

NV Chemicals Super Concentrated Dishwashing Liquid 20+

N.V. Chemicals (Aust) P/L

Chemwatch: 24-9193
Version No: 4.1.1.1
Material Safety Data Sheet according to NOHSC and ADG requirements

Chemwatch Hazard Alert Code: 3

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

Product Identifier

Product name	NV Chemicals Super Concentrated Dishwashing Liquid 20+
Synonyms	Not Available
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Multi-purpose detergent. Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

Registered company name	N.V. Chemicals (Aust) P/L
Address	24 Lisa Place Coolaroo VIC 3048 Australia
Telephone	+61 3 9351 1100
Fax	+61 3 9351 1077
Website	http://www.nvchemicals.com.au/
Email	info@nvchemicals.com.au

Emergency telephone number

Association / Organisation	N.V.Chemicals(Aust) P/L
Emergency telephone numbers	0411 387 097
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

ChemWatch Hazard Ratings

	Min	Max	
Flammability	1	1	
Toxicity	1	1	
Body Contact	3	3	
Reactivity	1	1	
Chronic	0	0	

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

Poisons Schedule	Not Applicable
Risk Phrases [1]	R41 Risk of serious damage to eyes.
	R52 Harmful to aquatic organisms.
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI



Relevant risk statements are found in section 2

Indication(s) of danger	Xi
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Safety advice

S02	Keep out of reach of children.
S23	Do not breathe gas/fumes/vapour/spray.
S26	In case of contact with eyes, rinse with plenty of water and contact Doctor or Poisons Information Centre.

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S35	This material and its container must be disposed of in a safe way.
S39	Wear eye/face protection.
S40	To clean the floor and all objects contaminated by this material, use water.
S46	If swallowed, seek medical advice immediately and show this container or label.
S56	Dispose of this material and its container at hazardous or special waste collection point.
S64	If swallowed, rinse mouth with water (only if the person is conscious).

Other hazards

Skin contact and/or ingestion may produce health damage*.

Cumulative effects may result following exposure*.

May produce skin discomfort*.

Possible respiratory and skin sensitizer*.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
61790-63-4	<10	<u>coconut oil diethanolamide</u>
27176-87-0	<10	<u>dodecylbenzenesulfonic acid</u>
9004-82-4	<10	<u>sodium lauryl ether sulfate</u>
Not Available	<1	dye, fragrance (green)
7732-18-5	balance	<u>water</u>

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire.
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	<ul style="list-style-type: none"> ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. ▶ Mists containing combustible materials may be explosive. <p>carbon dioxide (CO₂) nitrogen oxides (NO_x) sulfur oxides (SO_x) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by all means available, spillage from entering drains or water courses. ▶ Consider evacuation (or protect in place). ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse / absorb vapour. ▶ Contain or absorb spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Limit all unnecessary personal contact. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	25-50L Drums.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents, bases and strong reducing agents. ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Continued...

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Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
dodecylbenzenesulfonic acid	Dodecylbenzene sulfonic acid; (Laurylbenzenesulfonic acid)	2 mg/m3	21 mg/m3	130 mg/m3

Ingredient	Original IDLH	Revised IDLH
coconut oil diethanolamide	Not Available	Not Available
dodecylbenzenesulfonic acid	Not Available	Not Available
sodium lauryl ether sulfate	Not Available	Not Available
water	Not Available	Not Available


Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
coconut oil diethanolamide	E	≤ 0.1 ppm
dodecylbenzenesulfonic acid	C	> 1 to ≤ 10 parts per million (ppm)
sodium lauryl ether sulfate	E	≤ 0.01 mg/m ³

Notes: Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Personal protection	
Eye and face protection	<p>No special equipment for minor exposure i.e. when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE: Wear general protective gloves, e.g. light weight rubber gloves.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
BUTYL	A
NEOPRENE	A
VITON	A
NATURAL RUBBER	C
PVA	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type ABK-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	ABK-AUS / Class1 P2	-
up to 50	1000	-	ABK-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	ABK-2 P2
up to 100	10000	-	ABK-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
 A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

▶ Cartridge respirators should never be used for emergency ingress or in areas of

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unknown vapour concentrations or oxygen content.

- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Thick light green liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	~1.07
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	7-7.5	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	2.3 @ 20 C	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>A 30% fatty acid amide (cocoamide DEA solution) was a moderate skin irritant in rabbits. Test sites were scored for irritation according to Draize, and the Primary Irritation Index (PII) was 3.1 (maximum irritation is indicated by the score of 8). In products intended for prolonged contact with the skin, the concentration of cocoamide DEA should not exceed 5%. Fatty acid diethanolamides (C8-C18) and monoethanolamides are classified by CESIO as irritating.</p>

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	Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.	
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation. Low concentrations (0.6%) of fatty acid amides such as cocoamide DEA are severely irritating to the eyes of rabbits. Eye contact with fatty acid diethanolamides (C8-18) and fatty acid monoethanolamides may lead to a risk of serious damage to eyes (CESIO)	
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.	
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	Not Available	Not Available
coconut oil diethanolamide	TOXICITY	IRRITATION
	Not Available	Not Available
dodecylbenzenesulfonic acid	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (rat) LD50: 500-2000 mg/kg ^[2]	Skin: adverse effect observed (corrosive) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
sodium lauryl ether sulfate	TOXICITY	IRRITATION
	Oral (rat) LD50: 1600 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 25 mg/24 hr moderate
		Skin: adverse effect observed (irritating) ^[1]
water	TOXICITY	IRRITATION
	Oral (rat) LD50: >90000 mg/kg ^[2]	Not Available
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	

COCONUT OIL DIETHANOLAMIDE	<p>For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented.</p> <p>The Fatty nitrogen-derived amides (FND amides) comprise four categories: Subcategory I: Substituted Amides Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components) Subcategory III: Imidazole Derivatives Subcategory IV: FND Amphoteric</p> <p>Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies.</p> <p>Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II.</p> <p>Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories.</p> <p>Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories.</p> <p>Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.</p> <p>In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.</p> <p>Some typical applications of FND Amides are:</p>
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masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers.

The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health.

The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

for diethanolamine (DEA):

In animal studies, DEA has low acute toxicity via the oral and dermal routes with moderate skin irritation and severe eye irritation. In subchronic toxicity testing conducted via the oral route in rats and mice, the main effects observed were increased organ weights and histopathology of the kidney and/or liver, with the majority of other tissue effects noted only at relatively high dosages. In subchronic studies conducted via the dermal route, skin irritation was noted as well as systemic effects similar to those observed in the oral studies. DEA has not been shown to be mutagenic or carcinogenic in rats; however, there is evidence of its carcinogenicity in mice.

Subchronic toxicity: The subchronic toxicity of DEA has been studied in F344 rats and B6C3F1 mice by exposure through drinking water or dermal administration, in 2 week and 13 week studies.

Target organs for toxicity included blood, kidney, brain and spinal cord, seminiferous tubules and dermal application site in rats and liver, kidney, heart, salivary gland and dermal application site in mice. Effects on seminiferous tubules were accompanied by reductions in sperm count and reduced sperm motility. Hematological evaluations indicated normochromic, microcytic anemia in the dermal study in male rats (NOEL = 32 mg/kg) and females (LOEL = 32 mg/kg). Anemia was also observed in rats in the drinking water study with a LOEL of 14 mg/kg/d in females and a LOEL of 48 mg/kg/d in males for altered hematological parameters. These findings were similar to those observed in the 2 week studies, but the magnitude of the changes was greater in the 13 week studies. Hematological parameters were normal in controls. No associated histopathological changes were noted in femoral bone marrow. Haematological parameters were not evaluated in mice.

Developmental toxicity: In a developmental toxicity study conducted via the oral route, effects of concern were observed only in the presence of maternal toxicity. In a developmental toxicity study conducted via the dermal route using two species of mammals, developmental toxicity was observed only in one species and only at doses causing significant maternal toxicity. Metabolically, DEA is excreted largely unchanged in the urine.

