	In- use concen-tration min max	water L/m^2 min max	g as/m^2 min max	(f)
PT5: Disinfection of drinking water	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Continuous dosing	n.a.	n.a.	Initial concen-ntration : 25 mg/L	n.a.	n.a.	Hydrogen peroxide is dosed continuously by an automated on-line sensor into drinking water at the waterworks. Professional users
PT6: Preser-vative for paper additives	EU	Hydrogen peroxide	Bacteria, fungi and viruses	SL	49.9 or 35% (w/w); 499 or 350 g/kg	Mixing	1	n.a.	1.0% (w/w) 10 g/kg	n.a.	n.a.	Hydrogen peroxide is mixed into aqueous systems (e.g., paper additives) to preserve them during storage and transport. Professional users

SL - soluble concentrate (CIPAC formulation code); n.a. - not applicable

(a) *e.g.* wettable powder (WP), emulsifiable concentrate (EC), granule (GR)
(b) g/kg or g/l;
(c) Method, *e.g.* high volume spraying, low volume spraying, spreading, dusting, drench;
(d) Kind, *e.g.* overall, broadcast, aerial spraying, row, bait, crack and crevice equipment used must be indicated;
(e) Indicate the minimum and maximum number of application possible under practical conditions of use;
(f) Remarks may include: Extent of use/economic importance/restrictions
All abbreviations used must be explained

Appendix III: List of studies

Data protection is claimed by the applicant in accordance with Article 60 of Regulation (EU) No 528/2012.

Note: An additional column was introduced documenting the citation of each study as used in the EU Risk-Assessment Report, for ease of comparison.

CEFIC - CEFIC Peroxygens Sector Group, Hydrogen peroxide subgroup
* - Key study

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A3.1.1	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation, New York, 212 Non-GLP, published	N	
A3.1.2	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation, New York, 230 Non-GLP, published	N	
A3.1.3	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation, New York, 193, 195 and 199 Non-GLP, published	N	
A3.2	Weast R. et al. *	Weast and Melvin (1981)	1985 - 1986	Vapor pressure, Variation with Temperature, In: CRC Handbook of Chemistry and Physics, 66th edition, 1985-1986, Weast R., Astle M. and Beyer W. (eds), D-213 Non-GLP, published	N	
A3.2.1/01	Brachhold H. *	not applicable	2006	Estimation of the Henry`s Law Constant of Hydrogen Peroxide CAS-No.: 7722-84-1 by Quantitative Structure Activity Relationship (QSAR-Method = Calculation), Report No.: 2006-0180-DKB, 2006 Non-GLP, unpublished	Y	CEFIC
A3.2.1/02	Hwang H. and Dasgupta P.	Hwang and Dasgupta (1985)	1985	Thermodynamics of the hydrogen peroxide-water system, Environmental Science and Technology., 1985, Vol. 19, 255-258 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A3.3	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 444 Non-GLP, published	N	
A3.4/01	Goor G. et al.*	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 445 Non-GLP, published	N	
A3.4/02	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation, New York, 291 and 275 Non-GLP, published	N	
A3.5	Goor G. et al *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 444 Non-GLP, published	N	
A3.6	Weast R. et al. *	Weast and Melvin (1981)	1985 - 1986	Dissociation constants of inorganic acids in aqueous solutions, In: CRC Handbook of Chemistry and Physics, 66th edition, Weast R., Astle M. and Beyer W. (eds), D-163 Non-GLP, published	N	
A3.7	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 463 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A3.9	Brachhold H. *	not applicable	2006	Estimation of the Partition coefficient (n-Octanol/Water) of Hydrogen Peroxide CAS-No.: 7722-84-1 by Quantitative Structure Activity Relationship (QSAR-Method = Calculation), Report No.: 2006-0234-DKB, 2006 Non-GLP, unpublished	Y	CEFIC
A3.10	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 462 Non-GLP, published	N	
A3.11	Woodbury C. *	not applicable	1983	International Labour Office, Encyclopedia of Occupational Health and Safety, Vols. I&II, Geneva, Switzerland, 1089 Non-GLP, published	N	
A3.12	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 462 Non-GLP, published	N	
A3.13	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation, New York, 205 and 206 Non-GLP, published	N	
A3.14	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation, New York, 202 and 203 Non-GLP, published	N	
A3.15/01	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 461 and 462 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A3.15/02	Kratz *	Degussa (1977a)	1988	Thermische Sensibilitaet von hochkonzentriertem H2O2, Sicherheitstechnisches Prüfzentrum, Degussa FCPH-S, D, Degussa AG-US-IT-Nr. 88-0264-DKS, Non-GLP, unpublished	Y	CEFIC
A3.16	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 446 Non-GLP, published	N	
A3.17	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 461 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A4.2.b	Kilgour J D*	None	2001	Hydrogen peroxide: feasibility study. Central toxicology laboratory, Alderley Park, Macclesfield, Cheshire, UK Report no. CTL/TZ0357/SUM/REPT CEFIC reference CO0428. 10.10.2001 Non-GLP, unpublished	Y	CEFIC

Effectiveness against target organisms and intended uses

No references submitted. See the 49.9% product, below.

Toxicological studies

Note: An additional column was introduced documenting the citation of each study as used in the EU Risk-Assessment Report, for ease of comparison.

