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HYDROGEN PEROXIDE

CAS No: 7722-84-1

EINECS No: 231-765-0

Summary Risk Assessment Report

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SUMMARY RISK ASSESSMENT REPORT

Final report, 2003

Finland

The rapporteur for the risk assessment report on hydrogen peroxide is the Finnish Environment Institute, in co-operation with the National Product Control Agency for Welfare and Health and in consultation with the Ministry of Social Affairs and Health, Occupational Safety and Health Department.

The scientific work concerning the human health has been prepared by the Finnish Institute of Occupational Health.

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(The last full literature survey was carried out in 1997 - targeted searches were carried out subsequently).

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PREFACE

This report provides a summary, with conclusions, of the risk assessment report of the substance hydrogen peroxide that has been prepared by Finland in the context of Council Regulation (EEC) No. 793/93 on the evaluation and control of existing substances.

For detailed information on the risk assessment principles and procedures followed, the underlying data and the literature references the reader is referred to the comprehensive Final Risk Assessment Report (Final RAR) that can be obtained from the European Chemicals Bureau¹. The Final RAR should be used for citation purposes rather than this present Summary Report.

¹ European Chemicals Bureau – Existing Chemicals – <http://ecb.jrc.it>

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1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS Number: 7722-84-1
EINECS Number: 231-765-0
IUPAC Name: hydrogen peroxide
Synonyms: dihydrogen dioxide, hydrogen dioxide
Molecular weight: 34.02 g/mol
Molecular formula: H₂O₂
Structural formula: H - O - O - H

1.2 PURITY/IMPURITIES, ADDITIVES

Purity: > 99% w/w
Impurities:
Residue on evaporation <= 0.006% w/w
Total concentration of inorganic impurities <= 0.001% w/w
Organic impurities (TOC) 0.005-0.1% w/w

Additives:

Stabilisers

CAS-No	EINECS-No	IUPAC-Name	Value
7664-38-2	231-633-2	phosphoric acid	10-300 mg/l
10049-21-5	--	sodium phosphate	10-300 mg/l
12209-98-2	--	sodium stannate	10-300 mg/l
7783-20-2	231-984-1	ammonium sulphate	10-300 mg/l
1344-09-8	215-687-4	sodium silicate	..
103-84-4	203-150-7	acetanilide	..
1127-45-3	214-430-3	8-hydroxyquinoline	..
		pyridine carboxylic acids	..
147-71-7	205-695-6	tartaric acid	..
65-85-0	200-618-2	benzoic acids	..

Passivators: nitrate salts .. % w/w

1.3 PHYSICO-CHEMICAL PROPERTIES

Table 1.1 Physical and chemical properties of pure hydrogen peroxide (100%)

Property	Value
Melting point	-0.40 - 0.43°C
Boiling point	150-152°C decomposition
Density	1.4425 g/cm ³ (25°C)
Vapour pressure	3 hPa (25°C)
Water solubility	miscible in all proportions
Log Kow	-1.5 (calculated)
pK _a	11.62 (25°C)
Henry's law constant	7.5 · 10 ⁻⁴ Pa m ³ /mol (20°C) measured

Table 1.2 Physical and chemical properties of hydrogen peroxide-water solutions

H ₂ O ₂ %	35% w/w	50% w/w	70% w/w	90% w/w
Melting point	-33°C	-52°C	-40°C	-11°C
Boiling point	108°C	114°C	125°C	141°C
Density (25°C)	1.1282	1.1914	1.2839	1.3867
Vapour pressure (partial)	0.48 hPa (30°C)	0.99 hPa (30°C)	2 hPa (30°C)	
Vapour pressure (total)		24 hPa (30°C)	14.7 hPa (30°C)	6.7 hPa (30°C)
Saturated vapour concentration at 25 °C (mg/m ³)		787	1,685	3,049
Surface tension mN/m (20 °C)	74.6	75.7	77.3	79.2
Viscosity (1 · 10 ⁻³ kg/ms)	1.11	1.17	1.24	1.26

At atmospheric pressure vapours containing 26 mol % or more hydrogen peroxide can be exploded by a spark, by contact with catalytically active materials initially at room temperature, or by “non-catalytic” materials at elevated temperatures. Because of the high relative volatility of water to hydrogen peroxide, the danger of vapour phase explosion on storage of liquid hydrogen peroxide will be encountered only with concentrated solutions above 74% wt at elevated temperatures. At concentrations above 86% wt the liquid itself can be made to explode.

1.4 CLASSIFICATION

1.4.1 Current classification

Classification according to Annex I of Directive 67/548/EEC:

Hydrogen peroxide solution ...%

Classification O; R8
C; R34

Labelling O; C
R: 8
S: (1/2-)3-28-36/39-45

R8: Contact with combustible material may cause fire
R34: Causes burns
S1/2: Keep locked up and out of the reach of children
S3: Keep in a cool place
S28: After contact with skin, wash immediately with plenty of... (to be specified by the manufacturer)
S36/39: Wear suitable protective clothing and eye/face protection
S45: In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible)

Nota B

Specific concentration limits

$C \geq 20\%$: C; R34
 $5\% \leq C < 20\%$: Xi; R36/38

Footnote

$C \geq 60$: O; R8

1.4.2 Proposed classification

The classification and labelling of hydrogen peroxide has been agreed at technical levels (Status 18.11.2002, Rev. 11) to be listed in Annex I to Directive 67/548/EEC following the adoption of the 29th Adaptation to Technical Progress, as follows:

Hydrogen peroxide solution ...%

Classification R5
O; R8
C; R35
Xn; R20/22

Labelling O; C
R: 5-8-20/22-35
S: (1/2-)17-26-28-36/37/39-45

R5: Heating may cause an explosion

R8:	Contact with combustible material may cause fire
R35:	Causes severe burns
R20/22:	Harmful by inhalation/and if swallowed
S1/2:	Keep locked up and out of the reach of children
S17:	Keep away from combustible material
S26:	In case of contact with eyes, rinse immediately with plenty of water and seek medical advice
S28:	After contact with skin, wash immediately with plenty of... (to be specified by the manufacturer)
S36/37/39:	Wear suitable protective clothing, gloves and eye/face protection
S45:	In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible)

Nota B

Specific concentration limits

$C \geq 70\%$:	C; R20/22-35
$50\% \leq C < 70\%$:	C; R20/22-34
$35\% \leq C < 50\%$:	Xn; R22-37/38-41
$8\% \leq C < 35\%$:	Xn; R22-41
$5\% \leq C < 8\%$:	Xi; R36

Footnote

$C \geq 70\%$:	R5, O; R8
$50\% \leq C < 70\%$:	O; R8;
R22:	Harmful if swallowed
R34:	Causes burns
R36:	Irritating to eyes
R37/38:	Irritating to respiratory system and skin
R41:	Risk of serious damage to eyes

Environment

No classification.