Carcinogenicity: A two-year dermal cancer study bioassay results on DEA and three fatty acid condensates of DEA indicated that liver tumours occurred in male and female mice exposed to DEA and two of the condensates. In addition kidney tumours occurred in male mice exposed to DEA and one of the condensates. Compelling evidence suggested that the toxicity observed in mice and rats treated with the DEA condensates was associated with free DEA and not with other components of the condensates. A weight of evidence analysis of data relevant to the assessment of the liver and kidney tumours in mice resulted in the conclusion that these tumours are not relevant to humans under the expected conditions of exposure and that liver and kidney toxicity should be evaluated on a threshold basis. This conclusion is based on the following:

- DEA is not genotoxic
- tumour development occurred at doses also associated with chronic hyperplasia
- there was no dose-related increase in malignancy, multiplicity of tumours or decrease in latency period
- tumours occurred late in life
- tumour response was species-specific (only mice were affected, not rats)
- tumour response was sex-specific (only male mice were affected, not females)
- tumour development was site-specific, with only liver and kidney affected, both sites of DEA accumulation;
- there was no tumour response in skin, despite evidence of chronic dermal toxicity
- there is a plausible mechanism, supported by various data, to explain the renal toxicity of DEA
- data support threshold mechanisms of renal carcinogenesis for a number of non-genotoxic chemicals
- the exposure regime used in the mouse study (*i.e.*, lifetime continuous exposure to DEA in ethanol vehicle at doses causing chronic dermal toxicity) is not relevant to human exposure (exposure through cosmetic vehicles with daily removal, under non-irritating conditions).

In considering the aggregate data on a DEA basis from the four studies using DEA and related condensates, the NOEL for kidney toxicity was 19 mg/kg/d, which resulted from a dose of 100 mg/kg/d of cocamide DEA containing 19% free DEA.

Anaemia: Rats exposed to DEA condensates developed anaemia. This was considered to be of to be relevant for humans since anaemia in rodents and humans share common etiologies. The proposed mechanism by which DEA could cause anemia involves disruption of phospholipid metabolism leading to membrane perturbation and functional change to erythrocytes. Some doubt about the relevance of the findings arises because ethanol was used as the vehicle in the dermal studies, and ethanol is known to cause anaemia in rodents through a mechanism involving membrane disruption. The possibility of a synergistic or additive role for DEA and ethanol in combination cannot be ruled out.

In considering the aggregate data on a DEA basis from the four 13-week dermal studies using DEA and related condensates, the NOEL for microcytic anemia was 9.5 mg/kg/d, which resulted from a dose of 50 mg/kg/d of cocamide DEA containing 19% free DEA.

The NOELs for mice and rats derived in this hazard assessment were as follows:

Anaemia in rats: 9.5 mg/kg/d (based on microcytic anemia)

Organ toxicity in mice: 2.2 mg/kg/d (based on liver toxicity)

In extrapolating among species for the purposes of risk assessment, the prime consideration with respect to dermally applied DEA was differential dermal absorption. Evidence indicates that dermal penetration of

DEA is greatest in mice and lower in rats and humans. Interspecies extrapolation was accomplished in this assessment by converting applied doses to bioavailable doses (*i.e.*, internal doses) using dermal bioavailability determined in studies with rats and mice *in vivo*, so as to be able to compare these with internal doses expected to be experienced by humans through use of personal care products.

Based on measured bioavailability in mice and rats, the bioavailable NOELs corresponding to the foregoing were:

Anaemia in rats: 0.8 mg/kg/d (based on microcytic anemia)

Organ toxicity in mice: 0.55 mg/kg/d (based on liver toxicity)

Kidney toxicity: Effects on the kidney were observed in rats treated with DEA in drinking water or by dermal exposure after as little as 2 weeks of exposure. Effects included renal tubule hyperplasia, renal tubular epithelial necrosis, renal tubule mineralization and increased relative organ weight. Similar changes were observed after 13 weeks of exposure of rats to DEA in drinking water and by dermal administration. The NOEL in male rats was 250 mg/kg/d in the dermal study, while in female rats renal tubule mineralisation was observed at the lowest dose of 32 mg/kg/d. After 2 years of dermal exposure there were no histopathological changes in the kidneys of male rats given doses of up to 64 mg/kg/d. In females, there were no significant increases in the incidences of renal tubule epithelial necrosis, hyperplasia or mineralisation as was observed after 13 weeks of exposure, however, there was an increase in the severity and incidence of nephropathy. This was the result of a treatment-related exacerbation of a previously existing lesion, since the incidence in controls was 80%, increasing to 94-96% in treated groups. There was no significant increase in the incidence of kidney tumours in rats treated with DEA or any of the condensates in 2-year dermal studies.

