

Chip Quik Lead Free Solder Paste Series #146-6192, 146-6193, 146-6194, 146-6195, 146-6196, 146-6198

RS Components

Chemwatch Hazard Alert Code: 2

Issue Date: **20/08/2021** Print Date: **30/09/2022** L.GHS.AUS.EN.E

Chemwatch: **5287-30**Version No: **4.1**Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

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

Product Identifier		
Product name	Product name Chip Quik Lead Free Solder Paste Series #146-6192, 146-6193, 146-6194, 146-6195, 146-6196, 146-6198	
Chemical Name	Not Applicable	
Synonyms	Product Codes: 146-6192, 146-6193, 146-6194, 146-6195, 146-6196, 146-6198	
Chemical formula	Not Applicable	
Other means of identification	Not Available	

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

Relevant identified uses Soldering components for bonding semiconductor chips and packages to circuit boards. This product is for industrial use only. Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	RS Components	
Address	25 Pavesi Street Smithfield NSW 2164 Australia	
Telephone	+1 300 656 636	
Fax	+1 300 656 696	
Website	www.au.rs-online.com	
Email	Not Available	

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	+61 1800 951 288
Other emergency telephone numbers	+61 3 9573 3188

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

ChemWatch Hazard Ratings

	Min	Max	
Flammability	0		
Toxicity	2		0 = Minimum
Body Contact	2	- 1	1 = Low
Reactivity	1		2 = Moderate
Chronic	2		3 = High 4 = Extreme

Poisons Schedule	Not Applicable
Classification ^[1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 4, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

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Label elements

Hazard pictogram(s)





Signal word

Danger

Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P261	Avoid breathing mist/vapours/spray.	
P271	Use only outdoors or in a well-ventilated area.	
P280	Wear protective gloves, protective clothing, eye protection and face protection.	
P284	[In case of inadequate ventilation] wear respiratory protection.	
P264	Wash all exposed external body areas thoroughly after handling.	
P270	Do not eat, drink or smoke when using this product.	
P273	Avoid release to the environment.	
P272	Contaminated work clothing should not be allowed out of the workplace.	

Precautionary statement(s) Response

IF INHALED: Remove person to fresh air and keep comfortable for breathing.	
If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.	
IF ON SKIN: Wash with plenty of water and soap.	
IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.	
If skin irritation or rash occurs: Get medical advice/attention.	
If eye irritation persists: Get medical advice/attention.	
Take off contaminated clothing and wash it before reuse.	
IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.	
Rinse mouth.	

Precautionary statement(s) Storage

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P405	Store locked up.	
P403+P233	Store in a well-ventilated place. Keep container tightly closed.	

Precautionary statement(s) Disposal

P501 Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

mixtures		
CAS No	%[weight]	Name
7440-31-5	<96.5	<u>tin</u>
8050-09-7	<4.5	<u>rosin-colophony</u>
7440-22-4	<3	silver
8000-41-7	<0.5	terpineol mixed isomers
7440-50-8	<0.5	copper
Not Available	<0.5	rheological modifier, proprietary
110-16-7	<0.4	maleic acid
Not Available	<0.4	surfactants, proprietary

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Leaend:

Eve Contact

1. Classified by Chemwatch: 2. Classification drawn from HCIS: 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI: 4. Classification drawn from C&I · * FLLIOFI Vs available

SECTION 4 First aid measures

Description of first aid measures

If this product comes in contact with the eyes:

- Wash out immediately with fresh running water.
- Figure 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.
 - Seek medical attention without delay; if pain persists or recurs seek medical attention.
- Removal of contact lenses after an eve injury should only be undertaken by skilled personnel.
- Particulate bodies from welding spatter may be removed carefully.
- **DO NOT** attempt to remove particles attached to or embedded in eve.
- Lay victim down, on stretcher if available and pad BOTH eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.
- Seek urgent medical assistance, or transport to hospital.

If skin contact occurs:

- 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.

For thermal burns:

- Decontaminate area around burn.
- Consider the use of cold packs and topical antibiotics.

For first-degree burns (affecting top layer of skin)

- ▶ Hold burned skin under cool (not cold) running water or immerse in cool water until pain subsides.
- ▶ Use compresses if running water is not available.
- Cover with sterile non-adhesive bandage or clean cloth.
- Do NOT apply butter or ointments; this may cause infection.
- ▶ Give over-the counter pain relievers if pain increases or swelling, redness, fever occur.

For second-degree burns (affecting top two layers of skin)

- Cool the burn by immerse in cold running water for 10-15 minutes.
- Use compresses if running water is not available. Do NOT apply ice as this may lower body temperature and cause further damage.
- Do NOT break blisters or apply butter or ointments; this may cause infection.
- Protect burn by cover loosely with sterile, nonstick bandage and secure in place with gauze or tape.

To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):

- Lay the person flat.
- ► Elevate feet about 12 inches.
- ▶ Elevate burn area above heart level, if possible.
- Cover the person with coat or blanket.
- Seek medical assistance.

For third-degree burns

Seek immediate medical or emergency assistance.

In the mean time:

- Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound.
- Separate burned toes and fingers with dry, sterile dressings.
- ▶ Do not soak burn in water or apply ointments or butter; this may cause infection.
- ► To prevent shock see above.
- For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway.
- Have a person with a facial burn sit up.
- ▶ Check pulse and breathing to monitor for shock until emergency help arrives.

Inhalation

Skin Contact

- If fumes or combustion products are inhaled remove from contaminated area.
- Lay patient down. Keep warm and rested
- Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.
- Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.
- ▶ Transport to hospital, or doctor, without delay

► IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.