CEFIC - CEFIC Peroxygens Sector Group, Hydrogen peroxide subgroup
* - Key study

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.1.1/01	[redacted] *	[redacted] (1996)	1996	Acute oral toxicity study with hydrogen peroxide (70%) in male and female rats [redacted] GLP, unpublished	N	
A6.1.1/02	[redacted]	[redacted] (1983b)	1983	[redacted]. (1983) Acute Oral Toxicity of 35% Hydrogen Peroxide in Rats. Study No. I83-745. [redacted] GLP, unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.1.1/03	[redacted]	[redacted] (1997a)	1997	Hydrogen peroxide: determination of toxic effects following intravenous dosing of the rat. Final Report No. 514/23-1052. [redacted] GLP, unpublished	Y	CEFIC
A6.1.2/01	[redacted]*	[redacted] (1979b)	1979	Acute Dermal Toxicity of 70% Hydrogen Peroxide in Rabbits. Study No. ICG/T79027-02. [redacted] Non-GLP, unpublished	Y	CEFIC
A6.1.2/02	[redacted]*	[redacted] (1983a)	1983	[redacted] (1983). Acute Dermal Toxicity of 35% Hydrogen Peroxide in Rabbits. Study No. I83-746. [redacted] GLP, unpublished	Y	CEFIC
A6.1.2.1	[redacted]*	[redacted] (1979b)	1979	Acute Dermal Toxicity of 70% Hydrogen Peroxide in Rabbits. Study No. ICG/T79027-02. [redacted]. Non-GLP, unpublished	Y	CEFIC
A6.1.3	[redacted]*	[redacted] (1990a)	1990	An acute inhalation toxicity study of vapors of hydrogen peroxide (50%) in the rat [redacted] Project No.: 89-8233, FMC No. 189-1080 GLP, unpublished	Y	CEFIC
A6.1.3/01	[redacted] (a)	[redacted] 1995a	1995	An acute inhalation study of hydrogen peroxide aerosols in male mice [redacted] Laboratory Project ID: DT 94/03 GLP, unpublished	Y	CEFIC
A6.1.3/02	[redacted] (b)	[redacted] 1995b	1995	An evaluation of the respiratory irritating properties of hydrogen peroxide aerosols in male swiss mice [redacted] Laboratory Project ID: DT 94/04 GLP, unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.1.4.1/01	[redacted]*	[redacted] (1990c)	1990	[redacted] (1990); Hydrogen peroxide 10% standard grade – Primary skin irritation study in rabbits [redacted] Study number: I89-1078 GLP, unpublished	Y	CEFIC
A6.1.4.1/02	[redacted]*	[redacted] (1983d)	1983	[redacted] (1983); 35% Hydrogen peroxide – Primary skin irritation and skin corrosion [redacted] Report number: I83-747 GLP, unpublished	Y	CEFIC
A6.1.4.1/03	[redacted]*	[redacted] (1990d)	1990	Hydrogen peroxide 50% standard grade – Primary skin irritation study in rabbits [redacted] Study number: I89-1079 GLP, unpublished	Y	CEFIC.
A6.1.4.2/01	[redacted]*	[redacted] (1972c)	1972	Federal hazardous substance act test – rabbit eye irritation Haskell Laboratory for [redacted] Non-GLP, unpublished	N	
A6.1.4.2/02	[redacted]*	[redacted] (1987a)	1987	5% Hydrogen peroxide – Preliminary eye irritation study in rabbits [redacted] report number: I86-0949, GLP, unpublished	Y	CEFIC
A6.1.4.2/03	[redacted]*	[redacted] (1996)	1996	Eye irritation and skin corrosion evaluations with hydrogen peroxide [redacted] J Am Coll Toxicol, Vol 15, Suppl 1, S112-4, 1996 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.1.4.2/04	[redacted]*	[redacted] (1987b)	1987	[redacted] (1987); Hydrogen peroxide 8% STD, Preliminary eye irritation study in rabbits [redacted]A Study Number: I87-0950 GLP, unpublished	Y	CEFIC
A6.1.4.2/05	[redacted]*	[redacted] (1985)	1985	Preliminary eye irritation of 10% hydrogen peroxide in rabbits [redacted] report number: I84-851, GLP, unpublished	Y	CEFIC
A6.1.5	[redacted]	[redacted] (1953)	1953	Primary irritancy and skin sensitization test. Memphis hydrogen peroxide 3%. [redacted] Non-GLP, unpublished	N	Du Pont
A6.1.5/01 A6.12.6/01	Aguirre A et al.	Aguirre A, Cabala R, Sanz de Galdeano C, Landa L, and Diaz-Pérez JL (1991)	1994	Positive patch tests to hydrogen peroxide in 2 cases, Contact Dermatitis 30, 113 Non-GLP, published	N	
A6.1.5/02 A6.12.6/02	Kanerva L et al.	Kanerva L, Jolanki R, Riihimäki V and Kalimo K (1998)	1998	Patch test reactions and occupational dermatoses caused by hydrogen peroxide. Contact Dermatitis 39, 146 Non-GLP, published	N	
A6.2/01	Ogata M	Ogata, M (1991)	1991	Acatalasemia. Hum.Genet. 86,331-340 Non-GLP, published	N	
A6.2.1/01	Aebi H & Suter H	Aebi, H. and Suter, H. (1972)	1972	In: The Metabolic Basis of Inherited Diseases. Stanburg JB, Wyngaarden J.B. and Fredrickson D.S. (eds), McGraw-Hill, New York, 1710-1750 Non-GLP, published	N	
A6.2.1/02	Boveris A	Boveris, A. (1977)	1977	Mitochondrial production of superoxide radical and hydrogen peroxide. In: Tissue hyperoxia and Ischemia. Reivich M., Coburn R., Lahiri S. and Chance B (eds) Plenum, New York, 67-82 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.2.1/03	Bryan CL & Jenkinson SG	Bryan, C.L. and Jenkinson, S.G. (1987)	1987	Species variation in lung antioxidant enzyme activities. J. Appl. Physiol. 63, 597-602 Non-GLP, published	N	
A6.2.1/04	Chalmers RL	Chalmers, R.L. (1989)	1989	Hydrogen peroxide in anterior segment physiology: a literature review. Optometry and Vision Science 66, 796-803 Non-GLP, published	N	
A6.2.1/05	Chance B et al.	Chance, B.; Sies, H. and Boveris, A (1979)	1979	Hydroperoxide metabolism in mammalian organs. Physiological Reviews 59, 527-605 Non-GLP, published	N	
A6.2.1/06	Chance KV & Traub WA	Chance, K.V. and Traub,W.A. (1987)	1987	Evidence for stratospheric hydrogen peroxide. Journal of Geophysical Research 92, 3061-3066 Non-GLP, published	N	
A6.2.1/07	Desagher S et al.	Desagher, S.; Glowinski, J. and Premont, J (1996)	1996	Astrocytes protect neurons from hydrogen peroxide toxicity. J. Neurosci. 16, 2553-2562 Non-GLP, published	N	
A6.2.1/08	Engstrom PC et al.	Engstrom, P.C.; Easterling, L.; Baker, R.R. and Matalon, S (1990)	1990	Mechanism of extracellular hydrogen peroxide clearance by alveolar type II pneumocytes. J. Appl. Physiol. 69, 2078-2084 Non-GLP, published	N	
A6.2.1/09	Erzurum SC et al.	Erzurum, S.C.; Danel, C.; Gillissen, A.; Chu, C.S.; Trapnell, B.C. and Crystal, R.G. (1993)	1993	In vivo antioxidant gene expression in human airway epithelium of normal individuals exposed to 100% o2. J. Appl. Physiol. 75, 1256-1262 Non-GLP, published	N	
A6.2.1/10	Ferrari M et al.	Ferrari, M; Catena, S; Ferrari, F.; Bianchi, R. and Villani, A. (1994)	1994	Gave embolia polmonare gassosada somministrazione intraoperatoria di acqua ossigena. Minerva Anestesiol. 60, 403-406 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.2.1/11	Fridovich I	Fridovich, I (1978)	1978	The biology of oxygen radicals. Science 201, 875-880 Non-GLP, published	N	
A6.2.1/12	Fridovich I	Fridovich, I (1983)	1983	Superoxid radical: an endogenous toxicant. Ann. Rev. Pharmacol. Toxicol. 23, 239-257 Non-GLP, published	N	
A6.2.1/13	Fuson RL et al.	Fuson, R.L.; Kylestra, J.A.; Hochstein, P. and Saltzman, H.A. (1967)	1967	Intravenous hydrogen peroxide infusion as a means of extrapulmonary oxygenation. Clin.Res. 15, 74 Non-GLP, published	N	
A6.2.1/14	Giberson TP	Giberson, T.P. (1989)	1989	Near-fatal hydrogen peroxide ingestion. Ann Emerg. Med 18, 778-779 Non-GLP, published	N	
A6.2.1/15	Góth L et al.	Goth, L.; Németh, H. and Mészáros, I (1983)	1983	Serum catalase activity for detection of haemolytic disease. Clin. Chem. 29, 741-743 Non-GLP, published	N	
A6.2.1/16	Gutteridge LMC	Gutteridge, L.M.C. (1994)	1994	Biological origin of free radicals, and mechanisms of antioxidant protection. Chem. Biol. Interact. 91, 133-140 Non-GLP, published	N	
A6.2.1/17	Halliwell B & Gutteridge JMC	Halliwell, B. and Gutteridge, J.M.C. (1984)	1984	Oxygen toxicity, oxygen radicals, transition metals and disease. Biochem. J. 219, 1-14 Non-GLP, published	N	
A6.2.1/18	Hauschild F et al.	Hauschild, F; Ludewig, R.; Mühlber, H (1958)	1958	über die "ätzende Wirkung von Wasserstoffperoxyd. Naunyn-Schmiedeberg's Arch. Exp. Pathol. Pharmak. 235, 51-62 Non-GLP, published	N	
A6.2.1/19	Hochstein PJ	Hochstein, P.J. (1988)	1988	Perspectives of hydrogen peroxide and drug-induced haemolytic anemia in glucode-6-phosphate dehydrogenase deficiency. Free Radic.Biol. Med. 5, 387-392 Non-GLP, published	N	
A6.2.1/20	Kappus H	Kappus, H. (1987)	1987	Oxidative stress in chemical toxicity. Arch. Toxicol. 60, 144-149 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.2.1/21	Kelly SA et al.	Kelly, S.A.; Havrilla, C.M.; Brady, T.C.; Abramo, K.H. and Levin, E.D. (1998)	1998	Oxidative stress in toxicology: Established mammalian and emerzing piscine model systems. Env. Health Perspect 106, 375-384 Non-GLP, published	N	
A6.2.1/22	Kinnula VL et al.	Kinnula, V.L.; Yankaskas, J.R.; Chan, L.; Virtanen, I.; Linnala, A.; Kang, B.H. and Crapo, J.D. (1994)	1994	Primary and immortlized (BEAS 2B) human bronchial epithelial cell have significant antioxidative capacity in vitro. Am. J. Respir. Cell. Mol. Biol. 11, 568-576 Non-GLP, published	N	
A6.2.1/23	Langeveld CH et al.	Langeveld, C.H.; Jongenelen, C.A.M.; Schepens, E.; Stoof, J.C.; Bast; A. and Drukarch, B. (1995)	1995	Cutured rat striatal and cortical astrocytes protect mesencephalic dopaminergic neurons against hydrogen peroxide toxicity independent of their effect on neuronal development. Neurosci. Lett. 192, 13-16 Non-GLP, published	N	
A6.2.1/24	Ludewig R	Ludewig, R (1959)	1959	Zur intraoralen Anwendung von Wasserstoffperoxyd. Z. Gesamte Exp. Med. 131, 452-465 Non-GLP, published	N	
A6.2.1/25 A6.2.2/01	Ludewig R	Ludewig, R (1964)	1964	Hydroperoxidase-Verteilung in der Haut und transepidermale Penetration von Wasserstoffperoxid nach epikutaner Applikation Acta Histochem. 19, 303-315 Non-GLP, published	N	
A6.2.1/26	Ludewig R	Ludewig, R (1965)	1965	Nachweis von 18O in Expirationslift und Blut während sublingualer Entwicklung 18O-markierten Wasserstoffperoxids. Abhandl. Deutsch. Akad. Wiss. Berlin, Kl. Chem.Geol.Biol. 7, 549-552 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.2.1/27	Madden MC et al.	Madden, M.C.; Hanley, N.; Harder, S; Velez, G. and Raymer, J.H. (1997)	1997	Increased amounts of hydrogen peroxide in the exhaled breath of ozone-exposed human subjects. Inhalation Toxicol. 9, 317-330 Non-GLP, published	N	
A6.2.1/28	Makino N et al.	Makino, N.; Mochizuki, Y; Bannai, S. and Sugita, Y. (1994)	1994	Kinteic studies on the removal of extracellular hydrogen peroxide by cultured fibroblasts. J. Biol. Chem. 269, 1020-1025 Non-GLP, published	N	
A6.2.1/29	Manohar M & Balasubramanian KA	Manohar, M. and Balasubramanian, K.A. (1986)	1986	Antioxidant enzymes in rat gastrointestinal tract. Indian J. Biochem. Biophys. 23, 274-278 Non-GLP, published	N	
A6.2.1/30	Morikawa H et al.	Morikawa, H.; Mima, H. ; Fujita, H. and Mishima, S. (1995)	1995	Oxygen embolism due to hydrogen peroxide irrigation during cervical spinal surgery. Can. J. Anaesth. 42, 231-233 Non-GLP, published	N	
A6.2.1/31	Nahum A et al.	Nahum, A.; Wood, L.D.H. and Sznajder, J.I. (1989)	1989	Measurement of hydrogen peroxide in plasma and blood. Free Radical Biol. Med. 6, 479-484 Non-GLP, published	N	
A6.2.1/32	Olanow CW	Olanow, C.W. (1993)	1993	A radical hypothesis for neurodeneration. TINS 16, 439-444 Non-GLP, published	N	
A6.2.1/33	Pietarinen P et al.	Pietarinen, P.; Raivio, K.; Devlin, R.B.; Capro, J.D.; Chang, L.Y. and Kinnula, V.L. (1995)	1995	Catalase and glutathione reductase protection of human alveolar macrophages during oxidant exposure in vitro. Am. J. Respir. Cell. Mol. Biol. 13, 434-441 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.2.1/34	Saïssy JM et al.	Saïssy, J.M.; Guignard, B.; Pats, B.; Lenoir, B. and Rouvier, B (1994)	1994	Risque de l'irrigation au peroxide d'hydrogène en chirurgie de guerrre. Ann. Fr. Anesth. Réanim. 13, 749-753 Non-GLP, published	N	
A6.2.1/35	Salahudeen AK et al.	Salahudeen, A.K.; Clark, E.C. and Nath, K.A. (1991)	1991	Hydrogen peroxide-induced renal injury. A protective role for pyruvate in vitro and in vivo. J. Clin. Invest. 88, 1886-1893 Non-GLP, published	N	
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6.2/03	[redacted]	[redacted] (2003)	2003	Degradation of peracetic acid and hydrogen peroxide in rat blood Source: [redacted] No.: A.SOL.S.031 GLP; (unpublished) Doc. No.: 514-001	Y	CEFIC, PAR TF / LoA HP TF
A6.3.3	[redacted]*	[redacted] (2002)	2002	Hydrogen peroxide: 28 day inhalation study in rats [redacted] Study Nr.: MR0211 GLP, unpublished	Y	CEFIC
A6.4.1/01	[redacted]*	[redacted] (1997)	1997	Hydrogen Peroxide – 13-week drinking water study with 6-week recovery period in C57BL/6NCrlBR mice GLP, unpublished	Y	CEFIC