Liver toxicity: Effects on liver, including increases in relative organ weight and histopathological changes were observed in male and female mice in the 2 week drinking water study with DEA. Increases in liver weight were observed in the two week dermal study, but were not associated with histopathological changes. After 13 weeks of exposure, relative liver weights were increased compared to controls in male and female rats, with no associated histopathology. There is some doubt about whether these changes in liver weights were of toxicological significance, since there was no associated histopathology, the dose-response was not consistent and there were no effects on liver in the 2 year study in rats.

In the study with coconut diethanolamide (CDEA) (100 and 200 mg/kg/d) in which 19% of the applied dose was DEA, there were no liver effects

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in rats after 13 weeks or 2 years of dermal exposure. No liver toxicity in rats was observed in the 2 year dermal studies of lauramide or oleamide DEA

Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure.

Fatty acid diethanolamides (C8-C18) are classified by Comité Européen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41

Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common.

Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978).

Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen. Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in *Salmonella typhimurium* strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of *Salmonella typhimurium* when tested with or without metabolic activation

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljøministeriet (Danish Environmental Protection Agency)

In a study of dermal application in mice, coconut oil diethanolamine condensate (coconut diethanolamide) increased the incidence of hepatocellular carcinoma and hepatocellular adenoma in males and females, and of hepatoblastoma in males. The incidence of renal tubule adenoma and carcinoma combined was also increased in males. In a study of dermal application in rats, no increase in tumour incidence was observed.

Tumours of the kidney and hepatoblastoma are rare spontaneous neoplasms in experimental animals.

The carcinogenic effects of the coconut oil diethanolamine condensate used in the cancer bioassay may be due to the levels of diethanolamine (18.2%) in the solutions tested.

Mechanistic data are very weak to evaluate the carcinogenic potential of coconut oil diethanolamine condensate per se

According to IARC:

Coconut oil diethanolamine condensate is possibly carcinogenic to humans (Group 2B)

for acid mists, aerosols, vapours

Data from assays for genotoxic activity *in vitro* suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events *in vivo* in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH *in vivo* differ from exposures *in vitro* in that, *in vivo*, only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than *in vitro*.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce respiratory tract irritation. Symptoms of pulmonary irritation may include coughing, wheezing, laryngitis, shortness of breath, headache, nausea, and a burning sensation.

Unlike most organs, the lung can respond to a chemical insult or a chemical agent, by first removing or neutralising the irritant and then repairing the damage (inflammation of the lungs may be a consequence).

The repair process (which initially developed to protect mammalian lungs from foreign matter and antigens) may, however, cause further damage to the lungs (fibrosis for example) when activated by hazardous chemicals. Often, this results in an impairment of gas exchange, the primary function of the lungs. Therefore prolonged exposure to respiratory irritants may cause sustained breathing difficulties.

Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC. Linear alkylbenzene sulfonic acids (LABS) are strong acids (pKa<2) are classified as corrosive (R34)

Branched materials exhibit comparable toxicity to linear species.

Acute toxicity: The available data indicate minimal to moderate toxicity, with LD50 values ranging from 500 to 2000 mg/kg body weight (bw). Acute inhalation data also indicate a lack of significant toxicity. Available dermal exposure data also shows a lack of significant toxicity.

LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin. The bulk is metabolised in the liver to sulfophenyl carboxylic acids. The metabolites are excreted primarily via the urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion.

No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice.

LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation.

Concentration of < 0.1% LAS produced mild to no irritation.

Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS.

Repeat dose toxicity: A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency.