- For advice, contact a Poisons Information Centre or a doctor.
- Urgent hospital treatment is likely to be needed.
- In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition.
- If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist.
- If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS.

Ingestion

Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:

INDUCE vomiting with fingers down the back of the throat, ONLY IF CONSCIOUS. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce "metal fume fever" in workers from an acute or long term exposure.

- Onset occurs in 4-6 hours generally on the evening following exposure. Tolerance develops in workers but may be lost over the weekend. (Monday Morning Fever)
- Pulmonary function tests may indicate reduced lung volumes, small airway obstruction and decreased carbon monoxide diffusing capacity but these abnormalities resolve after

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several months.

- Although mildly elevated urinary levels of heavy metal may occur they do not correlate with clinical effects.
- The general approach to treatment is recognition of the disease, supportive care and prevention of exposure.
- Seriously symptomatic patients should receive chest x-rays, have arterial blood gases determined and be observed for the development of tracheobronchitis and pulmonary
 edema

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- ► Foam.
- ► Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide
- Water spray or fog Large fires only.

Do not use a water jet to fight fire.

Special hazards arising from the substrate or mixture

Fire Incompatibility

- Reacts with acids producing flammable / explosive hydrogen (H2) gas
- Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters	
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water courses. Use water delivered as a fine spray to control fire and cool adjacent 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. Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	 Clean up all spills immediately. Avoid contact with skin and eyes. Wear impervious gloves and safety goggles. Trowel up/scrape up. Place spilled material in clean, dry, sealed container. Flush spill area with water.
Major Spills	 Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Neutralise/decontaminate residue (see Section 13 for specific agent). 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

bevelop work practices and procedures that prevent particulate from conting in contact with worker skin, thair, or personal clothing.
If work practices and/or procedures are ineffective in controlling airborne exposure or visual particulate from deposition on skin, hair, or
clothing, provide appropriate cleaning/washing facilities.

Safe handling

Procedures should be written that clearly communicate the facility's requirements for protective clothing and personal hygiene. These clothing and personal hygiene requirements help keep particulate from being spread to non-production areas or from being taken home by

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the worker.

- Never use compressed air to clean work clothing or other surfaces.
- Fabrication processes may leave a residue of particulate on the surface of parts, products or equipment that could result in employee exposure during subsequent material handling activities.
- As necessary, clean loose particulate from parts between processing steps. As a standard hygiene practice, wash hands before eating or smoking.
- To prevent exposure, remove surface scale or oxidation formed on cast or heat treated products in an adequately ventilated process prior to working the surface.
- Exposure to elements found in the metal, its alloys or recycled materials, may result as a result of inhalation, ingestion, and skin contact, when melting, casting, dross handling, pickling, chemical cleaning, heat treating, abrasive cutting, welding, grinding, sanding, polishing, milling, crushing, or otherwise heating or abrading the surface of this material in a manner which generates particulates.
- Exposure may also occur during repair or maintenance activities on contaminated equipment such as: furnace rebuilding, maintenance or repair of air cleaning equipment, structural renovation, welding, etc.
- Particulate depositing on hands, gloves, and clothing, can be transferred to the breathing zone and inhaled during normal hand to face motions such as rubbing of the nose or eyes, sneezing, coughing, etc.

For molten metals:

- Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions.
- · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use.
- · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated
- Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. During melting operations, the following minimum guidelines should be observed:
- · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage.
- · Store materials in dry, heated areas with any cracks or cavities pointed downwards.
- Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours.

Store in the dark.

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

Other information

- CARE: Packing of high density product in light weight metal or plastic packages may result in container collapse with product release
- Heavy gauge metal packages / Heavy gauge metal drums
- Metal can or drum
 - Packaging as recommended by manufacturer.
 - Check all containers are clearly labelled and free from leaks.

Storage incompatibility

- Avoid reaction with oxidising agents
- Avoid strong acids, bases.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

-						
Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	tin	Tin, metal	2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silver	Silver, metal	0.1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
tin	6 mg/m3	67 mg/m3	400 mg/m3
rosin-colophony	72 mg/m3	790 mg/m3	1,500 mg/m3
silver	0.3 mg/m3	170 mg/m3	990 mg/m3
terpineol mixed isomers	59 mg/m3	650 mg/m3	1,000 mg/m3
copper	3 mg/m3	33 mg/m3	200 mg/m3
maleic acid	2.1 mg/m3	23 mg/m3	140 mg/m3

Ingredient	Original IDLH	Revised IDLH
tin	Not Available	Not Available
rosin-colophony	Not Available	Not Available
silver	10 mg/m3	Not Available
terpineol mixed isomers	Not Available	Not Available

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Ingredient	Original IDLH	Revised IDLH
copper	100 mg/m3	Not Available
maleic acid	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit	
rosin-colophony	Е	≤ 0.01 mg/m³	
terpineol mixed isomers	Е	≤ 0.1 ppm	
maleic acid	E	≤ 0.01 mg/m³	

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

Care: Atmospheres in bulk storages and even apparently empty tanks may be hazardous by oxygen depletion. Atmosphere must be checked before entry.

Requirements of State Authorities concerning conditions for tank entry must be met. Particularly with regard to training of crews for tank entry; work permits; sampling of atmosphere; provision of rescue harness and protective gear as needed

Metal dusts must be collected at the source of generation as they are potentially explosive.

