Extended Safety Data Sheet

According to Regulation (EC) No 1907/2006

vinyl acetate

Issue date: 22/01/2015 **Version 2.0**

Revision date: 27/06/2018 **eSDS Record Number: CSSS-TCO-010-116090**

Section 1 Identification of the substance/mixture and of the company/undertaking

1.1 Product identifier:

Identification on the label/Trade name:	vinyl acetate
Additional identification:	vinyl acetate
Identification of the product:	CAS#:108-05-4 ; EC#: 203-545-4
Index Number:	607-023-00-0
REACH registration No.:	01-2119471301-50-xxxx

1.2 Relevant identified uses of the substance and uses advised against:

1.2.1 Identified uses:

Polymer production

1.2.2 Uses advised against:

Not available.

1.3 Details of the supplier of the safety data sheet:

Supplier(Only representative):	Chemical Inspection & Regulation Service Limited
Supplier(Manufacturer):	SHANDONG CHEMICHASE CHEMICAL CO.,LTD
Address:	DONGYING DISTRICT, DONGYING CITY
Contact person(E-mail):	SHANDONG (Email:vipt@chemichase.com)
Telephone:	+86- 546-7265597
Fax:	+86-546-8275057

1.4 Emergency telephone Number:

+353 41 6871874

Available outside office hours?	YES		NO	X

Section 2 Hazards Identification

2.1 Classification of the substance/mixture

2.1.1 Classification:

The substance is classified as following according to REGULATION (EC) No 1272/2008:

REGULATION (EC) No 1272/2008	
Hazard classes/Hazard categories	Hazard codes
Flam. Liquid 2	H225
Acute Tox. 4	H332
STOT Single Exp. 3	H335
Carc. 2	H351
Aquatic Chronic 3	H412

For full text of H- phrases: see section 2.2.

2.2 label elements

Hazard Pictograms:

Signal Word(S): Danger

Hazard Statement:

H225: Highly flammable liquid and vapour.

H332: Harmful if inhaled.

H351:Suspected of causing cancer.

H335: May cause respiratory irritation.

H412: Harmful to aquatic life with long lasting effects

Precautionary statement

P201: Obtain special instructions before use.

P202: Do not handle until all safety precautions have been read and understood.

P210: Keep away from heat/sparks/open flames/hot surfaces. No smoking.

P233: Keep container tightly closed.

P240: Ground/bond container and receiving equipment.

P241: Use explosion-proof electrical/ventilating/lighting equipment.

P242: Use only non-sparking tools.

P243 : Take precautionary measures against static discharge.

P261: Avoid breathing dust/fume/gas/mist/vapours/spray.

P264: Wash thoroughly after handling.

P270: Do not eat, drink or smoke when using this product.

P271: Use only outdoors or in a well-ventilated area.

P273: Avoid release to the environment.

P280: Wear protective gloves/protective clothing/eye protection/face protection.

P281: Use personal protective equipment as required.

2.3 Other hazards

The substance is not considered a PBT/vPvB.

Section 3 Composition/information on ingredients

Substance/Mixture: Substance

Ingredient(s):

Chemical Name	Registration No.	CAS No.	EC No.	Concentration
Vinyl Acetate	01-2119471301-50-0063	108-05-4	203-545-4	99.95%
Unknown impurities	N/A	N/A	N/A	0.05%

Section 4 First aid measures

4.1 Description of first aid measures:

Immediately remove any clothing contaminated by the product. Symptoms of poisoning may occur after several hours. Medical observation for at least 48 hours after the accident is recommended. IF exposed or concerned: Get medical advice/attention.

4.1.1 In case of inhalation:

Move exposed person to fresh air. Keep person warm and at rest. If not breathing, give artificial respiration. If breathing is difficult,

give oxygen. If unconscious, place in recovery position and get medical attention immediately.

4.1.2 In case of skin contact:

Flush contaminated skin with plenty of soap and water. Remove contaminated clothing and shoes. If symptoms persist, call a physician.

4.1.3 In case of eyes contact:

Immediately flush eyes with plenty of water, occasionally lifting the upper and lower eyelids. Check for and remove any contact lenses. Continue to rinse for at least 15 minutes. Get medical attention if irritation occurs.

4.1.4 In case of ingestion:

Wash mouth out with plenty of water. If swallowed, do not induce vomiting - seek medical advice

4.2 Most important symptoms and effects, both acute and delayed

Vapours may cause irritation to the eyes, respiratory system and the skin. Gastrointestinal discomfort. Respiratory disorder.

4.3 Indication of any immediate medical attention and special treatment needed

In case of lung irritation first treatment with dexametason aerosol (spray). In case of choking, administration of activated charcoal and a saline laxative agent. In the case of absorption of large volumes, use gastroscopy with suction cleaning

Section 5 Fire-Fighting measures

5.1 Extinguishing media:

Suitable extinguishing media: Use foam, carbon dioxide (CO2) and extinguishing powder.

Unsuitable extinguishing media: Water spray.

5.2 Special hazards arising from the substance or mixture: Under conditions giving incomplete combustion, hazardous gases produced may consist of carbon monoxide, carbon dioxide (CO2). Combustion gases of organic materials must in principle be graded as inhalation poisons.

5.3 Special fire fighting methods and special protective actions for fire-fighters: Self-contained breathing apparatus (EN 133)

Section 6 Accidental release measures

6.1 Personal precautions, protective equipment and emergency procedures:

6.1.1 For non-emergency personnel: Provide adequate ventilation. Avoid inhalation of vapour or dust. Avoid skin and eye contact. Refer to section 8 of SDS for personal protection details.

6.1.2 For emergency responders: Wear an appropriate respirator.

6.2 Environmental Precautions: Avoid dispersal of spilt material and runoff and contact with soil, waterways, drains and sewers. Do not allow material to be released to the environment without proper governmental permits.

6.3 Methods for Containment and Cleaning up: Ensure adequate ventilation. Absorb with liquid-binding material (sand, diatomite, acid binders, universal binders, sawdust). Send for recovery or disposal in suitable containers. Dispose of the material collected according to regulations.

6.4 Reference to other sections: See Section 7 for information on safe handling.
See section 8 for information on personal protection equipment.
See Section 13 for information on disposal.

6.5 Additional information: Not applicable.

Section 7 Handling and storage

7.1 Precautions for safe handling:

7.1.1 Protective measures: Ensure good ventilation/exhaustion at the workplace. Ensure good interior ventilation, especially at floor level. Restrict the quantity stored in the work place. Prevent formation of aerosols. Do not inhale vapours/aerosols. Avoid contact with skin and eyes.

7.1.2 Advice on general occupational hygiene: Do not eat, drink and smoke in work areas. Wash hands after use. Remove contaminated clothing and protective equipment before entering eating areas. Use explosion-proof apparatus, fittings and spark-proof tools.

7.2 Conditions for safe storage, including any incompatibilities: Store in cool location. Observe regulations for storage of flammable liquids. Store away from oxidizing agents. Store in accordance with local regulations. Store in a dry place. Keep container tightly closed and sealed until ready for use.

7.3 Specific end use(s): Not applicable.

Section 8 Exposure Controls/Personal Protection

8.1 Control parameters:

8.1.1 Occupational exposure limits: Not available

8.1.2 Additional exposure limits under the conditions of use: Not available

8.1.3 DNEL/DMEL and PNEC-Values:

DN(M)ELs for workers

Exposure pattern	Route	Descriptor	DNEL / DMEL	(Corrected) Dose descriptor *)	Most sensitive endpoint	Justification
Acute - systemic effects	Dermal	No-threshold effect and/or no dose-response information available				A DNEL for acute toxicity should be derived if an acute hazard leading to acute toxicity (e.g. C&L) has been identified and there is a potential for high peak exposures. These "peaks" are normally associated with inhalation exposure, but are less common for skin contact and ingestion (Appendix R.8-8). Vinyl acetate does not present an acute hazard following skin contact and therefore no DNEL is proposed.
Acute - systemic effects	Inhalation	DNEL (Derived No Effect Level)	35.2 mg/m³	NOAEC: 35.2 mg/m³ (based on AF of 1)	irritation (respiratory tract)	A DNEL for acute toxicity should be derived if an acute hazard leading to acute toxicity (e.g. C&L) has been identified and there is a potential for high peak exposures. These "peaks" are normally associated with inhalation exposure, but are less common for skin contact and ingestion (Appendix R.8-8). Vinyl acetate does not present an acute hazard following ingestion or skin contact. It is, however, classified as harmful via inhalation exposure under DSD (Xn, R20) and therefore consideration of an acute DNEL(inhalation) is required. Since a STEL of 10 ppm (35.2 mg/m^3) has been agreed for vinyl acetate based on the reported threshold for irritancy in humans (SCOEL, 2005) this is used for the