2

GENERAL INFORMATION ON EXPOSURE

There are 22 plants producing hydrogen peroxide in the European Union in 1997. One plant has been stopped in 1995, but it has been included in the assessment. The total production volume of hydrogen peroxide in Europe has increased rapidly being 600,000 tonnes in 1993 and 750,000 tonnes in 1995 calculated as 100% H₂O₂. In 1995 about 80,000 tonnes were exported outside Europe.

Hydrogen peroxide is mainly used for pulp bleaching (48%) and manufacture of other chemicals (38%) such as sodium perborate, percarbonate and peracetic acid. Remaining 15% of the total volume consumed in Europe is used for different applications including textile bleaching, environmental applications, metal etching, sanitisation of chemical instruments and surfaces, metal semiconductor chips manufacturing, disinfection of drinking water, disinfectant in aseptic packaging and bleaching of certain foodstuffs. Less than 1-4% of the production volume is for personal and domestic use e.g. hair bleaching, dying or fixing of hair perm, household cleaning, tooth bleaching, food processing, disinfection of wounds and mouth and disinfection of eye contact lenses. Also cosmetics, toothpastes and deodorants contain or have contained hydrogen peroxide.

3

ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

Environmental releases

Hydrogen peroxide has both natural and anthropogenic sources. Environmental releases from anthropogenic sources may take place during production, formulation, processing and consumer use of products. Natural hydrogen peroxide may be formed by photochemical, chemical or biochemical process.

Environmental fate

General characteristics of H₂O₂ that are relevant for the exposure assessment are:

Degradation

- Abiotic degradation: Abiotic degradation of H₂O₂ is due to either reaction with itself (disproportionation), or reaction with transition metals, organic compounds capable to react with H₂O₂, reaction with free radicals, heat or light. Hydrogen peroxide is normally a short-lived substance in the environment but half-lives vary greatly depending on the circumstances. Thus, no abiotic half-life in water or soil has been determined. The estimated half-life in the atmosphere is ca. 24 hours.
- Biodegradation: Standard ready biodegradation tests are not applicable to the inorganic substances like hydrogen peroxide. However, the data set available is regarded as sufficient to draw conclusions upon the degradation of H₂O₂. Enzymes produced by aerobic bacteria convert hydrogen peroxide to water and oxygen. Based on specific degradation data, a biodegradation rate constant of 21 h⁻¹ (half-life 2 min) in STP is used. In surface waters a realistic worst-case half-life of 5 days is used.

Distribution

A Henry's Law constant of $7.1 \cdot 10^{-4}$ Pa·m³/mol at 20°C was measured. This indicates that volatilisation of H₂O₂ from surface waters and moist soil is expected to be very low. Using the measured log K_{ow} of -1.5, a K_{oc} of 0.2 can be estimated according to the Technical Guidance Document (TGD). Based on this value, hydrogen peroxide is expected to be highly mobile in soil.

Accumulation

There are no experimental results on bioaccumulation available. Hydrogen peroxide is reactive and a short-lived polar substance and no bioaccumulation is expected. This is supported by the calculated log K_{ow} of about -1.5. BCFs calculated according to the TGD for fish and earthworm are low, 1.4 and 3.3, respectively.

Environmental concentrations

The environmental exposure assessment of hydrogen peroxide will be based on the expected releases of the substance during production and processing. Site-specific data received from the industry are used to calculate local PECs in water for H₂O₂ production plants. For pulp bleaching a specific emission scenario based on measurement at several sites was generated. For other uses generic emission scenarios according to the TGD were used. Natural

background concentrations were not included in the PECs. See **Table 3.1** for scenarios assessed and local PECs in various compartments.

Table 3.1 Local PECs in various compartments

	Local PEC in surface water (mg/l)	PEC for microorganisms (mg/l)	Local PEC in soil (mg/kg)	Local PEC in air (mg/m ³)
Production	0.003 - < 0.1 (site specific)	3.29	$1.37 \cdot 10^{-4}$	0.00731
Processing I: Pulp bleaching	0.007 (specific data)	1.1	$1.12 \cdot 10^{-4}$	0.000818
Processing II: Manufacture of other chemicals	0.135	1.32	$1.14 \cdot 10^{-4}$	0.00126
Processing III: Textile bleaching	0.0083	0.053	$1.36 \cdot 10^{-4}$	0.00715
Processing IV: Environmental applications	0.00759	0.0459	$1.09 \cdot 10^{-4}$	$2.25 \cdot 10^{-6}$
Consumer use I: Hair bleaching or dyeing	0.00401	0.0101	$1.09 \cdot 10^{-4}$	$2.25 \cdot 10^{-6}$
Consumer use II: Household cleaning agents	0.00425	0.0125	$1.09 \cdot 10^{-4}$	$2.25 \cdot 10^{-6}$
Consumer use III: Tooth cleaning	0.003	0.0006	$1.09 \cdot 10^{-4}$	$2.25 \cdot 10^{-6}$

3.2 EFFECTS ASSESSMENT

Aquatic compartment (incl. sediment)

There are short-term toxicity data for fish, invertebrates and algae. In addition to algae studies, long-term data are available for zebra mussels. The lowest long-term aquatic toxicity test result is the NOEC of 0.1 mg/l for algae. According to the TGD an assessment factor of 50 should be used for deriving the PNEC in water. However, based on the data on natural background concentrations (typically <1 – 30 µg/l) it is obvious that this would overestimate the toxicity. Furthermore it is not probable that further long-term studies would show higher toxicity than the NOEC for the most sensitive group of organisms, i.e. algae. Therefore an assessment factor of 10 is considered to be appropriate. The extrapolation with the factor of 10 results in a PNEC_{water} of 10 µg/l.