- Avoid ignition sources.
- Good housekeeping practices must be maintained.
- Dust accumulation on the floor, ledges and beams can present a risk of ignition, flame propagation and secondary explosions.
- ▶ Do not use compressed air to remove settled materials from floors, beams or equipment
- Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation.
- Use non-sparking handling equipment, tools and natural bristle brushes. Cover and reseal partially empty containers. Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations.
- ▶ Do not allow chips, fines or dusts to contact water, particularly in enclosed areas.
- Metal spraying and blasting should, where possible, be conducted in separate rooms. This minimises the risk of supplying oxygen, in the form of metal oxides, to potentially reactive finely divided metals such as aluminium, zinc, magnesium or titanium.
- Work-shops designed for metal spraying should possess smooth walls and a minimum of obstructions, such as ledges, on which dust accumulation is possible.
- Wet scrubbers are preferable to dry dust collectors.
- Bag or filter-type collectors should be sited outside the workrooms and be fitted with explosion relief doors.
- Cyclones should be protected against entry of moisture as reactive metal dusts are capable of spontaneous combustion in humid or partially wetted states.
- Local exhaust systems must be designed to provide a minimum capture velocity at the fume source, away from the worker, of 0.5 metre/sec.
- Local ventilation and vacuum systems must be designed to handle explosive dusts. Dry vacuum and electrostatic precipitators must not be used, unless specifically approved for use with flammable/ explosive dusts.

Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
welding, brazing fumes (released at relatively low velocity into moderately still air)	0.5-1.0 m/s (100-200 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2.5 m/s (200-500 f/min.) for extraction of gases discharged 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection

Appropriate engineering

controls









- Safety glasses with side shields.
- Chemical goggles.
- 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

Eye and face protection

See Hand protection below

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Hands/feet protection

- ▶ Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.
- ▶ Protective gloves eg. Leather gloves or gloves with Leather facing

Body protection

See Other protection below

Other protection

- During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards.
 Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of ventilation, wet and vacuum cleaning methods, respiratory protection, decontamination, special protective clothing, and when necessary, restricted work zones.
- Protective over-garments or work clothing must be worn by persons who may become contaminated with particulate during activities such as machining, furnace rebuilding, air cleaning equipment filter changes, maintenance, furnace tending, etc. Contaminated work clothing and over-garments must be managed in a controlled manner to prevent secondary exposure to workers of third parties, to prevent the spread of particulate to other areas, and to prevent particulate from being taken home by workers.
- Personnel who handle and work with <u>molten metal</u> should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments).

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	СРІ
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
PVC	A

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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 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	Grey paste with no odour; does not mix with water.		
Physical state	Non Slump Paste Relative density (Water = 1) Not Available		
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	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 Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available

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Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (Not Available%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and 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

ormation on toxicological e	ffects
Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be harmful.
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. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individual 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. The material may accentuate any pre-existing dermatitis condition Particles and foreign bodies produced by high speed processes may be penetrate the skin. Even after the wound heals persons with retained foreign bodies may experiencing sharp pain with movement or pressure over the site. Discolouration or a visible mass under the epidermis may be obvious. Numbness or tingling ("pins and needles"), with decreased sensation, may be the result of a foreign body pressing against nerves. Persons with diabetes or a history of vascular problems have a higher potential for acquiring an infection Open cuts, abraded or irritated skin should not be exposed to this material

produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

Corneal abrasions caused by particles and foreign bodies usually cause pain, tearing, and a feeling that there is something in the eye. They may also cause redness (due to inflamed blood vessels on the surface of the eye) or, occasionally, swelling of the eye and eyelid. Vision may become blurred. Light may be a source of irritation or may cause the muscle that constricts the pupil to undergo a painful spasm

Injuries that penetrate the eye may cause similar symptoms. If a foreign object penetrates the inside of the eye, fluid may leak out.

Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial 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.

Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.

Chronic

Eve

Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.

Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance

Metallic dusts generated by the industrial process give rise to a number of potential health problems. The larger particles, above 5 micron, are nose and throat irritants. Smaller particles however, may cause lung deterioration, Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.

Metals are widely distributed in the environment and are not biodegradable. Biologically, many metals are essential to living systems and are involved in a variety of cellular, physiological, and structural functions. They often are cofactors of enzymes, and play a role in transcriptional control, muscle contraction, nerve transmission, blood clotting, and oxygen transport and delivery. Although all metals are potentially toxic at some level, some are highly toxic at relatively low levels. Moreover, in some cases the same metal can be essential at low levels and toxic at Chemwatch: **5287-30** Page **9** of **18** Issue Date: **20/08/2021**

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higher levels, or it may be toxic via one route of entry but not another. Toxic effects of some metals are associated with disruption of functions of essential metals. Metals may have a range of effects, including cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenicity, and genotoxicity. Biological half lives of metals vary greatly, from hours to years. Furthermore, the half life of a given metal varies in different tissues. Lead has a half life of 14 days in soft tissues and 20 years in bone.

In considering how to evaluate the toxicity of metals of potential concern, a number of aspects of metal toxicity should be kept in mind:

Different species vary in their responses to different metals; in some cases, humans are more sensitive than rodents. Thus, there is a need for broad-based testing of metals;

- ▶ The route of exposure may affect the dose and site where the metal concentrates, and thus the observed toxic effects;
- ▶ Metal-metal interactions can reduce or enhance toxicity; biotransformation can reduce or enhance toxicity;
- It is difficult to predict the toxicity of one metal based on the adverse effects of another; in trying to evaluate the toxicity of one particular metal compound, predictions based on similar compounds of the same metal may be valid.