						DNELacute (inhalation) value.
Acute - local effects	Dermal	No-threshold effect and/or no dose-response information available				Vinyl acetate is not classified for skin or eye irritation. Therefore, no DNEL is needed to be derived for this endpoint.
Acute - local effects	Inhalation	DNEL (Derived No Effect Level)	35.2 mg/m³	NOAEC: 35.2 mg/m³ (based on AF of 1)	irritation (respiratory tract)	The EU Technical Committee on Classification and labelling (TC C&L) indicated that vinyl acetate is irritating to the respiratory (Xi, R37) tract hence appropriate Risk Management Measures and Operational Conditions should be employed. The STEL of 10 ppm (35.2 mg/m^3) agreed for vinyl acetate is considered to be protection for local irritation effects.
Long-term - systemic effects	Dermal	DNEL (Derived No Effect Level)	0.42 mg/kg bw/day	NOAEL:	carcinogenicity	Dermal repeat dose and carcinogenicity studies are not available. It is of note that the high vapour pressure of vinyl acetate leads to low retention time on the skin (RAR, 2008). Dose descriptor The dermal NOAEL can be extrapolated from the IOELV [8 hr TWA, 5 ppm (17.6 mg/m^3)]. The IOELV is adjusted for differences in uptake between the two routes of exposure (TGD, Appendix R.8-2, Example B.4). It is assumed that uptake of vinyl acetate after inhalation is 15% while dermal absorption is 90% (as concluded in the RAR (2008)) corrected Dermal NOAEL = IOELV x wRV (human 8hr) x [ABSinhal-human/ABSdermal-human] corrected Dermal NOAEL = 17.6 mg/m^3 x 0.144 x [15%/ 90%] corrected Dermal NOAEL = 0.42 mg/kg bw/d No assessment factor is necessary
Long-term - systemic effects	Inhalation	DNEL (Derived No Effect Level)	17.6 mg/m³	NOAEC:	carcinogenicity	The IOELV will be used.
Long-term - local effects	Dermal	No-threshold effect and/or no dose-response information available				Vinyl acetate is not classified for skin irritation. Therefore, no DNEL is needed to be derived for this endpoint.
Long-term - local effects	Inhalation	DNEL (Derived No Effect Level)	17.6 mg/m³	NOAEC:	irritation (respiratory tract)	The DNELlong-term (local inhalation) is derived from the IOELV which is based on the NOAEC for local and systemic effects. The value of 17.6 mg/m^3 will be used for

						DNEL for long-term local and systemic effects.

**) The (corrected) dose descriptor starting points have been automatically calculated by multiplying the values of the fields "D(N)MEL" and "Assessment factor" provided in the Endpoint summary of IUCLID section 7. Toxicological information. It reflects the value after any corrections, e.g. route-to-route extrapolation. See column "Justification" for the rationale behind such modifications and the use of assessment factors.*

DN(M)ELs for the general population

Exposure pattern	**Route**	**Descriptor**	**DNEL / DMEL**	**(Corrected) Dose descriptor *)**	**Most sensitive endpoint**	**Justification**
Acute - systemic effects	Dermal	Exposure based waiving				see discussion
Acute - systemic effects	Inhalation	Exposure based waiving				see discussion
Acute - systemic effects	Oral	Exposure based waiving				see discussion
Acute - local effects	Dermal	Exposure based waiving				see discussion
Acute - local effects	Inhalation	Exposure based waiving				see discussion
Long-term - systemic effects	Dermal	Exposure based waiving				see discussion
Long-term - systemic effects	Inhalation	Exposure based waiving			irritation (respiratory tract)	see discussion
Long-term - systemic effects	Oral	Exposure based waiving		NOAEL:	repeated dose toxicity	see discussion
Long-term - local effects	Dermal	Exposure based waiving				see discussion
Long-term - local effects	Inhalation	Exposure based waiving				see discussion

**) The (corrected) dose descriptor starting points have been automatically calculated by multiplying the values of the fields "D(N)MEL" and "Assessment factor" provided in the Endpoint summary of IUCLID section 7. Toxicological information. It reflects the value after any corrections, e.g. route-to-route extrapolation. See column "Justification" for the rationale behind such modifications and the use of assessment factors.*

PNEC

PNEC	Value	Assessment factor	Remarks/Justification
PNEC $_{\text{aqua - freshwater}}$ (mg/L)	0.016	10	Extrapolation method: assessment factor Reliable short term studies are available for algae and Daphnia and valid NOECs are available for algae and fish. The acute data indicates that algae and Daphnia have approximately the same sensitivity, and that the Acute:Chronic ratio (ACR) for the algal effects is approx 2. Applying a conservative ACR to the Daphnia experimentally derived acute value would give an approximate predicited NOEC of 1 mg/l. The long-term NOEC for fish is 0.16

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<td>mg/l which is a further 10 fold lower than that predicted for Daphnia and so it is reasonable to assume that fish is the most sensitive trophic level.

To further support this assumption, a QSAR (based on the ecosar class vinyl/allyl esters) has been generated using EPI suite, the acute and chronic results are lowest for fish – see table below for a summary. Fish are predicted to be the most sensitive and an ACR of 14 is calculated for both fish and daphnia. Applying a further conservative ACR of 20 to the Daphnia experimental acute value would give a chronic data point for the invertebrates of 0.6 mg/l. Again this is higher (factor 4) than that of the fish.

All the available information indicates that fish is the most sensitive organism of those tested and that an Aplication Factor of 10 can be supported.

The NOEC reported in the fish study was not based on the Time Weighted Average, recommended in the OECD test guidelines, and therefore the NOEC has been recalculated, using the raw data provided in the report to give a NOEC of 0.16mg/l. The 34d NOEC for fish of 0.16 mg/l is the most conservative value available and therefore this has been used to derive the PNECaqua of 0.016 mg/l.

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<tr><th>Species</th><th>Acute</th><th>Chronic</th><th>ACR</th></tr>
<tr><td colspan="4">Measured data (all mg/l)</td></tr>
<tr><td>Algae (growth rate)</td><td>12.7</td><td>5.96</td><td>~ 2</td></tr>
<tr><td>Invertebrates (Daphnia)</td><td>12.6</td><td></td><td></td></tr>
<tr><td>Fish</td><td></td><td>0.16</td><td></td></tr>
<tr><td colspan="4">QSAR (all mg/l)</td></tr>
<tr><td>Algae</td><td>4.5</td><td>0.8</td><td>~ 6</td></tr>
<tr><td>Invertebrates (Daphnia)</td><td>15</td><td>1.1</td><td>~ 14</td></tr>
<tr><td>Fish</td><td>1</td><td>0.07</td><td>~ 14</td></tr>
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<td>PNEC _{aqua -marine water} (mg /L)</td>
<td>0.0016</td>
<td>100</td>
<td>Extrapolation method: assessment factor
Reliable short term studies are available for algae and Daphnia and valid NOECs are available for algae and fish. The acute data indicates that algae and Daphnia have approximately the same sensitivity, and that the Acute:Chronic ratio (ACR) for the algal effects is approx 2. Applying a conservative ACR to the Daphnia experimentally derived acute value would give an approximate predicited NOEC of 1 mg/l. The long-term NOEC for fish is 0.16 mg/l which is a further 10 fold lower than that predicted for Daphnia and so it is reasonable to assume that fish is the most sensitive trophic level.</td>
</tr>
</table>

			To further support this assumption, a QSAR (based on the ecosar class vinyl/allyl esters) has been generated using EPI suite, the acute and chronic results are lowest for fish – see table below for a summary. Fish are predicted to be the most sensitive and an ACR of 14 is calculated for both fish and daphnia. Applying a further conservative ACR of 20 to the Daphnia experimental acute value would give a chronic data point for the invertebrates of 0.6 mg/l. Again this is higher (factor 4) than that of the fish. On this basis an assessment factor of 100 has been used.
PNEC $_{\text{aqua - intermittent releases}}$ (mg /L)	0.126	100	Extrapolation method: assessment factor The intermittent release PNEC is derived using the lowest L(E)C50 which was measured for Daphnia (EC50 of 12.6 mg/l). The assessment factor used was 100 as, in accordance with the guidance, this factor is acceptable when the L(E)C50 used is the lowest of at least three short-term tests from three trophic levels.
PNEC $_{\text{fresh water sediment}}$ (mg/kg sediment dw)	0.067		Extrapolation method: partition coefficient No measured data are available for sediment and therefore the PNEC was derived using equilibrium partitioning using the equation referenced in ECHA CSA Guidance Chapter R.10: Dose –response evaluation. The fresh water PNEC of 0.016 and the calculated Koc of 5.583 were used in this equation. The PNEC derived is only an indicative PNEC.
PNEC $_{\text{marine-sediment}}$(mg/kg sediment dw)	0.0067		Extrapolation method: partition coefficient No measured data are available for sediment and therefore the PNEC was derived using equilibrium partitioning using the equation referenced in ECHA CSA Guidance Chapter R.10: Dose –response evaluation. The fresh water PNEC of 0.0016 and the calculated Koc of 5.583 were used in this equation. The PNEC derived is only an indicative PNEC.
PNEC $_{\text{soil}}$(mg/kg soil dw)	0.0035		Extrapolation method: partition coefficient No measured data are available for sediment and therefore the PNEC was derived using equilibrium partitioning using the equation referenced in ECHA CSA Guidance Chapter R.10: Dose –response evaluation. The fresh water PNEC of 0.016mg/l, the calculated Koc of 5.583 and the calculated Henry's Law constant of 51.6 Pa/m3/mol were used in this equation. The PNEC derived is only an indicative PNEC.
PNEC stp(mg/L)	6	1	Extrapolation method: assessment factor An EC3-value of 6 mg/l is reported for *Pseudomonas putida*, this result can be considered as a NOEC-value for microorganisms. An assessment factor of 1 is considered suitable for this type of test.
PNEC oral(mg/kg food)			This substance does not represent a risk of secondary poisoning and is not classified as "Toxic" or "Harmful" with at least R48 or R60-R64. Therefore this PNEC does not need to be derived.