The PNEC for microorganisms is extrapolated from the EC₅₀ activated sludge respiration test (466 mg/l) using an assessment factor of 100. This results in a PNEC_{microorganisms} of 4.66 mg/l.

Hydrogen peroxide does not adsorb to sediment and is rapidly degraded there. Sediment dwelling organisms are adequately protected by the PNEC for water phase.

Terrestrial compartment and atmosphere

No studies are available on the effects of hydrogen peroxide on soil-dwelling organisms. Some studies on plants are available but these data cannot be used for calculating the PNEC since there is no information on the actual concentration of the substance in soil. Thus the PNEC is calculated based on the equilibrium partitioning method:

$$PNEC_{\text{terrestrial}} = 1.19 \cdot 10^{-3} \text{ mg/kg wwt.}$$

Some experiments are available on fumigation of plants with H₂O₂. No NOEC or EC₅₀ levels were determined in these tests and as there are no data on dose-response, a quantitative assessment for the atmosphere cannot be performed.

3.3 RISK CHARACTERISATION

Aquatic compartment (incl. sediment)

Based on the site-specific data, PEC/PNEC ratios for aquatic organisms are calculated for all production plants; some of them also use H₂O₂ for manufacture of other chemicals. In 19 cases there is no need for further information and/or testing: **conclusion (ii)** but there are four plants for which there is a need for limiting the risks: **conclusion (iii)**.

Table 3.2 Local PEC/PNEC ratios for aquatic organisms and microorganisms

Scenario	Aquatic organisms	Microorganisms
Production	See above	0.706
Processing I: Pulp bleaching	0.7	0.236
Processing II: Manufacture of other chemicals	13.5	0.283
Processing III: Textile bleaching	0.83	0.0114
Processing IV: Environmental applications	0.759	0.00985
Consumer use I: Hair bleaching or dyeing	0.401	0.00216
Consumer use II: Household cleaning agents	0.425	0.00267
Consumer use III: Tooth cleaning	0.3	0.000135

There is no need for further information and/or testing: **conclusion (ii)** for any of the generic scenarios with the exception of the use of hydrogen peroxide in manufacture of other chemicals for which there is a need for limiting the risks: **conclusion (iii)**.

H₂O₂ does not adsorb to the sediment and is rapidly degraded there. Therefore a separate risk characterisation for sediment has not been performed. It is covered adequately by the risk assessment for surface water.

Terrestrial compartment

In the terrestrial compartment for all the scenarios, there is no need for further information and/or testing: **conclusion (ii)**.

Atmosphere

No quantitative risk assessment has been carried out for the atmospheric compartment due to the lack of EC₅₀ values for terrestrial plants.

In a test carried out with trees some effects on needles and leaves have been observed when the hydrogen peroxide concentrations in fog water varied from 0.2 to 5 mg/l. These values are comparable to typical concentrations in rainwater during Summer. Thus, effects of hydrogen peroxide on plants cannot be totally excluded but it must be born in mind that also plants have an enzymatic capacity of decomposing H_2O_2 to some extent: **conclusion (ii)**.

Secondary poisoning

Not relevant.

Regional risk characterisation

The PECs for air, water and soil calculated at a regional scale do not exceed the corresponding PNECs: **conclusion (ii)**.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

Occupational exposure

The major use of hydrogen peroxide (H_2O_2) is in bleaching processes of pulp and paper. H_2O_2 is also widely used in the production of inorganic and organic chemicals. The exposure evaluation in this report is performed on the basis of scarce published data, industrial hygienists' site visits and measurements in the reporting country, expert interviews in industry, authorities, and among Finnish H_2O_2 producers and importers.

The reasonable worst-case exposure levels for the workers' exposure categories were estimated from limited measured data, together with general knowledge of the production, transportation and use of H_2O_2 (expert judgment). In spite of the inadequacy of data, the material showed that in continuous processes 8-hour exposures rarely exceeded the OELs in the workplace. Higher short-term exposures were nevertheless common and could occur in every process especially when concentrated H_2O_2 was dosed or diluted manually.

Predicted exposures were also calculated with the EASE model revealing often values close to the levels measured. Sometimes EASE gave unrealistically high results. The best results with the EASE model were obtained when the evaluator had a good knowledge of the process in question.

Table 4.1 summarises the scenarios identified for the occupational exposure to H_2O_2 . The duration and frequency of exposure, numerical values for the proposed reasonable worst-case exposures, data sources and the results of the EASE estimations are given.

Table 4.1 Summary of occupational exposure estimates for hydrogen peroxide

Industrial category	Conditions of exposure		RWC/TWA/8h (unless indicated RWC/STE)	Reference ²	EASE model exposure estimation	
	Working time (h/d)	Frequency (d/a)	mg/m ³		Inhalation (mg/m ³)	Dermal (mg/cm ² /d)
Production of H ₂ O ₂ (synthesis, distillation, stabilisation, dilution, diverse tasks)	7-8	200	0.8	CEFIC (1997e)	4.2-14	0-0.1
Synthesis of other chemicals	7-8	200	0.2 closed process 0.5 batch process 2.0 batch, RWC/STE	CEFIC (1997e); Degussa-H (1999)	0-0.14 closed 0.7-1.4 batch -	very low very low -
Loading operations	~ 4	200	2	CEFIC (1997e); Degussa-H (1999)	4.2-14	0-0.1
Pulp and paper bleaching	8	200	0.7	FIOH (1997; 1998)	0-0.14	very low
Bleaching of textiles and laundering: automated batch bleaching	8 ½	200 100	0.2 1	FIOH (1998)	0-0.14 14.1-28.2	very low 0.1-1
Aseptic packaging: immersion bath (im), spray method (sp)	8	200	1.5	FIOH (1985, 1998); Dietschmann (1996) Kaelin (1988); Suenaka (1984)	0.7-4.2 (im) 14-70 (sp)	0.1-1
Hydrogen peroxide and peracetic acid use: brewery	1-2	200	0.5	FIOH (1998)	0.7-4.2	0-0.1
Peracetic acid use: meat processing	1-2	200	0.5	FIOH (1998)	4.2-7.0	0.1-1
Etching of circuit boards: modern process (closed) old process (batch)	8 8	200 100	0.2 1.5	FIOH (1989; 1996)	0-0.14 0.7-4.2	- 0-0.1
Metal plating	½	20	0.14 2 RWC/STE/30 min	Expert	14.1-28.2	0-0.1
Production of modified starch	8	200	0.2	FIOH (1998)	0-0.14	very low
Degrading of proteins	1	200	0.27 2 RWC/STE	FIOH (1998)	14.1-28.2	0-0.1
Water treatment: drinking water, wastewater			drinking 0.14 waste 1.0 waste 7.0 RWC/STE		Drink. 0.014 Waste 4.2-7	very low very low
Hairdresser's work	8	200	0.5 RWC/8h	FIOH (1999); Wella AG LAN (1992)	4.2-7.1	0.1-1