Genotoxicity: The mutagenic potential of LAS was tested using *Salmonella typhimurium* strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity.

Reproductive toxicity: In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed.

DODECYLBENZENESULFONIC ACID

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Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljøministeriet (Danish Environmental Protection Agency)

For aromatic sulfonic acids

Aromatic sulfonic acids are very corrosive as was demonstrated in skin and eye irritation studies, in the acute oral studies, and in the single repeated dose oral study.

Health records from industrial manufacturing exposure, including manufacturing plant book of injuries and a physician report, show toluene-4-sulphonic acid (as handled in manufacturing plants; i.e., a 65% aqueous solution with < 5% free sulphuric acid) is an irritant to the eye and skin.

Sensitisation:

There is a single, key study for sensitization of the aromatic sulphonic acids. None of the tested animals showed positive responses in a, well documented, GLP guinea pig sensitization study with toluene-4-sulphonic acid (CAS No. 104-15-4). The test substance can be considered a non-sensitizer in guinea pigs as none of the test animals showed a positive response to combined intradermal and topical induction followed by topical challenge.

Repeat dose toxicity:

A GLP guideline study with p-toluenesulphonic acid (CAS No. 104-15-4) reported no adverse effects to male and female rats exposed orally for 28 days. The highest dose was 500 mg/kg bw/day (>490 mg/kg bw/day based on >98% active ingredient). Therefore the NOAEL was set at 500 mg/kg bw/day.

Toxicity to reproduction:

No fertility studies are reported for the aromatic sulphonic acids. There are however studies for the chemically related hydrotrope substances that looked at reproductive organs and development of offspring. Hydrotropes are the salt form of the sulphonic acids and therefore are used as read-across for this endpoint. The 90-day oral rat and oral mouse studies and the 2-year chronic dermal rat and mouse studies with the closely related compound sodium xylene sulfonate (CAS No. 1300-72-7) included examination of sex organs of both sexes. No treatment related effects on reproductive organs were reported at doses roughly equivalent to those in the developmental toxicity study. The NOAEL for both maternal and foetal toxicity was the highest dose tested - 3000 mg/kg bw /day which is equivalent to 936 mg active ingredient per kilogram body weight per day. The conclusion of the study was no indications of developmental toxicity including teratogenesis.

Genetic toxicity:

There is a fully documented, GLP Guideline (OECD 471) Ames Test and a fully documented, GLP Guideline (OECD 473) Chromosome Aberration Test for one of the aromatic sulphonic acids, p-toluenesulphonic acid (CAS No. 104-15-4). Both tests were conducted with and without metabolic activation. The Ames test exposed up to 5000 micrograms/plate and the chromosome aberration test exposed up to 1902 micrograms per liter of the test substance. These studies conclude the substance is neither mutagenic nor cytotoxic.

There is an additional, published report of an Ames Test for another of the aromatic sulphonic acids, benzenesulfonic acid (CAS No. 98-11-3). Exposures up to 10,000 micrograms/plate were done with and without metabolic activation. The conclusion is the same as for the p-toluenesulphonic acid; that is, not mutagenic and not cytotoxic.

There are no in vivo mutagenicity studies for the aromatic sulphonic acids, but there are two in vivo mouse micronucleus studies for the related hydrotropes – sodium cumene sulfonate (CAS 28348-53-0) and calcium xylene sulfonate (CAS 28088-63-3). Both are GLP-compliant Guideline mouse micronucleus studies with full documentation. Both studies conclude the test substances were not mutagenic in these assays.

Disulfonic acids have not been the subject of concern.

Carcinogenicity:

There are no carcinogenicity studies for the aromatic sulphonic acids Two hydrotrope studies involve 2-year rat and mouse dermal exposures conducted under GLP. Up to 240 mg (rats) and 727 mg (mice) sodium xylenesulfonate/kg body weight in 50% ethanol were dosed 5 days per week for 104 weeks. There were no treatment related incidences of mononuclear cell leukemia, neoplasms, or nonneoplastic lesions of the skin and other organs. The increased incidence of epidermal hyperplasia may have been related to exposure to the test substance. The NOAEL was reported as 240 mg/kg bw/day for rats and 727 mg/kg bw/day for mice.