8.2 Exposure controls

8.2.1Appropriate engineering controls: Handle in accordance with good industrial hygiene and safety practice. Ventilation: Use

only with adequate ventilation.

8.2.2 Individual protection measures, such as personal protective equipment：

Eye/face protection:	Use chemical goggles. If exposure causes eye discomfort, use a full-face respirator. Use chemical goggles. Chemical goggles should be consistent with EN 166 or equivalent.
Hand protection:	Use chemical resistant gloves classified under Standard EN374. Recommended thickness of the material: ≥0.5 mm. Penetration time: ≥2 hours
Body protection:	Personal protective equipment for the body should be selected based on the task being performed and the risks involved and should be approved by a specialist before handling this product. Recommended: protective clothing.
Respiratory protection:	Use a properly fitted, air-purifying or air-fed respirator complying with an approved standard if a risk assessment indicates this is necessary. Respirator selection must be based on known or anticipated exposure levels, the hazards of the product and the safe working limits of the selected respirator.
Thermal hazards:	Wear suitable protective clothing to prevent heat.
8.2.3 Environmental exposure controls:	Avoid discharge into the environment. According to local regulations, Federal and official regulations.

Section 9 Physical and chemical properties

9.1 Information on basic physical and chemical properties

Appearance	liquid
Melting point/range (°C):	179.7 K at 1013 hPa
Boiling point/range (°C):	345.85 K at 1013 hPa
Flash point (°C):	265 K at 1013 hPa
Self-ignition temperature:	675.2 K at 1013 hPa
Vapour pressure (25°C):	113 hPa at 20˚C.
Relative Density:	932 at 20°C
Water solubility (g/l):	20000 mg/L at 20 °C
n-Octanol/Water (log Po/w):	0.73 at 20 °C
Viscosity:	Viscosity at 20°C: 0.42 mPa s (dynamic)
Surface tension:	Not available
Dissociation constant in water(pKa):	Not available

9.2. Other information:

Explosive properties :	Non explosive
Flammability:	highly flammable
Oxidising properties :	No
Granulometry :	Not available
Stability in organic solvents and identity of relevant degradation products :	Not available

Section 10 Stability and reactivity

10.1 Reactivity:	The substance is stable under normal storage and handling conditions.
10.2 Chemical stability:	Stable at room temperature in closed containers under normal storage and handling conditions.
10.3 Possibility of hazardous reactions:	Polymerization can occur. May polymerize violently or explosively if contaminated or overheated. Uncontrolled polymerization can cause rapid evolution of heat and

increased pressure which can result in violent rupture of storage vessels or containers.

10.4 Conditions to avoid: Avoid any source of ignition. Avoid contact with heat, sparks, open flame, and static discharge. Avoid temperatures above 30 °C/ 86 °F

10.5 Incompatible materials: Oxidizing agents, radical initiators, strong acids, amines.

10.6 Hazardous decomposition products: Carbon monoxide, carbon dioxide (CO2)

Section 11 Toxicological information

11.1 Toxicokinetics, metabolism and distribution

Introduction

The toxicokinetics of vinyl acetate have recently been reviewed in detail under the EU Existing Substances Regulations (EU RAR, 2008). This short summary extracts from that publicly available review including additional critical toxicokinetic information that supports the risk assessment of vinyl acetate.

Mode of Action Context for Vinyl Acetate Toxicokinetics

The toxicity and carcinogenicity of vinyl acetate is related to its extensive, rapid metabolic conversion to acetaldehyde and acetic acid, with concomitant production of tissue-acidifying protons. Metabolic clearance of acetaldehyde and acetic acid, as well as processes controlling cellular pH serve to reduce tissue exposure to these metabolites, providing some protection against toxicity. These metabolic processes occur in most tissues, including those at the points of contact for inhalation and oral exposures, generally leading to higher exposures to the metabolites in these tissues and lower systemic exposure. Thus, it is important to consider not just the kinetics of vinyl acetate itself, but the kinetics and exposure-tissue dose relationships for the metabolites, especially in the portal of entry tissues such as the oral cavity and nose, the target tissues for vinyl acetate. Further information on acetaldehyde and acetic acid can be obtained from reports e. g. prepared by the German MAK commission.

Overview of Toxicokinetic Data

The toxicokinetics of vinyl acetate in the nasal tissues is well described, with more limited datasets for oral and dermal exposure. The toxicokinetic database includes information on:

• Respiratory uptake and estimated systemic bioavailability following inhalation exposure;

• The rapid metabolic conversion of vinyl acetate to acetic acid and its effect on intracellular pH;

• The identification, activity and distribution of the enzymes involved in the metabolism of vinyl acetate and its metabolites acetaldehyde and acetic acid;

• Systemic bioavailability after oral or dermal exposure;

• Tissue distribution of vinyl acetate and/or metabolites;

• The preparation and experimental validation of PBPK models in support of risk assessment; and,

• Absorption values and scaling factors for risk assessment recommended in the EU RAR.

A summary containing the key toxicokinetic data and related conclusions supporting the risk assessment of vinyl acetate is provided below. The data are organized by the conventional absorption, distribution, metabolism and elimination scheme, followed by a section on the integration of the toxicokinetic data within PBPK models.

Absorption

During exposure of rats by inhalation, the degree of uptake of vinyl acetate in the upper respiratory tract was found to be inversely

proportional to atmospheric concentrations falling from 94% at 76 ppm to about 40% at 550 ppm; uptake then remained approximately constant at concentrations up to 2000 ppm (Plowchalk et al., 1997). For risk characterization of systemic effects, systemic bioavailability of intact vinyl acetate during inhalation exposure was reported to be controlled by blood flow extraction and was estimated to be <15% of total uptake (Plowchalk et al., 1997; Bogdanffy et al., 1998). A value of 15% for uptake during inhalation is recommended as a worst case scenario for risk assessment (EU RAR, 2008).

At higher concentrations (>76 ppm), where upper respiratory extraction is significantly reduced, or where oral breathing may predominate, a higher fraction, up to 60% (100%-40%) of material may pass to the conducting airways and pulmonary region, possibly increasing systemic exposure. Nonetheless, to the extent that the lower respiratory tract epithelium contains carboxylesterases, only a fraction of vinyl acetate extracted from the inhaled air would pass to the systemic blood. In addition, it should be kept in mind that vinyl acetate can be degraded in the blood with half lives between < 1 min and 4.1 min, further limiting systemic exposure to vinyl acetate (Fedtke and Wiegand, 1990).

Value used for CSA: Respiratory uptake (%): 15

After oral administration of 297 mg/kg/body weight [14C] vinyl acetate, 63 % of the applied radioactivity was excreted as metabolites in exhaled air (61.2%), and urine (1.8%). The carcass contained 5.4% of the administered radioactivity 96 hours after the last dose. The authors believed that the remaining 30% of the administered radioactivity not retrieved in the mass balance was eliminated as [14] CO_2 in exhaled air, but lost during the opening and closing of the chambers during the repeated dosing exercise (Hazleton, 1980a). A significant loss to exhaled air is consistent with the time course for elimination in exhaled air following oral dosing, since elimination was found to be rapid and greatest during the 6 hour dosing period. In addition, the authors reported finding only [14C] as CO_2, indicating that the [14C] in exhaled air was solely derived from absorbed, metabolized vinyl acetate. Summing the measured amount eliminated in exhaled air, the amount eliminated in urine, and the amount in the carcass gives a lower bound on the percent absorbed of 68.4%. By adding the 30% which was likely eliminated in exhaled air but lost to analysis in opening the chambers, an approximate upper bound on vinyl acetate absorption of 98.5% can be estimated for the oral route. The amount in faeces (1.4%) is not included because this material may not have been absorbed. The oral dosing studies provided no information on the chemical identity of radiolabeled material(s) absorbed. Thus, though 68.4-98.5% of the orally administered, radiolabeled carbon was absorbed, no firm conclusions can be made regarding whether the material was vinyl acetate or one of its metabolites. However, systemic exposure to vinyl acetate itself following oral exposure would likely be considerably less than the high fraction of radiolabelled material absorbed. Vinyl acetate can be metabolized in the upper GI tract epithelium, it can therefore be assumed that a considerable amount of metabolism takes place presystemically, limiting systemic exposure to vinyl acetate.