RWC/TWA (8 h): reasonable worst case; RWC/STE: reasonable worst case for short-term exposure

² For full references, see the comprehensive risk assessment report on the ECB website, <http://ecb.jrc.it>

Consumer exposure

Bleaching, disinfection and cleaning are the main uses of H₂O₂ in consumer products. Many consumer products, such as household cleaning and bleaching agents, hair dyeing and bleaching products, tooth bleaching agents, mouthwashes, disinfectants, contact lens disinfectants, and even food contain H₂O₂. These products cause exposure via the gastrointestinal tract and occasionally via inhalation, by deposition on the skin, eye exposure, and exposure to the gingiva and the teeth in some specific scenarios. Based on the current knowledge, the use of toothpaste and mouth rinses containing H₂O₂ is not a common practice. Moreover, parts of H₂O₂ in the toothpaste and mouthwash are likely to decompose before the residual amount in the mouth is swallowed. The oral exposure from tooth bleaching agents is occasional and therefore it is not added to the daily oral exposures.

Combining consumer exposures via the gastrointestinal tract, via inhalation and through the skin is not appropriate, because the expected effects are local and not systemic.

Table 4.2 summarises the scenarios identified for the consumer exposure to H₂O₂. The duration and frequency of exposure and values for the external, route-specific doses/concentrations are given.

Table 4.2 Consumer exposure to hydrogen peroxide

Scenario	Exposure time		Inhalation (mg/ m ³)		Ingestion (mg/kg of bw/d)	Skin / Eye deposition	
	Duration of treatment	Frequency of treatments per year	Measured	Estimated	Estimated	Concentration of H ₂ O ₂ in the product	Estimated dose
Hair dyeing and bleaching	30 min	4	<0.07-0.20	0.01-0.24 ^{a)}	na	1-6%	12 mg/kg bw, on the skin ^{b)}
Textile bleaching	5-10 min	25		0.02-0.13	na	<8 (35) %	0.6 mg/kg bw, on the skin ^{c)}
Cleaning agents	10-20 min	25		<0.13	na	usually about 8% (0.2-35%)	<0.6 mg/kg bw, on the skin ^{c)}
Contact lens disinfectants	1-5 min	365	na	na	na	15mg/l residual concentration in lenses	
Tooth bleaching	30 min -10 h, over up to 2 weeks	5-10		negligible ^{d)}	negligible	2-35%	<1 mg per application on gingival surfaces
Food items (natural and residual H ₂ O ₂)		365	na	na	0.033-0.13	na	na
Mouth care products	5 min	5 · 365	na	na	0.088	0.1%	

- a) 0.2 mg/m³ represents a realistic worst-case scenario, where the original solution contains 12% of hydrogen and the mixture, which is actually applied, contains 6% of hydrogen peroxide (SCIES modelling system was used)
- b) 12 mg/kg of body weight per day is the potential dermal deposition. Systemically distributed amount of hydrogen peroxide is considered insignificant
- c) 0.6 mg/kg of body weight per day is the potential dermal deposition (estimated by the EUSES)
- d) Evaporation from the gels used for tooth bleaching is assumed to be minimal,
na not applicable

Humans exposed via the environment

After H₂O₂ has been released to the environment, it rapidly decomposes in the presence of organic material. In human food, or in drinking water, no accumulation of exogenous H₂O₂ has been observed.

According to modelling done with EUSES, average air concentration of H₂O₂ and concentration during emission episodes near to a point source remains low (about 0.005 mg/m³). It was found that the concentration of H₂O₂ in human exhaled air is of the order $0.5 \cdot 10^{-8}$ M and thus much higher than the concentration in the ambient air. Therefore, ambient air does not represent a source for human exposure to H₂O₂.

Drinking water may contain low concentration of H₂O₂ originating from industrial point sources, natural sources and possibly from water treatment processes. EUSES modelling gave a H₂O₂ concentration of about 0.2 mg/l near to the production plant (maximum of different release scenarios). Further decomposition probably decreases this concentration level in water before it is used as drinking water. It can be estimated that the intake from drinking water would thus be 1 mg/day (0.017 mg/kg bw per day), which is low compared with other sources of oral exposure to H₂O₂. Overall contribution from drinking water is not significant.

EUSES predicted high estimates of H₂O₂ concentration in leaf crops (16.1 mg/kg) in a local scale, caused by releases from a local point source. This concentration causes an intake of 0.28 mg/kg of bw per day, which is relatively high compared with other sources of oral exposure. The route of H₂O₂ from the point of release to the leaf plants is unclear. EUSES predicted amounts of dietary intake from other food items e.g. from fish, milk and meat are negligible.

4.1.2 Effects assessment

Toxicokinetics, metabolism and distribution

H₂O₂ is an endogenous product of oxygen reduction in the aerobic cell and passes readily across biological membranes. At high-uptake rates H₂O₂ can pass the absorption surface entering the adjacent tissues and blood vessels where it is rapidly degraded by catalase liberating oxygen bubbles; consequently, mechanical pressure injury and oxygen embolism may be produced. Regarding H₂O₂ inhalation or skin contact at rates that would correspond to occupational exposures, there are no data on the systemic fate of the substance. In view of the high-degradation capacity for H₂O₂ in blood it is however unlikely that the substance is systemically distributed, and therefore the endogenous steady state levels of the substance in tissues are unlikely to be affected.