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A6.4.3	Oberst FW et al.	Oberst FW, Comstock CC and Hackley EB (1954)	1954	Inhalation toxicity of ninety per cent hydrogen peroxide vapour. AMA Arch. Ind. Hyg. Occup. Med. 10, 319-327 Non-GLP, published	N	
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A6.6.1/02 A6.6.2/02 A6.6.3/02	Abu-Shakra A & Zeiger E	Abu-Shakra A and Zeiger E (1990)	1990	Effects on Salmonella genotypes and testing protocols on H_2O_2-induced mutation. Mutagenesis 5, 469-189. Non-GLP, published	N	
A6.6.1/03 A6.6.2/03 A6.6.3/03	Beales S & Suter W	Beales S and Suter W (1989)	1989	DNA repair induction by UV irradiation and various mutagens requiring metabolic activation in rat hepatocytes maintained in culture for several days. Mutagenesis 4, 456-461 Non-GLP, published	N	
A6.6.1/04 A6.6.2/04 A6.6.3/04	Bosworth D et al.	Bosworth D, Crofton-Sleight C and Venitt S (1987)	1987	A forward mutation assay using ampicillin-resistance in Eschericha coli designed for investigating the mutagenicity of biological samples. Mutagenesis 2, 455-467 Non-GLP, published	N	
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A6.6.1/53 A6.6.2/53 A6.6.3/53	Wilmer JWGM & Natarajan AT	Wilmer JWGM and Natarajan AT (1981)	1981	Induction of sister-chromatid exchanges and chromosome aberrations by gamma-irradiated nucleic acid constituents in CHO cells. Mutat. Res. 88, 99-107 Non-GLP, published	N	
A6.6.1/54 A6.6.2/54 A6.6.3/54	Winquist L et al.	Winquist L, Rannug U, Rannug A and Ramel C (1984)	1984	Protection from toxic and mutagenic effects of H_2O_2 by catalase induction in Salmonella typhimurium. <mutation research 141, 145-147 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.6.1/55 A6.6.2/55 A6.6.3/55	Zhou R et al.	Zhou R, Sun X and Tang D (1991)	1991	Iron-mediated induction of the SOS responses by hydrogen peroxide. Bull. Environ. Contam. Toxicol. 46, 613-617 Non-GLP, published	N	
A6.6.1/56 A6.6.2/56 A6.6.3/56	Ziegler-Skylakakis K & Andrae U	Ziegler-Skylakakis K and Andrae U (1987)	1987	Mutagenicity of hydrogen peroxide in V79 Chinese hamster cells. Mutat.Res.192, 65-67 Non-GLP, published	N	
A6.6.4/01	[redacted]*	[redacted] (1995b)	1995	Micronucleus test by intraperitoneal route in mice [redacted] Study number: 12240 MAS Date: 23.08.1995 GLP, unpublished	Y	CEFIC
A6.6.4/02	[redacted]*	[redacted] (1995).	1995	An evaluation of the stability and palatability of hydrogen peroxide in water and its potential geno-toxicity in bone marrow when administered to mice in drinking water; [redacted] Date: 11.09.1995 GLP, unpublished	Y	CEFIC
A6.6.5	[redacted]*	[redacted] (1997b)	1997	Hydrogen peroxide: measurement of unscheduled DNA synthesis in rat liver using in vitro and in vivo/in vitro procedures [redacted] Report No.: 514/24-1052 GLP, unpublished	Y	CEFIC
A6.7/01	[redacted]	[redacted] (1981b)	1981	Prevalence of Gastric Erosions and Duodenal Tumors with a Continuous Oral Administration of Hydrogen Peroxide in C57BL/6J Mice. Study Report. [redacted] Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.7/02	Ito A et al.*	Ito A, Watanabe H, Naito M, Naito Y and Kawashima K (1984)	1984	Correlation between induction of duodenal tumor by hydrogen peroxide and catalase activity in mice. Gann 75, 17-21 Non-GLP, published	N	
A6.7/03	[redacted]	[redacted] (1980)	1980	Report on a Carcinogenicity Study. [redacted] Non-GLP, published	N	
A6.7/04	Ito A et al.	Ito A, Naito M and Watanabe H et al. (1981a)	1981	Implication of chemical carcinogenesis in the experimental animal. [Japanese, English translation]. Ann. Rep. of Hiroshima Univ. Res. Inst. Nuclear Medicine and Biology 22, 147-158 Non-GLP, published	N	
A6.7/05	Bock FG et al.	Bock FG, Myers HK and Fox HW (1975)	1975	Cocarcinogenic activity of peroxy compounds. J. Natl. Cancer Inst. 55, 1359-1361 Non-GLP, published	N	
A6.7/06	Ito A et al.	Ito A, Naito Y and Watanabe H (1982)	1982	Induction and characterisation of gastro-duodenal lesions in mice given continuous oral administration of hydrogen peroxide. Gann 73, 315-322 Non-GLP, published	N	
A6.7/07	Hankin L	Hankin L (1958)	1958	Hydrogen peroxide, ingestion and the growth of rats. Nature 181, 1453 Non-GLP, published	N	
A6.7/08	Klein-Szanto AJP & Slaga TJ	Klein-Szanto AJP and Slaga TJ (1982)	1982	Effects of hydrogen peroxide on rodent skin: Epidermal hyperplasia and tumor promotion. J. Invest. Dermatol. 79, 30-34 Non-GLP, published	N	
A6.7/09	Kurokawa Y et al.	Kurokawa Y, Takamura N, Matsushima Y, Imazawa T and Hayashi Y (1984)	1984	Studies on the promoting and complete carcinogenic activities of some oxidising in skin carcinogenesis. Cancer Lett. 24, 299-304 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.7/10	Marshall MV et al.	Marshall MV, Kuhn JO, Torrey CF, Fischman SL and Cancro LP (1996)	1996	Hamster cheek pouch bioassay of dentrifrices containing hydrogen peroxide and baking soda. J. Am. Coll. Toxicol. 15, 45-61 Non-GLP, published	N	
A6.7/11	Takahashi M et al.	Takahashi M, Hasegawa R, Furukawa F, Toyoda K, Sato H and Hayashi Y (1986)	1986	Effects of ethanol, potassium metabisulphite, formaldehyde and hydrogen peroxide on gastric carcinogenesis in rats after initiation with N-methyl-N′-nitro-N-nitrosoguanidine. Jpn. J. Cancer Res. (Gann) 77, 118-124 Non-GLP, published	N	
A6.7/12	Hirota N & Yokoyama T	Hirota N and Yokoyama T (1981)	1981	Enhancing effect of hydrogen peroxide upon duodenal and upper jejunal carcinogenesis in rats. Gann. 72, 811-812 Non-GLP, published	N	
A6.7/13	Weitzman SA et al.	Weitzman SA, Weitberg AB, Stossel TP, Schwartz J and Shklar G (1986)	1986	Effects of hydrogen peroxide on oral carcinogenesis in hamsters. J. Periodontal. 57, 685-688 Non-GLP, published	N	
A6.8/01	Moriyama I et al.	Moriyama I, Hiraoka K, Fujita M, Ichijo M and Ioka H (1982)	1982	Effects of food additive hydrogen peroxide studied in fetal development [Japanese, English translation]. Acta Obst. Gynaec. Japan 34, 2149-2154 Non-GLP, published	N	
A6.8/02	Wales AG et al.	Wales AG, White IG and Lamond DR (1959)	1959	The spermicidal activity of hydrogen peroxide in vitro and in vivo. J. Endocrin. 18, 236-244 Non-GLP, published	N	
A6.12.1/01 A6.12.5/04	Kaelin RM et al.	Kaelin RM, Kapanci Y and Tchopp JM (1988)	1988	Diffuse interstitial lung disease associated with hydrogen peroxide inhalation in a dairy worker. Ann. Rev. Respiratory disease 137, 1233-1235 Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.12.1/02 A6.12.5/05	Kondrashov VA	Kondrashov, V.A. (1977)	1977	Evaluation of the toxicity of hydrogen operoxide vapors for inhalation and percutaneous exposure [Russian, English translation] Gig. Tr. Zabol. 21, 22-25 Non-GLP, published	N	
A6.12.2/05	Grant WM	Grant WM (1993)	1993	Toxicology of the eye. 4th ed. Thomas Springfield, IL Non-GLP, published	N	
A6.12.2/06	Knopf HLS	Knopf HLS (1984)	1984	Amer. J. Ophthalmol. 796 Non-GLP, published	N	
A6.12.2/07	Paugh RJ et al.	Paugh JR, Brennan NA and Efron N (1988)	1988	Ocular response to hydrogen peroxide. Am. J. Optom. And Physiol. Oprtzics 65, 91-98. Non-GLP, published	N	
A6.12.2/01	Christensen DW et al.	Christensen DW, Faught WE, Black RE, Woodward GA and Timmons OD (1992)	1992	Fatal oxygen embolization after hydrogen peroxide ingestion. Crit. Care. Med. 20, 543-544 Non-GLP, published	N	
A6.12.2/02	Luu TA et al.	Luu TA, Kelley MT, Strauch JA and Avradopoulos K (1992)	1992	Portal vein gas embolism from hydrogen peroxide ingestion. Ann Emerg. Med. 21, 1391-1393 Non-GLP, published	N	
A6.12.2/03	Dickson KF & Caravati EM	Dickson KF and Caravati EM (1994)	1994	Hydrogen peroxide exposure – 325 exposures reported to a regional poison control center. Clinical Toxicology 32, 705-714 Non-GLP, published	N	
A6.12.2/04	Cannon G et al.	-	2003	Hydrogen peroxide neurotoxicity in childhood: Case report with unique magnetic resonance imagining features. Journal of Child Neurology, 18, No. 11, pg 805-808. Non-GLP, published	N	