There are no valid quantitative data on the systemic bioavailability of vinyl acetate and its metabolites following dermal exposure. Dermal uptake would depend on the balance between absorption, metabolism in the skin, and loss to volatilization, which would likely be extensive for vinyl acetate, which has a high vapour pressure (12000 Pa, EU RAR, 2008). Acute toxicity observed in rabbits following dermal exposure to 4-16 ml/kg of vinyl acetate (Mellon-Institute 1969) is an indication that systemic exposure occurs following dermal exposure, but the extent of exposure cannot be determined. A default or worst case assumption of 90% absorption during risk assessment was recommended (EU RAR, 2008).

Distribution

Exposure of rodents by either inhalation or oral administration to vinyl acetate labelled with [14C] in the vinyl group resulted in a wide distribution of radioactivity throughout the tissues (Hazleton, 1980). This is to be expected for highly absorbed water soluble compounds and their metabolites, particularly when major metabolites (acetaldehyde, acetate) enter the two-carbon metabolic cycle (e. g. acetaldehyde, acetate). Following inhalation by rats, vinyl acetate derived radioactivity is widely distributed with high levels (relative to the total amount detected in the body) in the liver, kidney, lung, brain, stomach, colon, ovaries, harderian gland, ileum, submaxillary salivary gland and the GI tract contents (Hazleton, 1980a). Similarly, following oral exposure to rats, high concentrations were found in the harderian gland, submaxillary salivary gland, liver, kidney stomach, ileum, colon and gastrointestinal tract contents (Hazleton, 1980a).

Metabolism

Vinyl acetate is hydrolysed by carboxylesterases to acetic acid and acetaldehyde, producing protons in the process. Acetaldehyde is oxidised to acetic acid (acetate) by aldehyde dehydrogenases. The resulting acetate enters the citric acid cycle as acetyl-coenzyme A (Bogdanffy et

al., 1998). The tissue distribution and activity of the major enzymes responsible for the metabolism of vinyl acetate, acetaldehyde and acetate are important factors in the disposition and toxicity of vinyl acetate.

The presence of carboxylesterases capable of hydrolysing vinyl acetate with high efficiency has been demonstrated in the epithelial tissues of the nose, oral cavity, and respiratory tract, as well as the skin, blood and liver of several species including man (Hazleton, 1980a; Simon et al., 1985; Bogdanffy and Taylor, 1993; Bogdanffy et al., 1998; Morris et al., 2002). Carboxylesterases present in tissues lining the major portals of entry following inhalation, oral and dermal exposure, lead to pre-systemic hydrolysis of vinyl acetate at these sites, reducing systemic exposure to intact vinyl acetate to a degree in proportion with enzyme activity, i. e. where enzyme activity is highest, systemic exposure will be lowest and visa-versa. Conversely, the presence of carboxylesterases at a portal of entry can increase local concentrations of vinyl acetate metabolites by increasing the air-tissue flux of vinyl acetate.

Aldehyde dehydrogenase (ALDH) is widely distributed and can be found in many tissues of experimental animals including mice, rats, hamsters and guinea pigs. In all species except guinea pig, data supports the presence of two isozymes characterised by high and low affinity forms (Morris, 1997). Similar enzyme activity has been obtained for human nasal and oral cavity tissues; additionally, ALDH activity has been demonstrated in tissue from the human oesophagus and stomach and in saliva (Dong et al., 1996; Yin et al., 1997; Bogdanffy et al., 1998). ALDH activity is present at major sites of inhalation, oral and dermal exposure to vinyl acetate. At exposure levels that do not exceed the capacity of ALDH to oxidise acetaldehyde to acetic acid, there will be proportionally lower local exposure to acetaldehyde, but higher production of acetic acid. Local elevations of acetic acid may cause modest, transient reductions in intracellular pH, but the toxicological significance of this effect is unresolved (EU RAR, 2008). Systemically available acetate is incorporated into the citric acid cycle, ultimately being incorporated into endogenous substances or eliminated as CO_2. At higher exposures, the capacity and protective action of ALDH will be exceeded and intracellular accumulation of acetaldehyde will occur, raising tissue levels above a threshold for local toxicity above which tumour formation may occur.

Human variability in ALDH capacity, related to polymorphisms in the high affinity form of the enzyme has been reported. The contribution of this enzyme to total acetaldehyde metabolism is believed to be relatively small (Teeguarden et al., 2008). Nonetheless, where polymorphisms were shown to increase tissue acetaldehyde following vinyl acetate exposure, a pharmacokinetic case for higher risks in this population could be made. The converse is also true. If ALDH polymorphisms reduce acetaldehyde metabolism and subsequently reduce acetic acid levels, the polymorphism could be protective. At this time, an intraspecies adjustment factor of 10 is recommended due to uncertainties in the mode of action for vinyl acetate toxicity.

More specifically, local metabolism was studied in human and rat nasal respiratory and olfactory tissue with whole turbinates in vitro (Bogdanffy et al., 1998). The studies indicated species differences of nasal respiratory carboxylesterase activities between rats and humans. Comparing enzyme activity on a per epithelial cell volume basis, rat respiratory carboxylesterase and ALDH activities were about three and two times higher than those of humans, respectively. Activities of the rat olfactory enzymes (carboxylesterase and ALDH) were about equivalent to those of humans. The Km values for both enzymes are not different between the two species. These species differences in metabolism are reflected in the vinyl acetate physiologically based pharmacokinetic model (Hinderliter et al., 2005). ALDH activities determined in whole nasal tissue homogenates from mouse, rat, hamster and guinea pig showed significantly different ratios of Vmax/Km (intrinsic clearance) for the various species indicating the existence of species differences (Morris, 1997)

Vinyl acetate hydrolysis has been studied in vitro in the oral mucosal tissues from the oral cavity of rats and mice. The hydrolysis activity of the oral tissues is at least 100-fold lower than that of the nasal tissues.

Excretion

In rats, radioactivity derived from inhaled [14C]-vinyl acetate was rapidly eliminated in expired air, urine and faeces. The radioactivity in expired air was present as CO_2 and acetaldehyde; concentrations of acetaldehyde increased as vinyl acetate exposure was increased, suggesting saturation of acetaldehyde metabolism (Plowchalk et al., 1997). Higher concentrations of acetaldehyde are expected following inhalation, compared with oral exposure, because in the former, acetaldehyde is generated in tissues lining the respiratory tract and equilibrates with exhaled air. This explains differences in the amount of exhaled acetaldehyde reported by Hazleton (1980a), for oral exposure and Plowchalk (1997) for inhalation exposure. Radioactivity from orally administered [14C]-vinyl acetate was also rapidly eliminated predominantly as CO_2 in exhaled air along with small amounts of radiolabel in both urine and faeces (Hazleton, 1980a). [13C]-Acetaldehyde

has been measured in exhaled air from human volunteers exposed by inhalation to [13C]-vinyl acetate (Hinderliter et al., 2005). Overall, these data support the conclusion that the principle route of elimination for absorbed vinyl acetate is through metabolism, leading to exhalation of the metabolite acetaldehyde and metabolically derived CO_2 and acetaldehyde.

Physiologically based pharmacokinetic (PBPK) models

A physiologically based pharmacokinetic model was developed which describes the deposition of vinyl acetate vinyl acetate in the nasal cavity of the rat. This model predicts steady state concentrations of the metabolite acetic acid after continuing 6 h-exposure in respiratory tissue that are approximately 2.4-5.3 times greater than in olfactory tissue at concentrations between 200 and 1000 ppm, but 46 times greater at the lowest bioassay concentration of 50 ppm. In the olfactory epithelium, acetaldehyde concentrations are approximately half those of acetic acid, except at the 50 ppm exposure level where they are twice the acetic acid concentration. Acetaldehyde concentrations are approximately 10 -fold lower than acetic acid concentrations in the dorsal and ventral epithelium (Plowchalk et al., 1997). As the concentration of acids is indicative of the local metabolism to acetate and increased concentration of protons, the model predicts the greatest reduction in intracellular pH for respiratory mucosa (Plowchalk et al., 1997). Hence, pH effects should be more pronounced in this tissue as compared to other nasal or systemic tissues. This physiologically based toxicokinetic/toxicodynamic model for rat was modified for the olfactory and respiratory epithelium of humans. The change in intracellular pH is predicted to be slightly greater for human compared to rat olfactory epithelium at concentrations above 50 ppm vinyl acetate, but slightly less than the rat at 50 ppm. To provide validation data for the human vinyl acetate PBPK model, controlled human exposures at exposure levels of 1, 5 and 10 ppm to inhaled vinyl acetate were conducted (Hinderliter et al., 2005). Air was sampled by a probe inserted into the nasopharyngeal cavity of five volunteers at bi-directional breathing through the nose. Data from ion trap mass spectrometry measurements of labeled vinyl acetate and acetaldehyde were compared with data from the human nasal model simulation. For the vinyl acetate data, a good fit was demonstrated (r = 0.9). Acetaldehyde data are fitted with a somewhat lower precision. The results show that the human nasal model predicts the experimental observations with regard to vinyl acetate concentrations and the acetaldehyde washout in the airstream of human nasopharyngeal cavity in a concentration range from 1 to 10 ppm. Though there are some uncertainties in the enzyme kinetic data used to establish the model, the PBPK model currently represents the best integration of the available data on the kinetics of vinyl acetate, acetaldehyde and acetate metabolism and well as other physiological processes affecting nasal tissue dosimetry of these compounds. Thoughtful consideration of the PBPK models strengths and limitations is appropriate when the outputs are applied in risk assessment.