Acute toxicity

The oral LD₅₀ values or lethal doses in rats range between 800 mg/kg for 70% H₂O₂ to more than 5,000 mg/kg for 10% H₂O₂. There are also a number of reported human incidents by oral ingestion of H₂O₂ water solutions, but few reports have given data on the dose. The mechanism of systemic effect has been oxygen embolism. Thus, the substance proved to be harmful if swallowed by a physical mode of action.

The dermal LD₅₀ values in animals range between 700-5,000 mg/kg for 90% H₂O₂. The test methods are mostly poorly described, but the studies indicate that H₂O₂ is not acutely toxic after skin application.

Acute inhalation toxicity studies have been performed with aerosols (mice) and vapours (rats and mice). Due to the corrosive nature of the substance after inhalation exposures to highly concentrated aerosols (70% H₂O₂ as “droplets”), lethality occurs at quite low air concentrations of this substance (0,92-2 mg/l). The lethal event can be attributed to the substance corrosivity rather than its systemic toxicity. Since exposure to significant concentrations of H₂O₂ aerosols was not found in the occupational assessment and the predominant exposure concerned vapours, the vapour exposure experiments should be preferred for hazard assessment purposes. A marked difference in susceptibility to H₂O₂ between mice and rats after inhalation uptake of vapours may be deduced from the available studies. In mice, vapour concentrations of up to 0,3 mg/l for 4 hours caused death of at least half of the animals within 2 weeks. In rat studies, no mortality was observed at comparative exposure concentrations. The substance is considered to be harmful by inhalation.

Irritation and corrosivity

In rabbits, H₂O₂ solutions of 10% were slightly irritating to the skin, 35% solutions proved to be moderately irritating and caused delayed epidermal necrosis and sloughing, while 50% solutions and more concentrated solutions were severely irritating and corrosive.

Eye irritation is reported in humans and animals. The effect of H₂O₂ in 5 and 10% solutions are known to cause adverse effects in humans. A 8% solution was highly irritating and caused irreversible effects in the rabbit eye.

H₂O₂ is a respiratory irritant at relatively high concentrations (aerosols generated from 70% H₂O₂), and an RD₅₀ value of 665 mg/m³ was derived. Slight nasal irritation was observed among workers exposed to the maximum mean level of 3.5 mg/m³ during one-hour periods of drum and tank filling at a H₂O₂ production facility, and slight respiratory irritation was reported in volunteers exposed to 10 mg/m³ of H₂O₂ vapours.

Sensitisation

There are two reported cases of positive patch tests to H₂O₂ and some uncertainty surrounds an outdated animal study with a negative result. However, when one takes into account the widespread occupational and consumer uses over many decades, it may be concluded that the potential of H₂O₂ to cause skin sensitisation is extremely low.

Repeated dose toxicity

Decreased body weight gain was a typical finding in gavage studies in rats employing a dose range of 50-500 mg/kg bw/day; regarding other effects, decreased values of blood parameters were not uncommon observations. When administered in drinking water H₂O₂ consistently decreased the body weight gain at 1,500 ppm (0,15%) in rats and at 3,000 ppm in mice. Reduction of water intake was also characteristic. This indicates that the NOAEL of H₂O₂ in drinking water was 100 ppm implying a daily dose of 26-37 mg/kg bw. The LOAEL was 300 ppm (76-103 mg/kg bw) based on reductions in food and water consumption and on duodenal mucosal hyperplasia.

Concerning repeated inhalation toxicity of H₂O₂, there are indications of local effects in the skin, airways, and the lungs at about 10 mg/m³ in rats and dogs. A recent 28-day range finding inhalation toxicity study in the rat showed a respiratory tract irritation and concentration-related necrosis and inflammation of the epithelium in anterior regions of the nasal cavity from 14.6 mg/m³, but not at 2.9 mg/m³.

Industrial experience from health surveillance of H₂O₂ production workers suggested no exposure-related effects on simple respiratory functions at airborne levels up to 0.8 mg/m³ or less than 1.4 mg/m³ with short-term peaks up to about 5 mg/m³. A health monitoring study of a small group of aseptic packaging workers linked sustained respiratory tract irritation and inflammation, susceptibility to respiratory infections, and asthma symptoms to airborne peroxide exposure at 2-3 mg/m³ (apparent LOAEL) as a time-weighted average over the whole shift with peaks up to 11 mg/m³. Since there is no evidence that exposure to airborne hydrogen peroxide at levels well below the OEL causes adverse effects in the respiratory system, it is provisionally considered that peroxide concentrations in excess of the OEL (1.4 mg/m³ for 8-hour TWA) cause risk. Acknowledging the uncertainties especially involving pulmonary effects, a careful follow up of relevant future studies in workers exposed to peroxide vapours, as well as all possible information on repeated inhalation toxicity on sodium perborate would be desirable. Also a single human case of an interstitial lung disease has been reported, which occurred during occupational exposure to about 12-41 mg/m³ of H₂O₂.

Mutagenicity

H₂O₂ is a mutagen and genotoxicant in a variety of *in vitro* test systems.

Regarding *in vivo* genotoxicity, studies have explored DNA repair in liver cells of rats, as well as micronucleus formation in mice, all with a negative outcome. At low concentrations (0.2-3.2% solutions), and with a low application frequency on the skin of mice, H₂O₂ did not induce local genotoxicity or mutagenicity. The available studies are not in support of significant genotoxicity/mutagenicity of H₂O₂ under *in vivo* conditions.

Carcinogenicity

Although 0.1-0.4% H₂O₂ in drinking water showed potential to induce local carcinogenic effects in the duodenum of a sensitive, catalase-deficient mouse strain, it is notable that the lesions showed a marked tendency of regression and even disappearance after the cessation of treatment. The mechanism of the carcinogenic effect is unclear. In rats, administration of H₂O₂ in drinking water was not associated with the occurrence of tumours. In another study, however, 1% H₂O₂ in drinking water induced squamous cell papillomas in the forestomach of rats. Tumour promotion studies with H₂O₂ revealed equivocal results. The special nature of the demonstrated carcinogenicity of H₂O₂, an endogenous reactive oxygen species, the existing biological defence mechanisms, and the overall evidence available, cast some doubt on whether H₂O₂ is a carcinogen of practical significance and the evidence is considered to be insufficient to trigger classification.