Silver is one of the most physically and physiologically cumulative of the elements. Chronic exposure to silver salts may cause argyria, a permanent ashen-grey discolouration of the skin, conjunctiva and internal organs (due to the deposit of an insoluble albuminate of silver). The respiratory tract may also be a site of local argyria (following chronic inhalation exposures) with a mild chronic bronchitis being the only obvious symptom.

Sub-chronic exposure to a substance containing silver results in elevated alkaline phosphatase levels along with pigmentation of the tissues and organs. These effects are commonly observed in studies on silver.

Organ and tissue pigmentation appears to be an intrinsic property of silver ions, constituting an early marker of silver toxicity. This effect is therefore taken into consideration for the derivation of toxicicological reference values.

The lowest NOAELs for the medium- and long-term toxicity of silver ions were based respectively on the 90-day study of rats conducted with silver sodium hydrogen and zirconium phosphate and on the 105-week combined chronic study on rats conducted with silver-zinc zeolite. These NOAELs were recalculated to take account of the silver content of the substance tested and the rate of release of the silver ions.

In order to derive the toxicological reference values, an oral absorption of 5% and a safety factor of 100 (10 for intra-species variability and 10 for inter-species variability) were used.

In the absence of any observed acute toxicity effect, it is not possible to define a toxicity reference value for short-term exposure. The conservative approach set out in the European assessment is to use the medium-term acceptable exposure limit (AEL) as the short-term AEL. This value is based on the no observed effect level in rats exposed for 90 days.

- \cdot Short/medium-term AEL = 0.3 mg/kg bw/d x 5% / 100 = 0.15 μ g/kg bw/d (silver ion equivalent)
- \cdot Long-term AEL = 0.09 mg/kg bw/d x 5% / 100 = 0.045 μ g/kg bw/d (silver ion equivalent)

In a 2015 opinion on the classification of silver-zinc zeolite, the ECHA Committee for Risk Assessment (RAC) concluded that there was a potential embryotoxic effect in rats at doses where the dams were not severely affected by the treatment. This was manifested primarily by a decrease in the viability of the foetuses/pups, observed to varying degrees in developmental toxicity studies conducted with silver chloride (post-implantation losses, mortality of all offspring, increased incidence of hydronephrosis and cryptorchidism) and silver acetate (slight increase in the percentage of litters with late foetal death) and in a two-generation study with silver-zinc zeolite (lower number of births (F19), higher stillbirth rate, lower live birth rate, reduced pup weight, lower thymus weight, increased incidence of hydronephrosis.

A two-generation study of rats conducted with a different active substance containing silver also observed a lower number of births (F1), along with a smaller live litter size on day 1 (F210), and a lower thymus weight.

Rosin (colophany) has caused allergic contact dermatitis in solderers using resin flux-cored solders, can be a sensitiser for strings players, and has caused dermatitis after use in adhesive tapes [NIOSHTEC]. It is found in many products that commonly come in contact with the skin, including cosmetics, sunscreens, veterinary medications, adhesives, sealants, polishes, paints and oils. Industrial use of rosins (both natural and modified) is common and they are found in such products as printing inks, cutting fluids, corrosion inhibitors and surface coatings. High-quality gloss paper may also be coated with rosin or its derivatives.

The main component of rosin is abietic acid, which by itself is non-sensitising.

Several allergens have been isolated from rosin; these include 15-hydroperoxyabietic acid (15-HPA) and 15-hydroperoxydehydroabietic acid (15-HPDA), a peroxide of dehydroabietic acid. In animal allergic-challenge testing, these two substances are cross-reactive despite differences in molecular weight and unsaturation. Both substances react via a radical mechanism generating structurally similar molecules which give rise to antigens producing the allergic reaction.

Gafvert et al: Arch Dermatol Res 284; 1992; pp 409-413

For a better understanding of the mechanisms of contact allergic reactions, the patterns of cross-reactivity between different resin acid oxidation products were studied.

The 13,14(a)-epoxide and the 13,14(b)-epoxide of abietic acid and 15-HPDA are contact allergens in experimental studies. The b-epoxide of abietic acid has been detected in gum rosins.

Cross reactivity has been observed between the a - and b- epoxides and also between the epoxides and 15-HPA (and also between 15-HPDA and 15-HPA). This can be explained if 15-HPA forms an epoxide which then reacts with skin protein to generate the complete antigen. Cross-reactivity between the two hydroperoxides might be preceded by the formation of similar alkoxy radicals which further react with skin protein. Cross-reactivity patterns of resin oxidation products indicate that 15-HPA may react with skin proteins either as a radical or as an epoxide, thus generating different antigens.

Gafvert et al: Chemical Research in Toxicology; 1994; pp 260-266

Esterification of rosin, with polyalcohols for example, reduces allergenic activity although some individuals still are allergic to the polyester. Reduced or diminished reaction to glycerol- and pentaerythritol- esterified rosins, is probably due to the formation of larger molecules (with reduced bioavailability).

Methyl ester of rosins, however, have molecular weights of similar magnitude to the parent rosin and when both are tested in sensitised patients, there is little difference in reactivity.

Shao et al: Contact Dermatitis 28; 1993; pp 229-234

Patch tests conducted using methyl resinate produced a lower level of response than similar tests on the same resin allergic individuals, conducted with glycerol, pentaerythritol and propylene glycol esters of rosin. It was not possible to determine whether those individuals who were methyl resin positive were cross-sensitised or were reacting to a non-specific irritant effect

Private Communication

The main compound formed in glycerol-modified rosins is glyceryl triabletate; lesser amounts of the monoabletate and diabletate are also formed. Whilst the triabletate elicits no or low allergenic activity, the monoabletate has been identified as a contact allergen.