Use of the validated PBPK model for cross species extrapolation of nasal tissue dosimetry can replace the standard interspecies adjustment factor for pharmacokinetic differences (3), reducing the interspecies uncertainty factor from 10 to 3. Because pharmacodynamic differences between species cannot be ruled out, and since local metabolism will limit systemic absorption and metabolism, the total interspecies adjustment factor of 3 appears justified and should be applied in a risk assessment.

Due to recognized human polymorphisms in ALDH activity, an intraspecies adjustment factor of 5 or 10 (worker or general population, respectively) is recommended to account for uncertainties in ALDH activity in mode of action for vinyl acetate toxicity.

Dermal absorption

Although the absorption of vinyl acetate following dermal exposure has not been directly measured, systemic toxicity was observed in rabbits exposed dermally to very high dose levels (4-16ml/kg), suggesting some absorption of vinyl acetate or its metabolites (Mellon Institute, 1969).

The following information is taken into account for any hazard / risk assessment:

A value of 90% absorption during risk assessment was recommended (EU RAR, 2008).

Value used for CSA: Absorption rate (%): 90

11.2 Information on toxicological effects

Acute toxicity:

LD50(Oral, Rat):	3500 mg/kg bw
LD50(Dermal, Rabbit):	7440 mg/kg bw

LC50(Inhalation, Rat): 15810 mg/m³ air

Skin corrosion/Irritation: May cause respiratory irritation.

Serious eye damage/irritation: Not classified

Respiratory or skin sensitization: Not sensitizing

Germ cell mutagenicity: Negative

Carcinogenicity: Suspected of causing cancer. Route of exposure: Oral

Reproductive toxicity: Not classified

STOT- single exposure: May cause respiratory irritation. Affected organs: respiratory tract. Route of exposure: Inhalation

STOT-repeated exposure: Not classified

Aspiration hazard: Not classified

Section 12 Ecological information

Toxicity:

Acute toxicity		Time	Species	Method	Evaluation	Remarks
LC50	N/A	96h	Fish	OECD 203	N/A	N/A
EC50	12.6 mg/L	48h	Daphnia	OECD 202	N/A	1 (reliable without restriction) key study experimental result
EC50	12.7 mg/L	96h	Algae	OECD 201	N/A	1 (reliable without restriction) key study experimental result

Persistence and degradability: readily biodegradable

Bioaccumulative potential: a low potential for bioaccumulation.

Mobility in soil: Not available

Results of PBT&vPvB assessment: The substance is not considered a PBT/vPvB.

Other adverse effects: Harmful to aquatic life with long lasting effects

Section 13 Disposal considerations

13.1 Waste treatment methods The material should be disposed of by incineration in a chemical incinerator in compliance with national and regional requirements.

13.2 Product / Packaging disposal: If empty container retains product residues, all label precautions must be observed. Return for reuse or dispose according to national or local regulations.

Section 14 Transport information

	Land transport(ADR/RID)	Sea transport (IMDG)	Air transport (ICAO/IATA)
UN-Number:	1301	1301	1301
UN Proper shipping name:	Vinyl Acetate, stabilized	Vinyl Acetate, stabilized	Vinyl Acetate, stabilized
Transport hazard Class:	3	3	3
Packaging group:	II	II	II
Environmental hazards:	No	No	No

Special precautions for user:	See section 2.2	See section 2.2	See section 2.2
Transport in bulk according to Annex II of MARPOL 73/78 and the IBC Code	Not Regulated	Not Regulated	Not Regulated

Section 15 Regulation information

15.1 Safety, health and environmental regulations/legislation specific for the substance or mixture

Relevant information regarding authorization: Not applicable.

Relevant information regarding restriction: Not applicable.

Other EU regulations: Employment restrictions concerning young person must be observed. For use only by technically qualified individuals.

Other National regulations: Not applicable

Chemical Safety Assessment has been carried out? YES [X] NO [ ]

Section 16 Other information

16.1 Indication of changes

Version 1.0 Amended by (EU) 2015/830

Version 2.0 Exposure scenarios are placed after section 16.

16.2 Training instructions:

Not applicable.

16.3 Further information:

This information is based upon the present state of our knowledge. This eSDS has been compiled and is solely intended for this product.

16.4 Notice to reader:

Employers should use this information only as a supplement to other information gathered by them, and should make independent judgment of suitability of this information to ensure proper use and protect the health and safety of employees. This information is furnished without warranty, and any use of the product not in conformance with this Extended Safety Data Sheet, or in combination with any other product or process, is the responsibility of the user.

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1 Exposure scenario 1. Use of vinyl acetate monomer in polymer production

1.1. Exposure scenario

Exposure Scenario 2 Use of Vinyl Acetate Monomer in Polymer Production ((co)polymerization processes of vinyl acetate monomer) – Worker and Environment	
1.1.1. Title	
Free short title	Use of Vinyl Acetate Monomer in Polymer Production ((co)polymerization processes of vinyl acetate monomer and associated laboratory activities
Systematic title based on use descriptor	SU 3: Industrial uses: Uses of substances as such or in preparations at industrial sites
Processes, tasks activities covered	Polymerisation processes in the large scale chemical industry and in downstream facilities
Assessment Method	The assessment is based on the results published in the EU RAR (2008) on vinyl acetate.
1.1.2. Operational conditions and risk management measures	
PROC 1 Closed process (no sampling) PROC 2 Closed continuous process (with sampling) PROC 3 Closed batch process (with sampling) PROC 4 Use in batch and other process (synthesis) where opportunity for exposure arises PROC 8a Transfer of chemicals from/to vessels/ large containers at non dedicated facilities PROC 8b Dedicated discharging to/from vessels PROC 15 Use as laboratory reagent ERC 6C for use as monomer (Co) polymerisation	
Processed volumes range from 280 t/a 110,000 t/a.	
1.1.2.1 Control of workers exposure	
Product characteristic	
Vinyl acetate is a highly flammable liquid. It is highly volatile leading to low retention times of the substance on the skin or on protective gloves. Revised classification will be a carcinogen category 3 material under 67/548/EEC (corresponding to class. 2 carcinogen under CLP).	
Amounts used	
Processing sites Amount used in the workplace calculated by dividing the tonnage processed per year by the number of emission days (300). Data from 27 sites. Range from 0.93 t/d to 367 t/d.	
Frequency and duration of use/exposure	
Use pattern is daily and for the entire length of the shift (8h) Frequency of exposure (annual) < 240 Days/year Frequency of exposure (weekly) > 4 Days/week Duration of exposure > 4 Hours/day	
Human factors not influenced by risk management	
In spite of the use of protective gloves, dermal exposure may occur due to e.g. unintended contamination during the handling of used gloves.	
Other given operational conditions affecting workers exposure	

Polymerisation takes place by batch or continuous processing in a closed system.
Inhalation and dermal exposure is possible during activities such as sampling, transfer, drumming, filter changing, and cleaning, maintenance and repair work.

Technical conditions and measures at process level (source) to prevent release

(Recommended RMMs representing good practice beyond REACH requirements are indicated in blue.
Standard Phrases and Codes used below are available from the CEFIC Worker Template at the CEFIC Website.)

General exposures (closed systems). Continuous process. No sampling.
General exposures (closed systems).Continuous process. No sampling. Handle substance within a closed system. {Wear suitable gloves tested to EN374 }.
Bulk transfers. Transport. with sample collection. Ensure material transfers are under containment or extract ventilation. Clear transfer lines prior to de-coupling. {Wear suitable gloves tested to EN374 }.
Polymerisation (bulk and batch). Continuous proces. Batch process. With sample collection. Handle substance within a closed system. Ensure material transfers are under containment or extract ventilation. {Wear suitable gloves tested to EN374 }.
Finishing operations. Batch proces. With sample collection. General process exposures: finishing section (catalyst inactivation and removal of unreacted monomers). Handle substance within a closed system. Ensure material transfers are under containment or extract ventilation. {Wear suitable gloves tested to EN374 }.
Finishing operations.Batch process. With sample collection. General process exposures: Finishing Washing/ Sparging. Handle substance within a closed system. Ensure material transfers are under containment or extract ventilation. . {Wear suitable gloves tested to EN374 }.
Laboratory activities: Handle in a fume cupboard or under extract ventilation {Provide a good standard of general or controlled ventilation (5 to 10 air changes per hour) } {Wear suitable gloves tested to EN374. }
Intermediate polymer storage. Handle substance within a closed system. Provide extract ventilation to points where emissions occur. {Clear transfer lines prior to de-coupling }. {Wear suitable gloves tested to EN374 }
Additivation and stabilisation. Handle substance within a closed system. Ensure material transfers are under containment or extract ventilation. {Wear suitable gloves tested to EN374 }
Mixing and blending. Batch process. Provide extract ventilation to points where emissions occur. {Wear suitable gloves tested to EN374 }
Bulk transfers. Continuous process. with sample collection. Bulk (silo) storage and transfer of pellets. Limit the substance in product to 1 %. Ensure operation is undertaken outdoors. {Wear suitable gloves tested to EN374 }
Transpo. with sample collection. Ensure material transfers are under containment or extract ventilation. {Wear suitable gloves tested to EN374 }
Equipment maintenance. Wear chemically resistant gloves (tested to EN374) in combination with 'basic' employee training. Clear up spills immediately and dispose of waste safely. Drain down system prior to equipment break-in or maintenance. Ensure operatives are trained to minimise exposures. {Only allow access to authorised staff }.