Toxicity for reproduction

No appropriate studies were available for a complete evaluation of reproductive and developmental toxicity. Limited studies with mice and rats exposed to H₂O₂ in drinking water suggested no grave disturbances on the male or female reproductive functions. Moreover, an appropriate 90-day drinking water study with catalase-deficient mice, and carcinogenicity studies with mice and F344 rats did not identify testes or ovaries as target organs. The only available developmental toxicity (feeding) study in rats showed foetotoxic effects, but the study contains major uncertainties regarding the exposure and mode of action; therefore, it could not be used for an evaluation.

Thus there is a clear data gap regarding developmental toxicity for H₂O₂. However, during the risk assessment process no further testing on this endpoint was required, since conventional study protocols were judged unlikely to show specific embryonal or foetal effects: firstly,

because it is doubtful whether H₂O₂ would reach the foetus (as opposed to degradation products oxygen and water) and secondly, because local effects and hence potential general toxicity are expected in the mother.

4.1.3 Risk characterisation

The toxicokinetic evaluation of H₂O₂ suggests that only under conditions of very high exposure rates the substance might enter the systemic circulation. When accidental swallowing is excluded, it is unlikely that such high exposures could be reached in any realistic scenario of occupational or consumer exposure. It is especially unlikely that the substance deposited on the skin is systemically absorbed to a meaningful degree. Results from animal studies also suggest local toxicity at the point of contact and no systemic effect as the primary mode of action. Although there were gaps in data, reproductive effects by H₂O₂ were not deemed to cause any concern, and they are not considered in the risk characterisation.

Workers

Based on the available data set for the various effect endpoints, irritation/corrosivity, depending on concentration, in the eyes, skin and airways and repeated dose toxicity by inhalation (lungs) are the most relevant adverse effects for workers.

From the risk point of view, these effect endpoints were evaluated for each of the 19 scenarios. All other endpoints: acute toxicity, sensitisation, repeated oral toxicity, repeated dermal toxicity, mutagenicity and carcinogenicity were not considered to cause concern for human health in the occupational setting: **conclusion (ii)**.

Conclusion (iii) applies to:

- irritation/corrosivity for the eye, skin and respiratory tract, depending on concentration, in H₂O₂ loading operations,
- to eye and skin irritation/corrosivity, depending on concentration, in bleaching of textiles (batch process), aseptic packaging (old type of immersion bath machines), hydrogen peroxide and peracetic acid use in breweries, etching of circuit boards (old process), metal plating, and degrading of proteins,
- eye irritation/corrosivity in hairdresser's work,
- repeated inhalation toxicity in loading operations, aseptic packaging (all types of machine), etching of circuit boards (old process) and wastewater treatment.

The summary of the conclusions is shown in **Table 4.3**.

Table 4.3 Risk characterisation for workers

Scenario	Irritancy/corrosivity			Repeated inhalation toxicity	Acute toxicity, sensitisation, repeated oral toxicity, repeated dermal toxicity, mutagenicity, carcinogenicity
	Eye	Skin	Airways		
Production of H ₂ O ₂	ii	ii	ii	ii	ii
Synthesis of other chemicals	ii	ii	ii	ii	ii
Loading operations	iii	iii	iii	iii	ii
Pulp and paper bleaching	ii	ii	ii	ii	ii
Bleaching of textiles, batch process	iii	iii	ii	ii	ii
Bleaching of textiles, automated process	ii	ii	ii	ii	ii
Industrial laundering	ii	ii	ii	ii	ii
Aseptic packaging: immersion bath process, old type	iii	iii	ii	iii	ii
Aseptic packaging: other types	ii	ii	ii	iii	ii
Hydrogen peroxide and peracetic acid use: brewery	iii	iii	ii	ii	ii
Peracetic acid use: meat processing	ii	ii	ii	ii	ii
Etching of circuit boards, modern process	ii	ii	ii	ii	ii
Etching of circuit boards, old process	iii	iii	ii	iii	ii
Metal plating	iii	iii	ii	not relevant	ii
Production of modified starch	ii	ii	ii	ii	ii
Degrading of proteins	iii	iii	ii	ii	ii
Drinking water treatment	ii	ii	ii	ii	ii
Wastewater treatment	ii	ii	ii	iii	ii
Hairdresser's work	iii	ii	ii	ii	ii

Consumers

Local irritation and, in extreme and uncommon cases, corrosion of the skin, eye, gingivae or the teeth are the critical adverse effects caused by exposure to H₂O₂. Most of the effects reported (e.g. eye irritation, irritation of the gingivae and the throat, sensitivity and inflammation of tooth pulp, and morphological changes in tooth surface) are transient or are considered mild. However, even rather dilute solution of H₂O₂ (3%) may cause danger, if swallowed in large enough volume accidentally. Furthermore, effects of splashes of strong solutions to the eye ($\geq 5\%$) and skin ($\geq 35\%$), and tooth resorption occasionally reported after tooth bleaching by dentists ($\geq 35\%$), represent some serious scenarios which are relevant in terms of consumer exposure.

Effect endpoints that are similarly relevant for worker and consumer exposures are described and discussed in detail in the previous section.

Thus, the conclusions regarding sensitisation, mutagenicity and carcinogenicity are **conclusions (ii)**.

Repeated inhalation exposures to H₂O₂ do not occur in consumer exposure scenarios, therefore this endpoint is not relevant. On the other hand, the effects on the gingivae and the teeth, and the oral exposure caused by food, mouthwash and toothpastes are relevant only for consumers and are covered in this section.

Risks were characterised for the following consumer exposure scenarios: hair dyeing and bleaching, textile bleaching, use as a cleaning agent, contact lens disinfection, tooth bleaching, ingestion in food, and mouth care products.

Only food and use of mouth care products cause repeated oral dosage of H₂O₂. Thus, for the other five scenarios, the toxicity caused by repeated oral exposure is not relevant: **conclusion (ii)**.