Some individuals react to glycerol-modified rosins: both unmodified abietic acid and the monoabietate have been identified in these modified rosins.

Gafvert et al. Contact Dermatitis; 31 1994; pp 11-17

Rosin modified with fumaric acid or maleic anhydride is often used in paper size. A major product of the paper size in the modification of the rosin is fumaropimaric acid (FPA) which is formed by Diels-Alder addition of fumaric acid to levopimaric acid (l-abletic anhydride), another of the major components of rosin. The allergenic activity of isomers of FPA, tested in guinea pigs is low but maybe present. After prolonged heating, however, FPA is converted to maleopimaric acid (MPA). MPA has been shown to be a potent allergen in previous studies. MPA also forms when abietic acid and fumaric acid are heated together at 220 deg. C and is present in commercially available fumaric acid.

modified rosins. Free abietic acid has also been detected in these modified rosins

Fumaric acid-modified rosins were shown to elicit positive test results in guinea pigs sensitised to MPA.

Gafvert et al: Nordic Pulp and Paper Research Journal 10: 1995; 139-144

Essential oils and isolates derived from the Pinacea family, including Pinus and Abies genera, should only be used when the level of peroxides is kept to the lowest practicable level, for instance by adding antioxidants at the time of production. Such products should have a peroxide value of less than 10 millimoles peroxide per liter. Based on the published literature mentioning sensitising properties when containing peroxides (Food and Chemical Toxicology 11,1053(1973); 16,843(1978); 16,853(1978).

Pine needles and their extracts may contain isocupressic acids. Isocupressic acids have been described as causing toxicity problems in beef cattle. It has been found that a substantial amount of isocupressic acid remains in the extracts

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It has surprisingly been found that isocupressic acids can be removed from pine needle extracts to form an extract which still exhibits therapeutic activity (such as the ability to lower blood pressure).

ip Quik Lead Free Solder Paste Series #146-6192,	TOXICITY	IRRITATION
146-6193, 146-6194, 146-6195, 146-6196, 146-6198	Not Available	Not Available
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
tin	Inhalation(Rat) LC50; >4.75 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >2000 mg/kg ^[1]	
	TOXICITY	IRRITATION
rosin-colophony	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >1000 mg/kg ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
. 1	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
silver	Inhalation(Rat) LC50; >5.16 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50; >2000 mg/kg ^[2]	
	TOXICITY	IRRITATION
terpineol mixed isomers	dermal (rat) LD50: >2000 mg/kg ^[1]	Not Available
	Oral (Mouse) LD50; 2830 mg/kg ^[1]	
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
copper	Inhalation(Rat) LC50; 0.733 mg/l4h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (Mouse) LD50; 0.7 mg/kg ^[2]	
	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 1560 mg/kg ^[2]	Eye (rabbit): 1% / 2m SEVERE
maleic acid	Inhalation(Rat) LC50; >0.18 mg/L4h ^[2]	Eye (rabbit): 100 mg - SEVERE
maiere acid	Oral (Rat) LD50; 708 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
		Skin (rabbit): 500 mg/24h-SEVERE

specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

ROSIN-COLOPHONY

No evidence of a sensitization response was observed in the Gum roins key study, a guideline Local Lymph Node Assay conducted in mice, or in ten supporting studies conducted in guinea pigs according to the GPMT or Buehler methods. Gum Rosin is not classified for dermal sensitization according to the UN Globally Harmonized System of Classification and Labelling of Chemicals (GHS). Gum Rosin is currently classified for Skin Sensitization according to Annex I to Directive 67/548/EEC as R43: May cause sensitization by skin contact. Gum Rosin is also classified according to EU Classification, Labelling and Packaging of Substances and Mixtures (CLP) Regulation (EC) No. 1272/2008. As part of the harmonized translation between Directive 67/548/EEC and EU CLP Regulation (EC) No. 1272/2008. Table 3.1 of EU CLP Regulation (EC) No. 1272/2008 classifies Gum Rosin as "Skin Sensitizer Category 1" and assigns the hazard statement H317: May cause an allergic skin reaction.

Table 3.2 of EU CLP Regulation (EC) No. 1272/2008 contains a list of harmonized classifications and labelling of hazardous substances from Annex I to Directive 67/548/EEC. Gum Rosin is assigned the risk phrase R43: May cause sensitization by skin contact in Table 3.2.

Subsequent evaluation determined that the single positive study for Gum Rosin was actually conducted with an oxidized form of the test material. Several esters of Rosin have been tested using similar protocols with similar results. When the Rosin esters were heated beyond the specified protocol, the oxidized material caused a positive sensitization response. When those same esters were retested using a different protocol which did not cause oxidation, all sensitization responses were negative. While the oxidized form of Gum Rosin should be considered a skin sensitizer, the recommendation is made to declassify non-oxidized Gum Rosin (CAS # 8050-09-7).