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A6.12.3/01	[redacted]	[redacted] (1999)	1999	Draft report of hydrogen peroxide production workers health survey. Summary of workers health data of 4 production sites for hydrogen peroxide of [redacted] Non-GLP, unpublished	Y	Degussa
A6.12.3/02	Riihimäki V et al.	Riihimäki V, Toppila A, Piirilä P, Kuosma E, Pfäffli P and Tuomela P (2002)	2002	Respiratory health in aseptic packaging with hydrogen peroxide. A report of two cases. J. Occup. Health 44, 433-438 Non-GLP, published	N	
A6.12.5/01 A6.12.7/01	Ellenhorn MJ et al.	-	1997	Ellenhorn's Medical Toxicology: diagnosis and treatment of human poisoning – 2nd ed., Williams & Wilkins, 1997, pg 1222-1223. Non-GLP, published	N	
A6.12.5/02 A6.12.7/02	Goldfrank LR et al.	-	1994	Goldfrank's Toxicologic Emergencies, 5th ed.; Appleton & Lange, 1994, pg 1089. Non-GLP, published	N	
A6.14.1	Ernstgård, L. et al.	Ernstgård, L.; Sjögren, B.; Johanson G (2012)		Acute effects of exposure to hydrogen peroxide in humans. Toxicol Lett. 2012 Jul 20;212(2):222-7. published	N	
A12.2	Mc Nally JJ	Mc Nally JJ (1990)	1990	Cilinical aspects of topical application of dilute hydrogen peroxide solution. The CLAO journal 16, S46-S51 Non-GLP, published	N	
II A Sec 3.5	Ball GL et al	Ball G.L, English J.C., and C J McLellan C.J. (2011)	2011	Health Risk Assessment for Hydrogen Peroxide to Determine Acceptable Drinking, poster, NSF International, Ann Arbor, MI, USA, published.	N	
IIA Sec 3.10	Mastrangelo G et al.	Mastrangelo G, Zanibellato R, Fedeli U, Fadda E, Lange JH (2005)	2005	Exposure to Hydrogen Peroxide at TLV Level does not induce Lung Function Changes: A Longitudinal Study. Int J Environ Health Res Aug; 15(4):313-7, published	N	