Conclusion (iii) applies to:

- eye irritation/corrosivity in hair dyeing and bleaching, in textile bleaching and in use as a cleaning agent,
- risk of tooth injury if tooth bleaching is performed (by dental personnel) using concentrated (35%) H₂O₂ preparations.

The summary of the conclusions is shown in **Table 4.4**.

Table 4.4 Risk characterisation for consumers

Scenario	Irritation/corrosivity			Repeated dose toxicity, oral	Acute toxicity; sensitisation; mutagenicity; carcinogenicity others
	Eye	Skin	Airways		
Hair dyeing and bleaching	iii ¹⁾	ii ²⁾	ii	ii	ii
Textile bleaching	iii ³⁾	ii	ii	ii	ii
Cleaning agents	iii ³⁾	ii	ii	ii	ii
Contact lens disinfection	ii	ii	ii	ii	ii
Tooth bleaching	ii	ii	ii	ii	iii ⁴⁾
Ingestion in food	ii	ii	ii	ii	ii
Mouth care products	ii	ii	ii	ii	ii

- 1) Eye irritancy is of concern if the concentration of hydrogen peroxide in the substance used is $\geq 5\%$.
- 2) Skin irritation has been observed. It is likely that not H₂O₂ alone, but the combined exposure with ammonium persulphate and dye compounds, e.g. amines causes irritation or allergic symptoms when hydrogen peroxide concentration in the applied mixture is as regulated (6% or lower).
- 3) Current data suggest that textile bleaching products and cleaning agents available for consumers normally contain less than 8% of H₂O₂ but in some cases may contain up to 35% of H₂O₂. Eye irritancy is of concern if the actual concentration of H₂O₂ in the substance used is $\geq 5\%$.
- 4) After treatment with 35 % of hydrogen peroxide by dentists, effects on tooth pulp, odontoblast destruction and resorption of non-vital teeth have been observed. Risk reduction should be considered in an appropriate forum.

Humans exposed via the environment

According to EUSES modelling, indirect exposure of humans to H₂O₂ via ambient air and drinking water resulting from local releases is low, and does not cause a concern: **conclusion (ii)** for all endpoints.

Compared to other recognised sources of oral exposure (notably endogenous content in food), EUSES predicted a rather high oral intake from leaf crops (0.28 mg/kg bw per day) at a local

scale, caused by releases from a local point source. When this oral exposure is compared with the NOAEL of 100 ppm H₂O₂ in drinking water (daily doses 26 and 37 mg/kg bw for male and female mice, respectively), derived from a 90-day study with mice, a margin of safety (MOS) of 93-132 is derived. This MOS is considered sufficient for repeated oral toxicity: **conclusion (ii)**.

Combined exposure

Combined exposure needs to be addressed regarding oral intake of H₂O₂ from consumer sources and from indirect environmental sources. The combined intakes may amount to 0.4-0.5 mg/kg bw per day. Compared to the repeated dose toxicity NOAELs of 26 and 37 mg/kg bw per day in male and female mice, respectively, obtained in a drinking water study, a MOS of 52-93 is derived. This MOS is considered sufficient for repeated oral toxicity: **conclusion (ii)**.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

Occupational exposure

See also Section 4.1.1, Occupational exposure. Exposure to high enough levels of H₂O₂ to present a potential physico-chemical hazard under normal handling and use could arise during production, transportation, storage and industrial use.

The production and major industrial uses (pulp and paper bleaching, manufacture of chemicals, textile bleaching) take normally place in automated, closed or partially closed systems and stringent exposure and hazard controls are mostly in place. Containers used for transportation and storage meet special safety requirements. Exposure of workers to high levels of H₂O₂ causing physico-chemical hazards could occur in accidental situations only.

The small industries use H₂O₂ often as diluted solutions (disinfection purposes in the dairy, refreshment and foodstuff industries), although concentrated solutions are also used (aseptic packaging, metal pickling, electronics industry, degrading organic materials). The small industries obtain the peroxide in smaller containers. Feeding of the substance to the process, or further dilution, is mainly done manually or at least partly manually (by decanting, with siphons or small movable pumps). The danger of leaks and spills during manual handling is high. Even transportation and storage of the small containers need special attention. In the small factories, workers awareness of the dangerous properties of H₂O₂ seems to be insufficient.

Consumer exposure

See also Section 4.1.1, Consumer exposure. An accident may occur if H₂O₂ (even diluted) is stored in a glass bottle with a tight stopper. In the course of time, overpressure will be generated in the bottle due to slow decomposition of the peroxide and the bottle may break. Larger spills of the concentrated peroxide (≥25%) on materials such as clothing, wood or paper may cause danger of fire if the substance remains unwashed.

Humans exposed via the environment

Humans are not exposed indirectly via the environment to levels of H_2O_2 causing any physico-chemical hazards.

4.2.2 Effects assessment

H_2O_2 is a reactive unstable chemical. Its decomposition is highly exothermic. During its application, it will react or decompose producing oxygen and water steam. In case of spillage or accident, rapid decomposition will take place in every natural compartment producing gaseous reaction products.

Concerning the reactivity of aqueous solutions of H_2O_2 used in industry, all major producers warn that various contaminations will cause its decomposition. The decomposition of concentrated solutions may be very vigorous with rapid generation of large volumes of oxygen and water steam.

Explosivity

H_2O_2 does not fulfil the criteria for classification as an explosive. At high concentrations, H_2O_2 alone has however explosion potential. At concentrations above 26 vol %, the vapour is explosive by means of decomposition. Thus H_2O_2 aqueous solutions of 74 w/w % strength or higher can produce explosive vapours at elevated temperature and/or at decreased pressure. At concentrations above 86 w/w %, the liquid itself can be made to explode.

Lower concentrations may cause spontaneous fire on contact with combustible materials.

Flammability

Autoignition and flammable limits, lower or upper, are not applicable and the compound is non-combustible.

While pure H_2O_2 does not burn, it can initiate spontaneous ignition of organic materials such as paper, wood or cloth via oxidation. Ignition may be rapid but it can also be delayed for several hours. Spontaneous ignition and fire can occur in the event of leaks or spills of even diluted (≥ 25 %) solutions if the peroxide solution is allowed to remain in the combustible material. The mechanism is that water is first volatilised thus causing the peroxide to concentrate, whereupon the peroxide sets the material on fire. Rapid oxygen evolution from decomposing H_2O_2 may increase the intensity of fire especially in closed unventilated spaces.