Technical conditions and measures to control dispersion from source towards the worker

Inhalation exposure normally minimized by technical equipment (e.g. special designed filling stations, local exhaust ventilation)

Organisational measures to prevent /limit releases, dispersion and exposure

Good standard of occupational hygiene is implemented.
Handle substances within a closed system. Ensure material transfers are under containment or extract ventilation. Provide extract ventilation to points where emissions occur.

Conditions and measures related to personal protection, hygiene and health evaluation

Good standard of occupational hygiene is implemented.
Workers normally use PPE (gloves, eye glasses) and, during cleaning activities, respiratory protection in addition. Suitable gloves tested to EN374 are used during the activities where skin contact is possible.

1.1.2.2 Control of environmental exposure
Product characteristics
The content of residual vinyl acetate monomer after manufacture is < 2 - 3000 ppm in the different (co)polymers, it can be adjusted during manufacture depending on the further application of the (co)polymer.
Amounts used
Processing per site ranges from 0.93 t/d to 367 t/d
Frequency and duration of use
Closed system facilities, largely batch process. Pattern of release: Continuous 300 days per year A generic worst case scenario is included in the EU RAR for VAM to account for a difference between nominal production volume and verified processing capacities. The generic worst case scenario is used to estimate the fraction of release at the local main source. The estimate is based on the following: • a fraction of main source of 0.15 • a duration of emission of 300d • a waste water flow of 10,000m^3/s and a dilution of D = 40 • an emission factor to waste water of 0.02% for processing • a release fraction of 0.1% of the polymerized material to air.
Environment factors not influenced by risk management
Flow rate of receiving surface water
Other given operational conditions affecting environmental exposure
During start-up, shut-down and purification operations may result in releases to waste products. Waste product and empty containers should be disposed of as hazardous waste in accordance with all local and national regulations
Technical conditions and measures at process level (source) to prevent release
Closed systems for polymerisation and processing, however diffuse releases from flanges, pumps etc. cannot be totally excluded. Use appropriate emission abatement equipment from LEV systems if required by local legislation. Store at temperatures not exceeding 30°C. Keep tightly closed in a dry, cool and well-ventilated place. Bulk storage of vinyl acetate at ambient temperatures is an acceptable practice when there is a routine turnover of the tank contents every 60 days or less. Inhibitor levels should be monitored if a stability problem is suspected.
Technical onsite conditions and measures to reduce or limit discharges, air emissions and releases to soil
Technical measures, e.g. on-site waste water and waste treatment techniques, scrubbers, filters and other technical measures aiming at reducing releases to air, sewage system, surface water or soil; this includes strictly controlled conditions to minimise emissions;
Organizational measures to prevent/limit release from site
Best practice in environmental management
Conditions and measures related to municipal sewage treatment plant
Results from laboratory studies provide further evidence that VAM is readily biodegraded by domestic sewage effluent microorganisms both under aerobic and anaerobic conditions of sludge digesters. VAM has been demonstrated to biotransform in samples of sludge and sewage under aerobic and anaerobic conditions, both of which yielded acetaldehyde as an intermediate and acetic acid as a final product. Pre-treatment of raw waste water is common practice, e.g. by adding special zeolites or by electroflotation. The solid phase is

subsequently removed (e.g. with a filter press), while the pre-purified waste water is sent to a municipal STP for further treatment. It is expected that at least 50% (assumed) of the residual VAM in waste water is retained with the filter cake. A preliminary purification step can therefore substantially reduce the content of total organic carbon in the waste water. Regarding the site specific emissions or concentrations in waste water that were used in the exposure calculations, it is assumed that any internal pre-treatment is already reflected in the figures provided.

Conditions and measures related to external treatment of waste for disposal

Companies processing VAM reported either incineration or landfill of STP sludge.

Conditions and measures related to external recovery of waste

Diffusive releases from end-use products is considered to be a major exposure pathway, however the majority of the residual monomer material will be released in the early stages of product lifetime and therefore releases to the aquatic compartment after disposal to landfills has been assumed to be relatively low compared to production and processing point sources.

1.1.3. Exposure estimation and reference to its source

Human Health

Workers

For the occupational risk assessment of vinyl acetate the MOS approach (outlined in the TGD) has been applied.

For the assessment of inhalation exposure during the production and polymerisation in areas with high levels of protection typical of the chemical industry an 8-h time weighted average concentration of 3.0 mg/m^3 (95th percentile, round off) is taken to represent the worst case situation.

For dermal exposure for unprotected workers, the EASE model yields an exposure level of 42-420 mg/person/day. For actual dermal exposure levels factors such as the substance being manufactured and processed in a closed system and the use of PPE during exposure relevant activities are taken into account, as these are generally adopted in the large-scale chemistry industry. A protection efficiency of 90% is taken as a default value leading to an exposure level of 42mg/person/day. Also, taking into account the short retention time of vinyl acetate on the skin, it has been determined that an exposure level of 0-42 mg/person/day should be taken to represent a reasonable worst case scenario.

Estimated workplace exposures are not expected to exceed DNELs when the identified risk management measures are adopted All RCRs are <1.

Man exposed indirectly via the environment

Conclusion: at present no need for further information and/or testing and for risk reduction measures beyond those which are already applied

Human health (risks from physic-chemical properties)

Conclusion: at present no need for further information and/or testing and for risk reduction measures beyond those which are already applied

Environment

From the combined measured and estimated data it has been concluded that emission to surface water from production totaled approximately 0.486 t/a and emission to air were about 2.867 t/a

A default emission factor for waste water of 0.7% is provided in the TGD for intermediates in chemical synthesis. This has been replaced by a more specific value of 0.02% to waste water (backed up by site-specific data). The default emission factor for air of 0.1% (TGD) is considered realistic for VAM.

Exposure estimations are based on a fraction of main source of 0.15, a duration of emission of 300d, a waste water flow of 10,000m^3/s and a dilution of D = 40, an emission factor to waste water of 0.02% for processing , a release fraction of 0.1% of the polymerized material to air (as VAM).

For the local main source, a release of 9.75 t/a to waste water, of 975kg/a to surface water, and of 48.75 t/a to the atmosphere has been calculated.

For the whole processing volume of 325,000 t/a, a release of 65 t/a to waste water, of 6.5 t/a to surface water and of 325 t/a to the atmosphere has been derived.

Assuming that (co)polymers have on average a residual vinyl acetate monomer content of 3,000ppm, the diffusive emissions from end-use products during service lifetime may amount to 3,000 t/a based on a market VAM supply of roughly 1,000,000 t/a.

This was calculated to be split into 2,700 t/a assumed to be released to air, while 300 t/a are assumed to enter the aquatic environment.

Environmental PECs

Generic worst case scenario

For the difference of 325,000 t/a between nominal production volume (800,000 t/a) and verified processing capacities (total of 475,000 t/a), a generic worst case scenario was calculated based on a fraction of main source of 0.15.

The PEC local$_{water}$ was derived by adding the PECregional$_{water}$ to the calculated value of Clocal$_{water}$, with PECregional$_{water}$ being taken from Simplebox calculations

PEC_{STP}	$PEClocal_{water}$	$PEClocal_{soil}$	$PEClocal_{seawater}$
325µg/l	8.13µg/l	0.014mg/kg	0.501µg/l

No PECs for freshwater and marine sediment were generated in the EU RAR (2008). Both PEC and PNEC can be calculated using the equilibrium partitioning method, which will result in the same risk ratios as were obtained for surface water.

This approach only considers uptake of vinyl acetate via the water phase, but uptake may also occur via other exposure pathways like ingestion of sediment. These other exposure pathways are considered negligible due to the low adsorptive properties of vinyl acetate and the low affinity to organic material. In addition, VAM is degraded hydrolytically and biologically in sediments. A significant transfer of VAM into the sediment-phase is therefore not expected.

On the basis of the available information, it is concluded that an unacceptable risk for sediments and sediment dwelling organisms is generally not to be expected from vinyl acetate.

Measured data

The following PECs are based on measured data.