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IIA Sec 3.10	Mastrangelo G et al.	Mastrangelo G, Zanibellato R, Fadda E, Lange JH, Scoizzato L, Rylander R (2009)	2009	Exposure to Hydrogen Peroxide and Eye and Nose Symptoms Among Workers in a Beverage Processing Plant. Ann Occup Hyg 53(2): 161-165 , published	N	
IIA Sec 3.6.2	Gaivão I and Sierra LM	Gaivão I and Sierra LM (2014)	2014	Drosophila comet assay: insights, uses, and future perspectives. Front Genet. 2014 Aug 29;5:304. Published	N	
IIA Sec 3.6.2	Møller P et al	Møller P, Jacobsen NR, Folkmann JK, Danielsen PH, Mikkelsen L, Hemmingsen JG, Vesterdal LK, Forchhammer L, Wallin H, and Loft S (2010)	2010	Free Radical Research, January 2010, Vol. 44, No. 1 : Pages 1-46 Published	N	
IIA Sec 3.6.2	Møller P et al	Møller P, Danielsen PH, Karottki DG, Jantzen K, Roursgaard M, Klingberg H, Jensen DM, Christophersen DV, Hemmingsen JG, Cao Y, Loft S. (2014)	2014	Oxidative stress and inflammation generated DNA damage by exposure to air pollution particles. Mutat Res Rev Mutat Res. 2014 October - December;762C:133-166. doi: 10.1016/j.mrrev.2014.09.001. Epub 2014 Sep 16. Published	N	