Oxidising property

Owing to its potential exothermic decomposition and high molecular oxygen content, H_2O_2 is a powerful oxidiser. H_2O_2 solutions containing ≥ 50 w/w % of the substance will be classified as oxidisers according to Directive 67/548/EEC. The compound is also classified as an oxidising agent in the transport sector. According to UN classification, aqueous solutions of H_2O_2 (UN no 2014) ≥ 8 % belong to class 5.1, i.e. oxidisers.

4.2.3 Risk characterisation

Workers

The assessment of physico-chemical hazards indicates that H₂O₂ is highly unstable, and its concentrated solutions are oxidising and apt to decompose gradually or even explosively if not free from contaminants, well stabilised, and if not used at normal temperature and pressure. The spontaneous exothermic decomposition presents a high risk of fire when the concentrated substance comes in contact with combustible materials. Thus **conclusion (iii)** is appropriate.

The major industrial uses in closed automated production systems, employment of special containers with safety valves for storage and shipping, and stringent safety controls, imply that the potential risk of physico-chemical hazards to workers in the major industries is minimal under conditions of normal handling and use. Even the highest measured peak levels of exposure (in the event of leaks: 9-15 ppm) are far below the levels that may cause explosion hazard. The risk of fire and explosion is also addressed in the safety data sheets provided by the major producers.

In minor industrial uses, hazards may be involved in manual operations with H₂O₂. The peroxide is manually delivered from small containers with cans or pails or by the use of siphons or movable pumps to the processes. Spills and leaks are commonplace. Workers knowledge of the oxidative property of H₂O₂, and of the hazard of rupture of the container due to spontaneous decomposition was not deemed sufficient. There were no safety management systems implemented for either the process use, or for storing or transporting the substance inside the factory.

Consumers

H₂O₂ products for consumer use are more diluted than the ones used in industry. However, exceptionally, products may contain higher concentrations of H₂O₂ and spills on combustible materials may involve a fire hazard, if the substance is not removed by washing. Thus **conclusion (iii)** is appropriate. Storing the substance in a bottle with a tight stopper may also result in increased pressure inside the vessel due to spontaneous decomposition, and violent rupture.

Humans exposed via the environment

There is no risk of physico-chemical hazard for the population due to indirect exposure to H₂O₂ via the environment.

5 RESULTS

5.1 ENVIRONMENT

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion is reached because of:

- concerns for effects on the aquatic compartment as a consequence of exposure arising from four production sites and use in manufacture of other chemicals.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to:

- the aquatic compartment for 19 production sites, pulp bleaching, textile bleaching, environmental applications and consumer use.
- microorganisms in the sewage treatment plant, the terrestrial environment and the atmosphere for production, all processing scenarios and consumer use.

5.2 HUMAN HEALTH

5.2.1 Human health (toxicity)

Workers

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion is reached because of:

- concerns for skin, eye and respiratory tract irritation and/or corrosivity, depending on concentration as a consequence of exposure arising from loading operations.
- concerns for skin and eye irritation and/or corrosivity, depending on concentration, as a consequence of exposure arising from bleaching of textiles (batch process), aseptic packaging (old types of immersion bath machines), hydrogen peroxide and peracetic acid use in breweries, etching of circuit boards (old process), metal plating, degrading of proteins.
- concerns for eye irritation and/or corrosivity, depending on concentration, as a consequence of exposure arising from hairdresser's work.
- concerns for repeated inhalation toxicity in loading operations and aseptic packaging (all types of machines), etching of circuit boards (old process) and wastewater treatment.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to:

- acute toxicity, sensitisation, repeated oral toxicity, repeated dermal toxicity, mutagenicity and carcinogenicity for all exposure scenarios.
- skin, eye and respiratory tract irritation and/or corrosivity in production of H₂O₂, synthesis of other chemicals, pulp and paper bleaching, bleaching of textiles (automated process), industrial laundering, aseptic packaging (other than old types of immersion bath processes), peracetic acid use in meat processing, etching of circuit boards (modern process), production of modified starch, drinking water treatment, and wastewater treatment.
- respiratory tract irritation in bleaching of textiles (batch process), aseptic packaging (old types of immersion bath machines), hydrogen peroxide and peracetic acid use in breweries, etching of circuit boards (old process), metal plating, degrading of proteins.
- both skin and respiratory tract irritation in hairdresser's work.
- repeated inhalation toxicity in production of hydrogen peroxide, synthesis of other chemicals, pulp and paper bleaching, bleaching of textiles (batch and automated processes), industrial laundering, hydrogen peroxide and peracetic acid use in breweries, peracetic acid use in meat processing, etching of circuit boards (modern process), production of modified starch, degrading of proteins, drinking water treatment, and hairdresser's work.

Consumers

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion is reached because of:

- concerns for eye irritation as a consequence of exposure arising from hair dyeing and bleaching and concerns for eye irritation/corrosivity in use of textiles bleaches and cleaning agents, if the actual concentration of hydrogen peroxide is >5%.
- concerns for specific adverse effects on tooth pulp and teeth as a consequence of exposure arising from tooth bleaching with 35% of hydrogen peroxide by a dentist.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to:

- acute toxicity, sensitisation, repeated oral toxicity, repeated dermal toxicity, mutagenicity and carcinogenicity for all exposure scenarios.
- skin, eye and respiratory tract irritation in the context of contact lens disinfection, tooth bleaching, ingestion in food, and use of mouth care products.
- both skin and respiratory tract irritation in hair dyeing and bleaching, in textile bleaching and use as a cleaning agent.

Humans exposed via the environment

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to:

- all effect endpoints for humans exposed to hydrogen peroxide via the environment.

Combined exposure

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

5.2.2 Human health (risks from physicochemical properties)

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the workers and to the consumers because of:

- concerns for the risk of fire hazard caused by spills of the more concentrated ($\geq 25\%$) hydrogen peroxide solutions on combustible materials.