Different rosin types are used interchangeably and are often chemically modified. Colophony (rosin) is the nonvolatile fraction of the exudates from coniferous trees, and its main constituent is abietic acid. Abietic acid has been described as the allergenic constituent. Because it is not an electrophile, its sensitizing capacity was questioned when investigations regarding the allergenic properties of colophony started many years ago. It was found that highly purified abietic acid is nonallergenic but rapidly autooxidises forming a hydroperoxide which subsequently was identified as a major allergen of colophony. A variety of other oxidation products from abietic acid and dehydroabietic acid (the other major resin acid in colophony) were isolated and identified, some of which were shown to be sensitizers in guinea pig studies. Clinical investigations have shown that patch testing with the hydroperoxide detects about 50% of the patients with contact allergy to colophony. Abietic acid, a rosin acid, is converted into a highly reactive hydroperoxide by contact with air.

Unmodified colophony is a complex mixture of diterpenoid acids (i.e., resin acids, ca. 90%), diterpene alcohols, aldehydes, and hydrocarbons To cause sensitization, a chemical must bind to macromolecules (proteins) in the skin (producing so-called haptenation).

Hydroperoxy resin acids are dermal sensitizers, with haptenation thought to occur via radical mechanisms. Conjugation of L-lysine to the resin is predicted, with a Schiff base (or imine) linkage formed between the C-7 of the resin and the free amino group of lysine. Resin acids accumulate in the plasma membrane, a non-aqueous environment apparently conducive to conjugation of hydroperoxy resin acids with lysine side chains of membrane proteins, through covalent binding. Such binding might lead to interaction with immune cells having resin acid specificity. The haptenation mechanism may be involved in allergic contact dermatitis and occupational asthma observed from exposure to resin acid solids and aerosols.

For a better understanding of the mechanisms of contact allergic reactions, the patterns of cross-reactivity between different resin acid oxidation products were studied. The 13,14(alpha)-epoxide and the 13,14(beta)-epoxide of abietic acid and 15-hydroperoxydehydroabietic acid (15-HPDA)

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were shown in experimental sensitization studies to be contact allergens. Cross-reactivity was observed between the alpha- and beta-epoxides and also between the epoxides and the previously identified rosin allergen 15-hydroperoxyabietic acid (15-HPA). This indicates that 15-HPA may form an epoxide which then reacts with skin protein to generate the complete antigen. 15-HPA and 15-HPDA cross-reacted as well. This can be explained by the formation of similar alkoxy radicals from both hydroperoxides which further react with skin protein. Cross-reactivity patterns of the resin acid oxidation products indicate that 15-HPA may react with skin proteins either as a radical or as an epoxide, thus generating different antigens. The presence in rosin of the epoxides of abjetic acid was also studied. The beta-epoxide was detected in gum rosin. Moreover, the epoxides elicited reactions in rosin-allergic individuals. Thus, the 13,14(beta)-epoxide of abietic acid was identified as a new, important rosin allergen.

Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and connubial contact dermatitis occur.

Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.

Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.

Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.

Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.

Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a suffcient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.

Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation .Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.

Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.

Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of of being fragrance allergic.

Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this, Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported . The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.

Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified.. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.

Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plantderived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.

General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.

In the case of prehaptens, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxyl radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the

TERPINEOL MIXED ISOMERS

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hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.

It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clincal studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

Prohaptens

600 mg/kg/d

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens.

In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases; phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin . These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha, beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation. For terpenoid tertiary alcohols and their related esters:

Substances assigned to this category, as part of the HPV Challenge Program, possess close structural relationships, similar physicochemical properties and participate in the same pathways of metabolic detoxification and have similar toxicologic potential.

Acute Toxicity: Oral and dermal LD50 values for members of this chemical category indicate a low order of both oral and dermal toxicity. All rabbit dermal, and mouse and rat oral LD50 values exceed 2000 mg/kg with the majority of values greater than 5000 mg/kg Repeat dose toxicity: In a safety evaluation study, a 50/50 mixture of linalool and citronellol was fed to male and female rats (number and strain not specified) in the diet. The daily intake was calculated to be 50 mg/kg bw of each. Measurements of haematology, clinical chemistry, and urinalysis at weeks 6 and 12 showed no statistically significant differences between test and control groups. Histopathology revealed no dose-related lesions. A slight retardation of growth was observed in males only, but was concluded by the authors to be biologically insignificant Reproductive toxicity: Four groups of 10 virgin Crl CD rats were administered 0,250,500, or 1000 mg/kg bw of an essential oil (coriander oil) known to contain 73% linalool by mass. The test material was given by gavage once daily, 7 days prior to cohabitation, through cohabitation (maximum of 7 days), gestation, delivery, and a 4-day post-parturition period. The duration of the study was 39 days. Maternal effects reported included increased body weight and increased food consumption at 250 mg/kg/d, a non-statistically significant decrease in body weight and food consumption and decreased gestation index and decreased length of gestation at 500 mg/kg/d, and a statistically significant decrease in body weight and food consumption, statistically significant decrease in gestation index, length of gestation, and litter size at 1000 mg/kg/d. The only effect on pups was a decrease in viability of pups at the highest dose level. The authors concluded that there were no effects observed in the dams at the low dose of 250 mg/kg bw/d or in the offspring at the 250 and 500 mg/kg bw/d levels. The authors concluded that the maternal NOAEL was 250 mg/kg/d and the developmental NOAEL was 500 mg/kg/d.

Four groups of 10 virgin Crl CD rats were administered 0,375,750, or 1500 mg/kg bw of an essential oil (cardamom oil) known to contain greater than 65 % tertiary terpenoid alcohols with 5 1% alpha-terpineol acetate by mass. Maternal observations included a non-statistically significant decrease in body weight gain and food consumption at 375 mg/kg/d.