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IIA Sec 3.6.2	Shukla AK et al	Shukla AK, Pragya P, Chowdhuri DK (2011)		A modified alkaline Comet assay for in vivo detection of oxidative DNA damage in Drosophila melanogaster. Mutat Res. 2011 Dec 24;726(2):222-6. Published	N	
IIA Sec 3.6.2	Ye ZW et al	Ye ZW, Zhang J, Townsend DM, Tew KD (2014)		Oxidative stress, redox regulation and diseases of cellular differentiation. Biochim Biophys Acta. 2014 Nov 15. pii: S0304-4165(14)00387-0. doi: 10.1016/j.bbagen.2014.11.010. [Epub ahead of print] . Published	N	
IIA Sec 3.6.2			2014	http://www.amsbio.com/protocols/comet_assay.pdf /23.12.2014	N	
Doc II B Appendix 1	ECETOX	CEFIC (1997e)	1997	Hydrogen peroxide, risk assessment/comprehensive report (Draft 3), Appendix D, ECETOX update report on OEL. CEFIC Peroxygen Sector Group. Risk Assessment Working Group (1997) Non-GLP, published	N	
Doc IIB Appendix 1	[redacted]	[redacted] (1997e)	1997	Hydrogen peroxide, Risk Assessment/Comprehensive Report (Draft 3), Appendix D, ECETOC Update Report on OEL. [redacted] Non-GLP, unpublished	Y	CEFIC

Ecotoxicological studies

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A7.1.1.2.1/02	Larish BC & Duff SJB	Larish and Duff, 1997	1997	Effect of H2O2 on characteristics and biological treatment of TCF bleached pulp mill effluent. Wat. Res. 31, 1694-1700 Non-GLP, published	N	
A7.1.1.2.4	Spain JC, Milligan JD, Downey DC & Slaughter JK	Spain et al., 1989	1989	Excessive bacterial decomposition of H2O2 during enhanced biodegradation. Groundwater 27, 163-167 Non-GLP, published	N	
A7.1.2.2.1/01	Cooper JW & Lean DRS	Cooper and Lean, 1989a	1989	Environmental Science and Technology 23, 1425-1428. Non-GLP, published	N	
A7.1.2.2.1/02	Cooper WJ, Lean DRS & Carey JH	Cooper, et al., 1989	1989	Spatial and temporal patterns of hydrogen peroxide in lake Waters. Can. J. Fish. Aquat. Sci. 46, 1227-1231. Non-GLP, published	N	
A7.1.2.2.1/03	Sturzenegger VT	Sturzen-egger, 1989	1989	Wasserstoffperoxid in Oberflächengewässern: Photochemishe Production und Abbau. Dissertation an der Eidgenössischen Technischen Hochschule, Zurich Non-GLP, published	N	
A7.1.2.2.1/04	Air Liquide	Air Liquide, 1991	1991	Evolution de la Concentration en Peroxyde d'Hydrogène dans des Echantillons Tirés de la Saône Non-GLP, unpublished	N	Air Liquide
A7.1.2.2.1/05	Herrmann R & Herrmann K	Herrmann and Herrmann, 1994	1994	Chemodynamics of hydrogen peroxide in shallow lagoons on theMediterranean coast. Aqua Fennica 24, 3-8 Non-GLP, published	N	
A7.1.2.2.1/06	Petasne RG & Zika RG	Petasne and Zika, 1987	1987	Fate of superoxide in coastal sea water. Nature 325, 516-518. Non-GLP, published	N	

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A7.2.1/01	Aggarwal et al.	Aggarwal et al., 1991	1991	Use of hydrogen peroxide as an oxygen source for in situ biodegradation, Part II Laboratory studies. Journal of Hazardous Materials 27, 301-314. Non-GLP, published	N	
A7.2.1/02	ECETOC	ECETOC 1993	1993	Joint Assessment of Commodity Chemicals No. 22, Hydrogen Peroxide. ECETOC, Brussels. Non-GLP, published	N	
A7.2.1/03	Pardieck DL, Bouwer EJ & Stone AT	Pardieck et al., 1992	1992	Hydrogen peroxide use to increase oxidant capacity for in situ bioremediation of contaminated soils and aquifers: A review. Journal of Contaminant Hydrology 9, 221-242 Non-GLP, published	N	
A7.3.1/01	Olszyna KJ, Meagher JF & Bailey EM	Olszyna, 1988	1988	Gas-phase, cloud and rain-water measurements of hydrogen peroxide at a high-elevation site. Atmospheric Environment 22, 1699-1706 Non-GLP, published	N	
A7.3.1/02	Sakugawa H, Kaplan IR, Tsai W & Cohen Y	Sakugawa et al., 1990	1990	Atmospheric hydrogen peroxide. Environmental Science and Technology 24, 1452-1462 Non-GLP, published	N	
A7.3.1/03	Kleinman LIJ	Kleinman 1986	1986	Elf Atochem Unpublished "Hydrogen Peroxide Risk Assesment, Atmospheric Part", as cited in Geophys. Res. 10889-10904. Non-GLP, published	N	
A7.4.1.1/01	[redacted]*	[redacted] (1989a)	1989a	Interox America Sodium Percarbonate and Hydrogen Peroxide - Acute toxicity to the freshwater fish Pimephales promelas. [redacted] Report no. not given Non-GLP, unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A7.4.1.1/02	[redacted]	[redacted] (1977b)	1977	Vorversuche zum Fischtest – Ermittlung der kritischen Konzentration an H2O2 in Wasser. [redacted] Report no. 2122 Non-GLP, unpublished	Y	CEFIC
A7.4.1.1/03 A7.4.1.2/03	Kay SH et al.	Kay et al. (1982)	1982	H2O2: A potential algicide for aquaculture. Proc. South Weed Sci Soc, Vol 35, ISS New Perspect. Weed Sci Non-GLP, published	N	
A7.4.1.2/01	[redacted]*	[redacted] (1989b)	1989b	Interox America Sodium Percarbonate and Hydrogen Peroxide - Acute toxicity to the freshwater invertebrate Daphnia pulex. [redacted] Report no. not given Non-GLP, unpublished	Y	CEFIC
A7.4.1.2/02	Bringmann G & Kühn R	Bringmann and Kuhn (1982)	1982	Ergebnisse der Schadwirkung wassergefährdender Stoffe gegen Daphnia magna in einem weiterentwickelten standardisierten Testverfahren. Z. Wasser Abwasser Forsch., 15 Nr. 1, 1-6 Non-GLP, published	N	
A7.4.1.3/01	Knight et al.*	Knight et al. (1995)	1997	Hydrogen peroxide as Paramove – Marine alga, growth inhibition test (72 h, EC_{50}). Inveresk Research, Tranent, Scotland, Report no. 10913 GLP, unpublished	Y	CEFIC
A7.4.1.3/02	Walzer & Lotz A*	Degussa (1991)	1982	Chlorella vulgaris – Algenwachstumstest mit Wasserstoffperoxid 35% G. Degussa, Geschäftsbereich Industrie- und Feinchemikalien, Anwendungstechnik, Germany, Report no. 91/11/01 Non-GLP, unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
A7.4.1.3/03	Kavanagh NA	Kavanagh (1992)	1992	Hydrogen peroxide as a growth inhibitor for blue-green algae. Solvay Interox, 11-83-92 Non-GLP, unpublished	Y	CEFIC
A7.4.1.3/04	Barroin G & Feuillade M	Barroin and Feuillade (1986)	1986	Hydrogen peroxide as a potential algicide for Oscillatoria rubescens D.C. Wat. Res., 20 (5), 619-623 Non-GLP, published	N	
A7.4.1.3/05	Clarke CA	Clarke (1991)	1991	The anti-algal activity of peroxygen compounds. Ph.D. Thesis, University of Bath Non-GLP, unpublished	Y	CEFIC
A7.4.1.3/06	Florence TM & Stauber JL	Florence and Stauber (1986)	1986	Toxicity of copper complexes to the marine diatom Nitzschia closterium. Aquatic Toxicology, 8, 11-26 Non-GLP, published	N	
A7.4.1.4/01 A7.1.1.2.1/01	Groeneveld AHC & de Groot WA*	Groeneveld and de Groot (1999)	1999	Activated sludge, respiration inhibition test with hydrogen peroxide. Solvay Pharmaceuticals, Weesp, The Netherlands, Report no. A.SOL.S.003 GLP, unpublished	Y	CEFIC
A7.4.1.4/02	Knie J et al.	Knie et al. (1983)	1983	Ergebnisse der Untersuchungen von chemischen Stoffen mit vier Biotests. Deutsche Gewässerkundliche Mitteilungen., 27, Heft 3, 77-79 Non-GLP, published	N	
A7.4.1.4/03	Baldry MGC	Baldry (1983)	1983	The bacterial, fungicidal and sporicidal properties of hydrogen peroxide and peracetic acid. Journal of Applied Bacteriology, 54, 417-423 Non-GLP, published	N	
A7.4.3	Xenopoulos MA & Bird DF	Xenopoulos and Bird (1997)	1997	Effect of acute exposure to hydrogen peroxide on the production of phytoplankton and bacterioplankton in a mesohumic lake. Photochemistry and Photobiology, 66 (4), 471-478 Non-GLP, published	N	