Elimination:

The US EPA has evaluated the metabolism of analogs in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated."

ADI: 2.5 mg/kg/day NOEL: 250 mg/kg/day

* [CESIO]

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used

Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology <http://doi.org/10.5487/TR.2015.31.2.105>

Alkyl ether sulfates (alcohol or alkyl ethoxysulfates) (AES) (syn: AAASD ,alkyl alcohol alkoxy sulfate sulfates, SLES) are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R36 (Irritating to eyes). An exception has been made for AES (2-3E) in a concentration of 70-75% where R36 is substituted with R41 (Risk of serious damage to eyes).

AES are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC.

In assessing this family the Cosmetic Ingredient Review (CIR) Expert Panel recognized that most of the acute oral toxicity, dermal irritation and sensitization, subchronic and chronic oral toxicity, reproductive and developmental toxicity, carcinogenicity, and photosensitization studies have been conducted on ammonium laureth sulfate and sodium laureth sulfate. Sodium and ammonium laureth sulfate have not evoked adverse responses in any toxicological testing, including acute oral toxicity, sub-chronic and chronic oral toxicity, reproductive and develop-mental

SODIUM LAURYL ETHER SULFATE

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toxicity, carcinogenicity, and photosensitization studies. These data, however, are considered a sufficient basis for concluding that the other ingredients are safe in the practices of use and concentration described in the safety assessment because of the fundamental chemical similarities between them and because they all are chemically similar salts (salts are expected to be dissociated in any product formulation independent of whether the salt is sodium, ammonium, magnesium, or zinc) of sulfated ethoxylated alcohols, and they all function as surfactants in cosmetic formulations. Based on these considerations, safety test data on one ingredient may be extrapolated to all of them. The panel noted that sodium laureth sulfate and ammonium laureth sulfate can produce eye and/or skin irritation in experimental animals and in some human test subjects; irritation may occur in some users of cosmetic formulations containing these ingredients. The irritant effects, however, are similar to those produced by other detergents, and the severity of the irritation appears to increase directly with concentration.

Acute toxicity: AES are of low acute toxicity. Neat AES are irritant to skin and eyes. The irritation potential of AES containing solutions depends on concentration. Local dermal effects due to direct or indirect skin contact with AES containing solutions in hand-washed laundry or hand dishwashing are not of concern because AES is not a contact sensitizer and AES is not expected to be irritating to the skin at in-use concentrations. The available repeated dose toxicity data demonstrate the low toxicity of AES. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. The consumer aggregate exposure from direct and indirect skin contact as well as from the oral route via dishware residues results in an estimated total body burden of 29 µg/kg bw/day.

AES are easily absorbed in the intestine in rats and humans after oral administration. Radiolabelled C11 AE3S and C12 AE3S were extensively metabolized in rats and most of the ¹⁴C-activity was eliminated via the urine and expired air independently of the route of administration (oral, intraperitoneal or intravenous). The main urinary metabolite from C11 AE3S is propionic acid-3-(3EO)-sulfate. For C12 and C16 AE3S, the main metabolite is acetic acid-2-(3EO)-sulfate. The alkyl chain appears to be oxidised to CO₂ which is expired. The EO-chain seems to be resistant to metabolism.

AES are better tolerated on the skin than, e.g., alkyl sulfates and it is generally agreed that the irritancy of AES is lower than that of other anionic surfactants. Alkyl chain lengths of 12 carbon atoms are considered to be more irritating to the skin compared to other chain lengths. The skin irritating properties of AES normally decrease with increasing level of ethoxylation. Undiluted AES should in general be considered strongly irritating. Even at concentrations of 10% moderate to strong effects can be expected. However, only mild to slight irritation was observed when a non-specified AES was applied at 1% to the skin.

Subchronic toxicity: A 90-day subchronic feeding study in rats with 1% of AE3S or AE6S with alkyl chain lengths of C12-14 showed only an increased liver/body weight ratio. In a chronic oral study with a duration of 2 years, doses of C12-AE3S of 0.005 - 0.05% in the diet or drinking water had no effects on rats. The concentration of 0.5% sometimes resulted in increased kidney or liver weight.

Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2 (CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.

Reproductive and developmental toxicity: No evidence of reproductive and teratogenic effects was seen in a two-generation study in rats fed with a mixture (55:45) of AES and linear alkylbenzene sulfonates. Dietary levels of 0.1, 0.5, and 1% were administered to the rats either continuously or during the period of major organogenesis during six pregnancies. No changes in reproductive or embryogenic parameters were observed.

Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives.

Carcinogenicity: Chronic dietary studies conducted with rats showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).

NOTE: Some products containing AES/ SLES have been found to also contain traces (up to 279 ppm) of 1,4-dioxane; this is formed as a by-product during the ethoxylation step of its synthesis. The U.S. Food and Drug Administration recommends that these levels be monitored. The U.S. Environmental Protection Agency classifies 1,4-dioxane to be a probable human carcinogen (not observed in epidemiological studies of workers using the compound, but resulting in more cancer cases in controlled animal studies), and a known irritant with a no-observed-adverse-effects level of 400 milligrams per cubic meter at concentrations significantly higher than those found in commercial products. Under Proposition 65, 1,4-dioxane is classified in the U.S. state of California to cause cancer. The FDA encourages manufacturers to remove 1,4-dioxane, though it is not required by federal law.

Sensitising potential: Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture.

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

Toxicokinetics:

Following oral exposure, AES is readily absorbed in the gastrointestinal tract in human and rat and excreted principally via the urine or faeces depending on the length of the ethoxylate chain but independently of the route of administration. Once absorbed, AES is extensively metabolized by beta- or omega oxidation. The alkyl chain appears to be oxidized to CO₂ which is expired. The EO-chain seems to be resistant to metabolism. Regarding the different anions, it is expected that the salts will be converted to the acid form in the stomach. This means that for all types of parent chemical the same compound structure eventually enters the small intestine. Hence, the situation will be similar for compounds originating from different salts and therefore no differences in uptake are anticipated.

The length of the ethoxylate portion in an AES molecule seems to have an important impact on the biokinetics of AES in humans and in the rat. Alcohol ethoxysulfates with longer ethoxylate chains (>7-9 EO units) are excreted at a higher proportion in the faeces. This is however not of interest for the AES within this category as their ethoxylation grade is 1 to 2.5.

Dermal absorption

There are two reliable and relevant studies available assessing the dermal absorption rate of AES. The study with AES (C12 -14; 2 EO) Na (CAS 68891-38-3) was performed according to OECD guideline 428 with human skin of the abdomen region (3 donors, n=2). The test substance was applied at a concentration of 10% for 24 h.

The mean amount removed from the skin surface (skin wash) ranged from 87.16% to 94.56% of the dose applied. The amounts in the receptor could not be quantified, since it was below the analytical limit of quantification (LOQ). The mean recovery in the two first tape strips was 1.48% during all performed experiments. In the further 18 tape strips a mean recovery of 2.86% was documented. The recovery values for the cryocuts have accounted 0.56% in mean.

The mean absorbed dose, sum of the amounts found in the viable epidermis, dermis and receptor medium was 0.56%. The mean recovery values have varied from 90.90% to 100.21%, which complies with the acceptance criteria of 100 ± 15%.

There is also an in vivo study according to OECD guideline 427 for AES (C12 -14; 2 EO) Na (CAS 68891-38-3) available (Aulmann, 1996).

Wistar rats were exposed to 1% aqueous solutions of the test item for 15 min and 48 h under semi-occlusive conditions. The mean amount of AES (C12-14; 2 EO) Na (CAS 68891-38-3) removed from the skin surface after the 15 min exposure period (via washing) ranged from 92.8% to 97.2% of the dose and from 91.6% to 98.4% after 48 h when the skin was not washed until sacrifice. The amounts in faeces and skin could not always be quantified, since it was below the analytical limit of quantification (LOQ).