Mortality, clinical signs, a statistically significant decrease in body weight gain and food consumption, and gross lesions at necropsy were seen at 750 and 1500 mg/kg/d. The only effects on pups were a reduced body weight gain in pups at 750 and 1500 mg/kg/d and increased mortality at 1500 mg/kg/d. The authors concluded that there were no significant adverse effects in the dams or offspring at the 375 mg/kg/d dose. A maternal NOEL was reported to be less than 375 mg/kg/d based on reduced body weight gain and food consumption at 375 mg/kg/d and a developmental NOAEL was reported to be 375 mg/kg/d

Developmental toxicity: A range finding study and follow-up teratology study was performed with pine oil. Pregnant Crl:CD(SD) BR rats were given 0, 50, 100, 500,750,or 1000 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Laparotomies were performed, corpora lutea were counted, and the uterus of each rat was removed, weighed and then examined for number, placement and viability of implantations. Live foetuses were weighed, sexed and gross external alternations were identified. There were no deaths or abortions during the course of this study. Necropsy revealed no gross lesions. Maternal effects included local alopecia, decreased body weight gain and food consumption for the 3 highest dose levels. At 750 and 1000 mg/kg, average gravid uterine weight was reduced. In foetuses, decreased body weight was observed at dose levels of 100 mg/kg and above, and at dose levels of 500 and above there was a slight increase in average number of resorptions/litter. In the follow-up teratology study, pregnant Crl:CD(SD) BR rats were given 0, 50, 600, or 1200 mg/kg/d by gavage in corn oil on days 6 to 20 of gestation. Six of the 25 rats in 1200 mg/kg dose group died and necropsies revealed that adrenal weights were significantly increased in these rats. At 1200 mg/kg/d, foetuses exhibited increased incidences of delayed ossification, delayed brain development, decreased weights, increased embryo -foetal mortality, and sunken eye bulge with associated soft and hard tissue findings, a dose that also resulted in maternal death and a low incidence of embryo-foetal death (resorption). The maternal and developmental NOEL for pine oil was greater than 50 mg/kg/d but less than

Genotoxicity: Mutagenicity/genotoxicity testing has been performed on six members of this chemical category, including a complete battery of in vitro genotoxicity tests using linalool. In nineteen separate in vitro tests on the mutagenicity and genotoxicity of terpenoid tertiary alcohols and related esters, all but two were negative. One of the positive results for linalool was observed in a rec assay using differences in growth rates in two strains of Bacillus subtilis as a measure of DNA changes In contrast, no evidence of mutagenicity was observed in the same test at a higher concentrations nor was DNA damage observed in a rat hepatocyte UDS assay. The authors of the mouse lymphoma assay which gave a weak positive result for linalool, emphasized that positive results in this assay are commonly observed for polar substances in the absence of S-9 and may be associated with changes in physiologic culture conditions (pH and osmolality).

Based on a weight of evidence evaluation of the available in vitro and in vivo mutagenicity and genotoxicity assays on terpenoid tertiary alcohols and related esters, this group of flavouring substances would not be expected to exhibit a low genotoxic potential in vivo

Metabolic fate: Based on the results of hydrolysis, the reactivity of linalool in aqueous media, and data on metabolism it is concluded that

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members of this chemical category exhibit similar chemical and biochemical fate. The esters are readily hydrolyzed to the corresponding alcohols, linalool and alpha-terpineol. Linalool is then partial converted to alpha-terpineol mainly under acidic conditions. Alicyclic and aliphatic tertiary alcohols are efficiently detoxicated by two principal pathways: conjugation primarily with glucuronic acid and excretion primarily in urine, and omega-oxidation to eventually yield diacids and their reduced or hydrated analogs. These polar metabolities will be efficiently excreted primarily in the urine either unchanged or as the glucuronic acid conjugates. The physiochemical and toxicological properties of these substances are consistent with their known reactivity and common metabolic fate.

Esters belonging to this category can be hydrolysed to their corresponding terpenoid alcohol and organic acid. Hydrolysis can also be catalysed by a class of esters known as carboxylesterases or B-type esterases that predominated in hepatocytes.

Esters of tertiary terpenoid alcohols are readily hydrolyzed in animals, including fish. Once hydrolysed, the resulting alcohols undergo excretion unchanged or as the glucuronic acid conjugate. To a minor extent, CYP-450 mediated oxidation at the omega or omega-1 position yields polar oxidized metabolites capable of excretion primarily in the urine Terpenoid alcohols formed in the gastrointestinal tract are readily absorbed. During hydrolysis under acidic condition cyclisation may occur.

In humans and animals, terpenoid tertiary alcohols primarily conjugate with glucuronic acid and are excreted in the urine and feces. Terpenoid alcohols with unsaturation may also undergo allylic oxidation to form polar diol metabolites that may be excreted either free or conjugated. If the diol contains a primary alcohol function, it may undergo further oxidation to the corresponding carboxylic acid. In a minor pathway, the endocyclic alkene of alpha-terpineol is epoxidised and then hydrolyzed to yield a triol metabolite 1,2,8-trihydroxy--p-menthane which also has been reported in humans following inadvertent oral ingestion of a pine oil disinfectant containing alpha-terpineol.