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A7.4.3.1	Kelly JD et al.	Kelly et al. (1992)	1992	Dietary Hydrogen peroxide enhances hepatocarcinogenesis in trout: correlation with 8-hydroxy-2′-deoxyguanosine levels in liver DNA. Carcinogenesis, 13, 1639-1642 Non-GLP, published	N	
A7.4.3.4	Meinertz JR, Greseth SL, Gaikowski MP and Schmidt LJ	None	2008	Chronic toxicity of hydrogen peroxide to Daphnia magna in a continuous exposure, flow-through test system US Geological Survey, La Crosse, Wisconsin, USA Science of the Total Environment 392 (2008), pp. 225-232 Non-GLP, published	N	
A7.4.3.4	Klerks PL & Fraleigh PC	Klerks and Fraleigh (1991)	1991	Controlling adult zebra mussels with oxidants. Journal AWWA, 92-100 Non-GLP, published	N	

Product 35 %

B3 Physical chemical properties

Note: An additional column was introduced documenting the citation of each study as used in the EU Risk-Assessment Report, for ease of comparison.

CEFIC - CEFIC Peroxygens Sector Group, Hydrogen peroxide subgroup
* - Key study

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B3.1	Badt-Tognucci A.*	not applicable	2007	Determination of Color, Physical State and Odor of Hydrogen peroxide Peroxal 35DS. RCC Ltd. Study No.B07920 GLP unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
B3.5	Badt-Tognucci A.*	not applicable	2007	pH-Determination and Determination of the Free Acidity or Alkalinity of Hydrogen peroxide Peroxal 35DS. RCC Ltd. Study No.B07907 GLP unpublished	Y	CEFIC
B3.6	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 445 Non-GLP, published	N	
B3.10.1	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation (ed), New York, 206 Non-GLP, published	N	
B3.10.2	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation (ed), New York, 202 and 203 Non-GLP, published	N	

Product 49.9 %

* - Key study

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B3.1	Badt-Tognucci A.*	not applicable	2007	Determination of Color, Physical State and Odor of Hydrogen peroxide Peroxal 50DS. RCC Ltd. Study No.B07885 GLP unpublished	Y	[redacted]
B3.5	Badt-Tognucci A.*	not applicable	2007	pH-Determination and Determination of the Free Acidity or Alkalinity of Hydrogen peroxide Peroxal 50DS. RCC Ltd. Study No.B07863 GLP unpublished	Y	[redacted]
B3.6	Goor G. et al. *	Goor et al. (1989)	1989	Hydrogen peroxide, In: Ullmann´s Encyclopedia of Industrial Chemistry, 5th completely revised edition, 1989, Vol. A 13. Elvers B., Hawkins S., Ravenscroft M. and Schulz G. (eds), VCH, Weinheim, 445 Non-GLP, published	N	
B3.10.1	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation (ed), New York, 206 Non-GLP, published d	N	
B3.10.2	Schumb W., et al. *	Schumb et al. (1955)	1955	Hydrogen peroxide, American Chemical Society Monograph Series, 1955, Reinhold Publishing Corporation (ed), New York, 202 and 203 Non-GLP, published	N	

Analytical methods

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
B4.1/01	CEFIC*	None	2003	Hydrogen peroxide for industrial use. Determination of hydrogen peroxide content. Titrimetric method. Method no. AM-7157. CEFIC Peroxygens Sector Group, Brussels, March 2003. http://www.cefic.org/Templates/shwAssocDetails.asp?NID=473&HID=27&ID=66 Non-GLP, published	N	
B4.1/02	Crommelynck F*	None	1993	Peroxyde d'hydrogène à usage industriel. Détermination de la teneur en peroxyde d'hydrogène. Methode titrimetrique. Chemoxal/Chalon. Controle qualité. 3 December 1993. Non-GLP, unpublished	Y	CEFIC
B4.1/03	Degussa AG*	None	2005	Analytical method for H_2O_2: Determination of chloride, phosphate, sulphate and nitrate. Degussa AG Dept. O2-AO-AT. January 2005 Non-GLP, unpublished	Y	CEFIC

Effectiveness against target organisms and intended uses

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
B5.10/01	Crane, E.*	None	2007a	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 1040 (2005). Chemical disinfectants and antiseptics – Basic bactericidal activity (Phase 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/02	Crane, E.*	None	2007 b	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 1275 (2005). Chemical disinfectants and antiseptics – Basic fungicidal activity (Phase 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/03	Crane, E.*	None	2007c	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 14347 (2005). Chemical disinfectants and antiseptics – Basic sporicidal activity (Phase 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/04	Crane, E.*	None	2007 d	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 1276 (1997). Quantitative suspension test for the evaluation of bactericidal activity of chemical disinfectants and antiseptics (Phase 2/ Step 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC

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B5.10/05	Crane, E.*	None	2007e	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 1650 (1998). Quantitative suspension test for the evaluation of fungicidal activity of chemical disinfectants and antiseptics (Phase 2/Step 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/06	Crane, E.*	None	2007f	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 13704 (2002). Quantitative suspension test for the evaluation of sporicidal activity of chemical disinfectants and antiseptics (Phase 2/Step 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/07	Woodall, C.*	None	2007	EN 14476: 2005: Chemical disinfectants and antiseptics – Virucidal quantitative suspension test for chemical disinfectants and antiseptics used in human medicine-test method and requirements (phase 2/step 1). BlueScientific Test Data. Report No: not reported Non-GLP, unpublished	Y	CEFIC
B5.10/08	Crane, E.*	None	2007 g	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 13697 (2001). Quantitative non-porous surface test for the evaluation of bactericidal and/or fungicidal activity of chemical disinfectants (Phase 2 / Step 2). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
B5.10/09	Crane, E.*	None	2007h	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 1656 (2000). Chemical disinfectants and antiseptics – Quantitative suspension test for the evaluation of bactericidal activity of chemical disinfectants and antiseptics used in the veterinary field (Phase 2/ Step 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/10	Crane, E.*	None	2007i	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 1657 (2005). Chemical disinfectants and antiseptics – Quantitative suspension test for the evaluation of fungicidal and yeasticidal activity of chemical disinfectants and antiseptics used in the veterinary field (Phase 2 / Step 1). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/11	Crane, E.*	None	2007j	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 14349 (2004). Chemical disinfectants and antiseptics – Quantitative surface test for the evaluation of bacterial activity of chemical disinfectants and antiseptics used in the veterinary field on non-porous surface without mechanical action (Phase 2 / Step 2). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC

Section no. / Reference no.	Author(s)	Citation in style of EU-Risk Assessment Report	Year	Title. Source (where different from company) Company, Report no. GLP (where relevant) / (un)published	Data Protection Claimed (Yes/No)	Owner
B5.10/12	Crane, E.*	None	2007k	INTEROX ST 50 Hydrogen Peroxid Solution – BS EN 14349 (2004). Chemical disinfectants and antiseptics – Quantitative surface test for the evaluation of bacterial activity of chemical disinfectants and antiseptics used in the veterinary field on non-porous surface without mechanical action (Phase 2 / Step 2). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/13	Crane, E.*	None	2007l	INTEROX ST 50 Hydrogen Peroxid Solution – ASTM 723 (1997). Efficacy of antimicrobials as preservatives for aqueous-based products used in the paper industry (Phase 2 / Step 2). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC
B5.10/14	Crane, E.*	None	2007m	INTEROX ST 50 Hydrogen Peroxid Solution – ASTM 875 (2000). Efficacy of antifungals as preservatives for aqueous-based products used in the paper industry (Phase 2/ Step 2). MGS Laboratories Ltd. MGS-No.: 12319/ SO No.: 1037 Non-GLP, unpublished	Y	CEFIC

Appendix IV: Tables of risk characterization for human health

Intended use (PT)	Exposure scenario (H_2O_2 concentration)	Method for exposure assessment	Inhalation exposure, concentration in air (mg/m^3)	Exposure/ $AEC_{inhalation}$ (1.25 mg/m^3)		PPE
PT 1 Disinfection of human skin	mixing and loading (professionals, once a month) (35%/**49.9%)**	a) TNsG m&l model 7 for inhalation; m&l model 4 for dermal b) ART	a) 0.47 b) 0.27	a) 38% b) 22%		gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	skin application (regular use 25x1min day) (a) **7.4%** b) **4.9%**)	ConsExpo (constant rate release)	a) 1.04 (as 8-h TWA) 1.83/event b) 0.69 (as 8-h TWA) 1.21 (event)	a) 83% 146% b) 55% 97%		-
PT 2.01 Surface disinfection by VHP process	evaporation (max 3x/day for 200days/y) (**35%** (350-560 mg/m^3 in the sealed space)	- (no exposure, sealed space)	1.4 mg/m^3 is used as a worst case estimate.	Tier 1	112%	RPE in re-entry if the concentration over AEC
				Tier 2 RPE with APF=5	22%	
PT 3 Disinfection of animal housing	mixing and loading (1 or 0.5 h; professional operators: max 200days/y; trained farmers: max 10 operations/y) (35%/**49.9%**)	a) US-EPA PHED surrogate exposure guide for inhalation; TNsG Loading DEGBE model in BEAT for dermal b) ART	a) 0.26-0.5 b) 3.9	Tier 1	a) 40% b) 312%	gloves, coverall, goggles/face shield, RPE
				Tier 2 RPE with APF=5	b) 78%	
	spraying (professional operators: max 200days/y, 120–400 min/operation; trained farmers 6-10 operations/y, 400min/operation) (**7.4%**)	a) TNsG spraying model 2 b) ConsExpo (exposure to spray) c) ART	a) 5.6 b) 0.4 c) 4.0	Tier 1	a) 448% b) 32% c) 320%	gloves, coverall, goggles/face shield, RPE
				Tier 2 RPE with APF=5	a) 90% c) 64%	
PT 4 Aseptic packaging	loading (**35%**)	-	incidental	-		gloves, coverall, goggles/face shield, RPE if insufficient ventilation or

Intended use (PT)	**Exposure scenario (H_2O_2 concentration)**	**Method for exposure assessment**	**Inhalation exposure, concentration in air (mg/m^3)**	**Exposure/ $AEC_{inhalation}$ (1.25 mg/m^3)**		**PPE**
						no LEV
	application (around machines)	measured	0.14-0.7	56%		-
	maintenance work	measured	0.7-1.4	Tier 1	112%	gloves, coverall, goggles/face shield, RPE
				Tier 2 RPE with APF=5	22%	
PT 4 Surface disinfection by VHP process (same as in PT 2)	evaporation (max 3x/day for 200days/y) (**35%** (350-560 mg/m^3 in the sealed space)	- (no exposure, sealed space)	1.4 mg/m^3 is used as a worst case estimate	Tier 1	112%	RPE in re-entry if the concentration over AEC
				Tier 2 RPE with APF=5	22%	

Intended use (PT)	Exposure scenario (H_2O_2 concentration)	Method for exposure assessment	Inhalation exposure, concentration in air (mg/m^3)	Exposure/ $AEC_{inhalation}$ (1.25 mg/m^3)		PPE
PT 4 Disinfection of distribution systems for drinking water	mixing and loading (20 min on 1 to 2 days/wk) (35%/**49.9%**)	a) TNsG m&l model 7 b) ART	a) 0.47 b) 0.43	a) 38% b) 34%		gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	application (0.5-2h/day, max 200days/year) (**4%**)	a) TNsG spraying model 2 b) ART	a) 3.04 b) 7.1	Tier 1	a) 243% b) 568%	gloves, coverall, goggles/face shield, RPE
				Tier 2 RPE with APF=10	a) 24% b) 57%	
PT 5 Disinfection of water	loading (20 minutes, 10-100 days/y) (35%/**49.9%**)	a) TNsG m&l model 7 b) ART	a) 0.47 b) 0.19	a) 38% b) 15%		gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	application (25 mg/l)	automated process	none/very low	-		-
PT 6.02 In-can preservatives of paper additives	mixing and loading (35%/**49.9%**)	ART	0.23	18%		gloves, coverall, goggles/face shield, RPE if insufficient ventilation or no LEV
	application (evaporation, exposure times short in automated continuous processes) (1%)	measured	max. 0.7	56%		gloves, coverall, goggles/face shield, RPE in maintenance work

Efficiency of RPE expressed as a Assigned Protection Factor, APF, e.g. if APF=20, it means 5% penetration.
For acceptability the Exposure/$AEC_{inhalation}$ ratio must be $\leq$100%