PEC_{STP}	$PEClocal_{water}$	$PEClocal_{soil}$	$PEClocal_{seawater}$
100µg/l	10.3µg/l	1.4E-03 mg/kg	0.501µg/l

Environment RCRs

Generic worst case scenario

RCRs generated from the generic worst case scenario developed in the EU RAR (2008) , not supported by measured environmental data

STP	Water	Soil	Seawater
0.054	0.51	4.0	0.31

The RCR soil when refined with measured data from processing sites using STP is < 1.

Measured data

RCRs generated from available data (only maximum RCR reported)

Environmental exposure	PEC	PNEC	RCR
In STP	100 µg/l	6000 µg/l	0.017
In local freshwater	10.3 µg/l	16 µg/l	0.64
In local soil	1.4E-03 mg/kg	0.0035 mg/kg	0.4
In local marine water (mg/l)	5.01E-01 µg/l	1.6 µg/l	0.31

The PNECsoil has been calculated using the PNECwater and the equilibrium partitioning method. The PNECwater reported in this report is lower than the PNECwater calculated in the EU RAR (2008) as it was necessary to re-calculate the chronic NOEC for fish. Consequently, although no new emission data has been received, the RCRs are different than those in the EU RAR (2008) due to this change in the PNECs.

In particular the PECsoil reported for Site 16 (5.16E-03 mg/kg) and the PNECsoil of 3.5E-03 mg/kg gives rise to a RCR of 1.47. Sites with operating parameters similar to this site (see the ESR for further details) and with the emission characteristics of this site (again see the ESR), including this site, will need to address the risk assessment of the soil compartment and address whether they need to implement additional RMMs or re-assess their emissions data.

The 14th Risk Reduction Strategy Meeting of the Member States for the Implementation of Council Regulation (EEC) 793/93 on the Evaluation and Control of Risks of Existing Substances endorsed the following recommendations on the environment:

"The conclusion of the assessment of the risks to the terrestrial ecosystem is that there is a need for specific measures to limit the risks. The conclusion is reached because of: concerns for the local terrestrial compartment as a consequence of exposure arising from vinyl acetate processing, more specifically for processing ((co)polymerisation) sites exceeding 20,000 t/a. Sites already applying advanced techniques would not require further consideration of risk reduction measures.

For processing sites with a processing capacity > 20,000 t/a it is recommended:

- that competent authorities in the Member States concerned should lay down, in the permits issued under Directive 2008/1/EC[1], conditions, emission limit values or equivalent parameters or technical measures regarding vinyl acetate, in order for the installations concerned to operate according to the best available techniques (BAT) taking into account the technical characteristic of the installations concerned, their geographical location and the local environmental conditions.
- that Member States should carefully monitor the implementation of BAT regarding vinyl acetate and report any important developments to the Commission in the framework of the exchange of information on BAT.
- to facilitate permitting and monitoring under Directive 2008/1/EC vinyl acetate should be included in the ongoing work to develop guidance on best available techniques (BAT).
- that local emissions to the environment should, where necessary, be controlled by national rules to ensure that no risk for the environment is expected."

1.1.4. Guidance to DU to evaluate whether he works inside the boundaries set by the ES

Guidance how the DU can evaluate whether he operates within the conditions set in the exposure scenario.

The worker exposure and environmental emissions have been evaluated on the basis of the EU RAR. If the local conditions deviate significantly from the values in the EU RAR, then further site specific evaluation is required.

Additional good practice advice beyond the REACH CSA

Note: The measures reported in this section have not been taken into account in the exposure estimates related to the exposure scenario above. They are not subject to obligation laid down in Article 37 (4) of REACH

Use specific measures expected to reduce the predicted exposure beyond the level estimated based on the exposure scenario when possible.

1.2. Exposure estimation

1.2.1. Workers exposure

A summary of relevant measured inhalation exposure data of vinyl acetate exposures at workplaces in the chemical industry during production of vinyl acetate monomer and polymerisation processes was compiled in the recently finalized EU risk assessment (2008) and is provided in the table below.

Job category / activities	Year of measure-ment	Number of samples	Range of measurement data [mg/m^3]	Geometric mean [mg/m^3]	95th-percentile [mg/m^3]	Duration and frequency
8-h time weighted average						

[1] Directive 2008/1/EC of 15 January 2008 concerning integrated pollution prevention and control, OJ L 24, 29/1/2008, p. 8–29

Job category / activities	Year of measure-ment	Number of samples	Range of measurement data [mg/m^3]	Geometric mean [mg/m^3]	95th-percentile [mg/m^3]	Duration and frequency
Polymerisation	since 1994	427	0 – 22.9	0.2	2.6	-
	1997 –1999	20 (s)	0.007 – 4.5	0.1	2.9	-
	1994 – 1999	265 (p)	<0.006 – 11	<0.03	0.4	-
	1994 – 1999	38 (p)	<0.004 – 2.4	<0.03	2	-
			2.6			
	1995 – 2000	5		-	-	-
Polyvinyl alcohol production	-	2/year (p)	0.2 – 1.4		-	-

p: personal sampling; s: stationary sampling

For the occupational risk assessment of vinyl acetate the MOS approach (outlined in the TGD) has been applied.

For the assessment of inhalation exposure during the production and polymerisation in areas with high levels of protection belonging to the chemical industry an 8-h time weighted average concentration of 3.0mg/m3 (95th percentile, round off) is taken to represent the worst case situation.

For dermal exposure for unprotected workers, the EASE model yields an exposure level of 42-420 mg/person/day. For actual dermal exposure levels, factors such as the substance being manufactured and processed in a closed system and the use of PPE during exposure relevant activities are taken into account, as these measures are generally adopted in the large-scale chemistry industry. A protection efficiency of 90% is taken as a default value leading to an exposure level of 42mg/person/day. Also, taking into account the short retention time of vinyl acetate on the skin, it has been determined that an exposure level of 0-42 mg/person/day should be taken to represent a reasonable worst case scenario.

1.2.2. Consumer exposure

Not applicable.

1.2.3. Indirect exposure of humans via the environment

The EU RAR (2008) has determined the indirect exposure to humans via the environment according to Appendix VII of chapter 2 of the TGD.

As a worst case scenario, the maximum intake due to exposure in the vicinity of a vinyl acetate production facility has been calculated. This is compared to an average intake due to exposure via the regional background concentration.

The resulting total daily dose is: DOSEtot_local = 36 µg / kg bw d

DOSEtot_regional = 2.47 E-03 µg / kg bw d

The calculated total doses comprise the following routes:

Route	**Percent of total dose**	
	local	**regional**
Drinking water	1.26	9.64
Fish	2.97E-05	0.44
Stem	0.36	0.33
Root	0.24	0.37
Meat	2.04E-04	2.41E-04
Milk	3.80E-03	4.50E-03
Air	98.13	89.22

The main route of indirect exposure is the intake via inhalation of air.

1.2.4. Environmental exposure

VAM releases from processing sites to the aquatic and atmospheric compartments (EU RAR, 2008)

Site	Processing (to/a)	Release to surface water (kg/year)	Comment	Direct release to atmosphere (kg/year)
1	30,000	15.8	calculated from d.l. of 50 µg/l, specific STP flow	430
2	10,000	1550	d.l. of 10 µg/l specific STP flow	2,630
3a 3b 3c 3d	6,000 10,000 8,000 4,000	100.2	calculated from given effluent concentrations and STP default values	28,000
5	90,000	331	see production site 4	367
6	25,000	22.2	calculated from annual emission to waste water and STP flow	11,100
7*	10,810	3	generic calculation	7,110
8	15,000	230	annual emission to waste water, specific STP flow	31,000
9	9,100	5	annual emission to waste water, specific STP flow	14,000
10	15,000	1.13	annual emission to waste water, specific STP flow	14,000
11	110,000	84.5	d.l. of 5.7 µg/l and STP flow	-
12	36,000	720	no specific data on emissions but on STP flow	7,500
13	280	-	no water involved in process	280
14	2,000	40	generic calculation	2,000
15	2,200	6.5	annual emission to waste water, specific STP flow	1,500
16	400	-	no water involved in process	70
17	300	6	generic calculation	400

Site	Processing (to/a)	Release to surface water (kg/year)	Comment	Direct release to atmosphere (kg/year)
18	14,000	10	annual emission to waste water, specific STP flow	8,000
19	25,000	249	annual emission to surface water, specific STP flow	7,000
20**	0	0.1	annual emission to waste water	1
23	17,000	-	pers. commun.	< 1.05E-05
24	5,000	225	calculated from d.l. of 5 mg/l and effluent flow from process	24,925
25	6,910	138	generic calculation	3,500
28	8,000	160	generic calculation	8,000
29	6,500	7.5	annual emission to waste water, specific STP flow	200
30	8,400	1.39	calculated from given effluent concentrations and STP flow	-
Σ	**474,900 t/year**	**3,906 kg/year**		**172,013 kg/year**

* Aggregated data. The processing volume of 10,810 t/a in the table refers to the two companies still conducting polymerisation of VAM themselves. The total volume of ready to use VAM polymers in manufacture, however, added up to 43,524 tonnes in 2000. The magnitude of emissions in relation to total VAM use was specified as a maximum of 30 kg/year to waste water treatment plants, and a maximum of 7,110 kg/year to air by industry association CEPE.