The mean absorbed dose, sum of the amounts found in urine, faeces and skin in the experiment with washing was about 0.1% and 0.9% without

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	<p>washing. The mean recovery values varied from 98.6% to 103%.</p> <p>Taking the results of both studies together the dermal absorption is very low. The in vitro study with human skin indicated the dermal absorption to be 0.56% within 24 h and the in vivo study indicated the dermal absorption to be 0.9% within 48 h. The mean recovery rates on the skin are greater than 87%. These data demonstrate that the test substance remains on the skin surface. Thus, the value of 0.9% dermal absorption is taken for the dermal absorption.</p> <p>References: Danish EPA - Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products (2001). Environmental Project No. 615, pp. 24-28 HERA (2003). Human & Environmental Risk Assessment on ingredients of European household cleaning products Alcohol Ethoxysulphates, Human Health Risk Assessment Draft, 2003. http://www.heraproject.com. Final Report of the Amended Safety Assessment of Sodium Laureth Sulfate and Related Salts of Sulfated Ethoxylated Alcohols: (International Journal of Toxicology 29 (Supplement 3) 151S-161S: 2010 http://journals.sagepub.com/doi/pdf/10.1177/1091581810373151 The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p>
COCONUT OIL DIETHANOLAMIDE & DODECYLBENZENESULFONIC ACID	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p>
SODIUM LAURYL ETHER SULFATE & WATER	No significant acute toxicological data identified in literature search.

Acute Toxicity	✗	Carcinogenicity	✗
Skin Irritation/Corrosion	✗	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✗
Respiratory or Skin sensitisation	✗	STOT - Repeated Exposure	✗
Mutagenicity	✗	Aspiration Hazard	✗

Legend: ✗ – Data either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity

NV Chemicals Super Concentrated Dishwashing Liquid 20+	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
coconut oil diethanolamide	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
dodecylbenzenesulfonic acid	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96	Fish	1.67mg/L	2
	EC50	48	Crustacea	2.5mg/L	2
	EC50	96	Algae or other aquatic plants	5.549mg/L	3
sodium lauryl ether sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC	48	Fish	0.26mg/L	5
water	Endpoint	Test Duration (hr)	Species	Value	Source
	LC50	96	Fish	897.520mg/L	3
	EC50	96	Algae or other aquatic plants	8768.874mg/L	3

Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

DO NOT discharge into sewer or waterways.

Persistence and degradability

Continued...

NV Chemicals Super Concentrated Dishwashing Liquid 20+

Ingredient	Persistence: Water/Soil	Persistence: Air
dodecylbenzenesulfonic acid	HIGH	HIGH
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
dodecylbenzenesulfonic acid	LOW (BCF = 140)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
dodecylbenzenesulfonic acid	LOW (KOC = 16830)
water	LOW (KOC = 14.3)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▸ DO NOT allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

SECTION 15 Regulatory information

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

coconut oil diethanolamide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

dodecylbenzenesulfonic acid is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

sodium lauryl ether sulfate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

NV Chemicals Super Concentrated Dishwashing Liquid 20+

National Inventory Status

National Inventory	Status
Australia - AIIC	Yes
Australia - Non-Industrial Use	No (coconut oil diethanolamide; dodecylbenzenesulfonic acid; sodium lauryl ether sulfate; water)
Canada - DSL	Yes
Canada - NDSL	No (coconut oil diethanolamide; dodecylbenzenesulfonic acid; sodium lauryl ether sulfate; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (coconut oil diethanolamide)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (coconut oil diethanolamide; sodium lauryl ether sulfate)
Vietnam - NCI	Yes
Russia - ARIPS	No (coconut oil diethanolamide)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 Other information

Revision Date	04/08/2020
Initial Date	01/11/2009

SDS Version Summary

Version	Issue Date	Sections Updated
3.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
4.1.1.1	04/08/2020	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Chronic Health, Classification, Disposal, Engineering Control, Exposure Standard, Fire Fighter (fire/explosion hazard), Fire Fighter (fire incompatibility), Handling Procedure, Ingredients, Instability Condition, Personal Protection (other), Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container), Use

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average
 PC – STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit.
 IDLH: Immediately Dangerous to Life or Health Concentrations
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index

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