** confirmation of conducting merely compounding operations, i.e. handling of the (co)polymers

Emissions of VAM to surface water from processing (polymerisation) sites of VAM in the European Union (fibre industry and paint/coating industry excluded) (EU RAR, 2008)

Site	Processing (t/year)	Release to STP (kg/year)	Release to surface water (kg/year)	$Clocal_{water}$ (µg/l)	Specific information provided/used
1	30,000	1.58E+02	1.58E+01	4.81E-04	calculated from d.l. of 50 µg/l, STP effluent flow, dilution
2	10,000	1.55E+04	1.55E+03	6.60E-02	calculated from d.l. of 10 µg/l, STP effluent flow, dilution
3a	6,000	6.00E+02	6.00E+01	1.00E+01	calculated from d.l. of 100 µg/l

Site	Processing (t/year)	Release to STP (kg/year)	Release to surface water (kg/year)	$Clocal_{water}$ (µg/l)	Specific information provided/used
3b	10,000	3.00E+02	3.00E+01	5.00E-01	effluent concentr. of 50 µg/l
3c	8,000	2.40E+01	2.40E+00	4.00E-01	effluent concentr. of 4 µg/l
3d	4,000	7.80E+01	7.8E+00	1.3E+00	effluent concentr. of 13 µg/l
5	90,000	3.31E+03	3.31E+02	2.09E-01	calculated from d.l. of 50 µg/l, STP effluent flow, dilution
6	25,000	2.22E+02	2.22E+01	3.04E+00	calculated from emission to waste water, dilution
7*	10,810	3.00E+01	3.00E+00	5.00E-01	generic calculation
8	15,000	2.30E+03	2.30E+02	2.30E+00	emission to waste water, STP effluent flow
9	9,100	5.00E+01	5.00E+00	9.81E-01	emission to waste water, STP effluent flow
10	15,000	1.13E+01	1.13E+00	3.10E-03	emission to waste water, STP effluent flow
11	110,000	8.45E+02	8.45E+01	1.32E-01	calculated from d.l. of 5.7 µg/l, STP effluent flow, dilution
12	36,000	7.20E+03	7.20E+02	2.10E+00	STP effluent flow, dilution
13	280	0.00E+00	0.00E+00	0.00E+00	no water used in process
14	2,000	4.00E+02	4.00E+01	7.69E+00	generic calculation
15	2,200	6.50E+01	6.50E+00	1.03E+01	emission to waste water, STP effluent flow, dilution
16	400	0.00E+00	0.00E+00	0.00E+00	no water used in process
17	300	6.00E+01	6.00E+00	1.16E-05	generic calculation
18	14,000	1.00E+02	1.00E+01	1.27E+00	emission to waste water, STP effluent flow
19	25,000	2.49E+03	2.49E+02	9.61E+00	emission to surface water, STP effluent flow
20**	0	1.00E+00	1.00E-01	1.91E-07	emission to waste water
23	17,000	0.00E+00	0.00E+00	0.00E+00	no emission to waste water
24	5,000	2.25E+03	2.25E+02	7.23E-03	calculated from d.l. of 5 mg/l, STP effluent flow, dilution
25	6,910	1.38E+03	1.38E+02	2.67E-05	generic calculation
28	8,000	1.60E+03	1.60E+02	1.33E+00	generic calculation
29	6,500	7.50E+01	7.50E+00	2.03E-03	emission to waste water, STP effluent flow, dilution

Site	Processing (t/year)	Release to STP (kg/year)	Release to surface water (kg/year)	$Clocal_{water}$ (µg/l)	Specific information provided/used
30	8,400	1.39E+01	1.39E+00	1.78E-03	emission to waste water, STP effluent flow, dilution
Σ	**474,900 t/year**	**39,063 kg/year**	**3906 kg/year**		

* The processing volume of 10,810 t/a in the table refers to the two companies still conducting polymerisation of VAM themselves. The total volume of ready to use VAM polymers in manufacture, however, added up to 43,524 tonnes in 2000. The magnitude of emissions in relation to total VAM use was specified as a maximum of 30 kg/year to waste water treatment plants by industry association CEPE.

** confirmation of conducting merely compounding operations. i.e. handling of the (co)polymers

Local concentrations of VAM in soil and in soil porewater (EU RAR, 2008)

	DEP_{total_ann} ($mg/m^2 \cdot d^1$)	C_{sludge} (mg/kg)	Clocal for					
			soil (mg/kg)	agric. soil (mg/kg)	grass-land (mg/kg)	soil porewater (mg/l)	agric. soil porewater (mg/l)	grassland porewater (mg/l)
16	1.40E-01	No STP	5.16E-03	5.16E-03	6.03E-03	9.42E-03	9.42E-03	1.10E-02
3a	8.09E-03	0.56	6.13E-04	3.56E-04	3.63E-04	1.12E-03	6.50E-04	6.62E-04
3b	1.30E-02	0.28	6.36E-04	5.08E-04	5.67E-04	1.17E-03	9.27E-04	1.04E-03
3c	1.10E-02	0.02	4.18E-04	4.08E-04	4.74E-04	7.63E-04	7.44E-04	8.66E-04
3d	5.35E-03	0.07	2.40E-04	2.05E-04	2.34E-04	4.38E-04	3.77E-04	4.28E-04
6	1.20E-02	1.71	1.40E-03	6.17E-04	5.58E-04	2.55E-03	1.13E-03	1.02E-03

The following table summarizes the estimated emissions of vinyl acetate from different point and diffuse sources to water and atmosphere (EU RAR, 2008):

	Emission to waste water (tonnes/year)	Direct release to air (tonnes/year)
Release from point-sources: verified processing sites	40.31	174.52
Release from point-sources: non-verified (based on 325,000 t/a)	65.00	325.00
Diffuse release from end-use products containing VAM	300.00	2,700.00
Total	405.31	3,199.52
Regional (10 %)	40.53	319.95
Continental (90 %)	364.78	2,879.56

1.3. RISK CHARACTERISATION

1.3.1 Human Health Exposure

Estimated workplace exposures are not expected to exceed DNELs when the identified risk management measures are adopted. All RCRs are <1.

The adopted STEL is 10 ppm (35.2 mg/m3) and 8 hrTWA, 5 ppm (17.6 mg/m3), as concluded by SCOEL in October 2005 (SCOEL/SUM/122) and published in the above directive.

1.3.2 Environmental Exposure

RCRs generated from the generic worst case scenario developed in the EU RAR (2008) , not supported by measured environmental data

RCRs generated from the generic worst case scenario developed in the EU RAR (2008) , not supported by measured environmental data

STP	Water	Soil	Seawater
0.054	0.51	4.0	0.31

The RCR soil when refined with measured data from processing sites using STP is < 1.

RCRs generated from available data (only maximum RCR reported)

Environmental exposure	PEC	PNEC	RCR
In STP	100 µg/l	6000 µg/l	0.017
In local freshwater	10.3 µg/l	16 µg/l	0.64
In local soil	1.4E-03 mg/kg	0.0035 mg/kg	0.4
In local marine water (mg/l)	5.01E-01 µg/l	1.6 µg/l	0.31

The RCR soil when refined with measured data from processing sites using no STP is > 1 (worst case RCR = 1.47)

The 14th Risk Reduction Strategy Meeting of the Member States for the Implementation of Council Regulation (EEC) 793/93 on the Evaluation and Control of Risks of Existing Substances endorsed the following recommendations on the environment:

"The conclusion of the assessment of the risks to the terrestrial ecosystem is that there is a need for specific measures to limit the risks. The conclusion is reached because of: concerns for the local terrestrial compartment as a consequence of exposure arising from vinyl acetate processing, more specifically for processing ((co)polymerisation) sites exceeding 20,000 t/a. Sites already applying advanced techniques would not require further consideration of risk reduction measures.

For processing sites with a processing capacity > 20,000 t/a it is recommended:

- that competent authorities in the Member States concerned should lay down, in the permits issued under Directive 2008/1/EC[2], conditions, emission limit values or equivalent parameters or technical measures regarding vinyl acetate, in order for the installations concerned to operate according to the best available techniques (BAT) taking into account the technical characteristic of the installations concerned, their geographical location and the local environmental conditions.
- that Member States should carefully monitor the implementation of BAT regarding vinyl acetate and report any important developments to the Commission in the framework of the exchange of information on BAT.
- to facilitate permitting and monitoring under Directive 2008/1/EC vinyl acetate should be included in the ongoing work to develop guidance on best available techniques (BAT).
- that local emissions to the environment should, where necessary, be controlled by national rules to ensure that no risk for the environment is expected."

1.3.2.1 Indirect human exposures

Based on the identified uses for vinyl acetate, general population DNELs need not be generated and therefore the risk characterization ratios cannot be calculated for indirect human exposures.