Bicyclic tertiary alcohols are conjugated with glucuronic acid and excreted primarily in the urine. In rabbits the structurally related bicyclic tertiary alcohols thujyl alcohol (4-methyl-1-(I-methylethyl)bicyclo[3.1.0]-hexan-3-ol) and beta-santenol (2,3,7-

trimethylbicyclo[2.2.1]-heptan-2-ol) are conjugated with glucuronic acid. In a metabolism study using the terpenoid tertiary alcohol trans-sobrerol, in humans, dogs, and rats, ten metabolites were isolated in urine, eight of which were characterised in humans. Two principle modes of metabolism were observed, allylic oxidation of the ring positions and alkyl substituents, and conjugation of the tertiary alcohol fractions with glucuronic acid. These metabolic patterns are common modes of converting tertiary and secondary terpenoid alcohols to polar metabolites, which are easily excreted in the urine and faeces. Menthol forms similar conjugation products in rats

With few exceptions * (see below) there are no safety concerns regarding certain cyclic and non-cyclic terpene alcohols **, as fragrance ingredients, under the present declared levels of use and exposure for the following reasons

- The non-cyclic and cyclic terpene alcohols have a low order of acute toxicity
- No significant toxicity was observed in repeated dose toxicity tests; it is concluded that these materials have dermal and oral NOAELs of 50 mg/kg body weight/day or greater.
 - These materials were inactive in mutagenicity and genotoxicity tests.
- · Based on data on metabolism it is concluded that members of this category exhibit similar chemical and biochemical fate.
- Although there is some indication for the production of reactive metabolites by some materials, these metabolites appear to be efficiently detoxicated and not expected to result in overt toxicity. There is no indication for the production of persistent metabolites.
- The results from materials studied to date are indicative of the group and there are no grounds for environmental concern with respect to cyclic and non-cyclic terpene alcohol compounds as currently used in fragrance compounds.
- Human dermatological studies show that, at current use levels, these materials are practically non-irritating.
- The sensitization potential is generally low.
- The margin of safety is generally greater than 100 times the maximum daily exposure.

Sufficient data are available from farnesol, linalool, menthol and a-terpineol, i.e., compounds that contain all key structural elements and potential sites of metabolism of all other members in the group, to demonstrate that the non-cyclic and cyclic terpenes share common metabolic pathways. In most cases, metabolism yields innocuous metabolites. Some materials, however, may generate alpha, b-unsaturated compounds or be oxidized to hydroperoxides. Such compounds have the capacity to participate in a range of nucleophilic and electrophilic addition reactions with biological material.

- * Safety concerns exist for:the following substances for the following reasons.
 - 6,7-Dihydrogeraniol, hydroabietyl alcohol and 6-isopropyl-2-decahydro-naphthalenol are potent skin sensitizers. These materials are prohibited for use in fragrance materials by IFRA Standards.
 - Farnesol is a weak sensitizer. Its use in fragrance materials is therefore restricted by IFRA Standards.
 - Sclareol and linalool may contain impurities and/or oxidation products that are strong sensitizers. For use in fragrance materials, these compounds must comply with the purity criteria stated in their IFRA Standards.
 - No sensitization test results were available for 2(10)-pinen-3-ol, 2,6-dimethyloct-3,5-dien-2-ol, and 3,7-dimethyl-
 - 4,6-octadien-3-ol. These materials should be regarded as potential sensitizers until tested.
- ** The common characteristic structural element of acyclic -noncyclic- and cyclic terpene alcohols is the typically branched isoprene unit 2-methyl-1,3-butadiene

The Research Institute for Fragrance Materials (RIFM) Expert Panel

WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever. for copper and its compounds (typically copper chloride):

Acute toxicity: There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an excutation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs.

No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation.

Repeat dose toxicity: In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. Genotoxicity: An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen.

Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride.

Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).

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Tremor, convulsions, muscle weakness, ulceration with bleeding from the stomach recorded The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of MALEIC ACID 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. **TIN & TERPINEOL MIXED** No significant acute toxicological data identified in literature search. **ISOMERS** The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact **ROSIN-COLOPHONY &** eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria TERPINEOL MIXED ISOMERS involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the & COPPER & MALEIC ACID distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible TERPINEOL MIXED ISOMERS airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal & MALEIC ACID lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production. **Acute Toxicity** Carcinogenicity × Skin Irritation/Corrosion Reproductivity Serious Eye Damage/Irritation V STOT - Single Exposure

Legend:

STOT - Repeated Exposure

Aspiration Hazard

💢 – Data either not available or does not fill the criteria for classification

– Data available to make classification

×

SECTION 12 Ecological information

Respiratory or Skin

sensitisation Mutagenicity

Toxicity

Chip Quik Lead Free Solder Paste Series #146-6192, 146-6193, 146-6194, 146-6195, 146-6196, 146-6198	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
tin	Not Available	Not Available	Not Available	Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC0(ECx)	48h	Crustacea	2.15mg/l	1
	EC50	72h	Algae or other aquatic plants	>10<20mg/	2
rosin-colophony	EC50	48h	Crustacea	4.5mg/l	1
	LC50	96h	Fish	1.5mg/l	2
	EC50	96h	Algae or other aquatic plants	0.031mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	120h	Fish	<0.001mg/L	4
. 9	EC50	72h	Algae or other aquatic plants	11.89mg/l	2
silver	EC50	48h	Crustacea	0.001mg/l	2
	LC50	96h	Fish	0.006mg/l	2
	EC50	96h	Algae or other aquatic plants	0.002mg/L	4
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	41mg/l	2
terpineol mixed isomers	EC50	48h	Crustacea	83.3mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	<3.2mg/l	2
	LC50	96h	Fish	62.3mg/l	2
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
copper	EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
соррег	EC50	48h	Crustacea	<0.001mg/L	4
	LC50	96h	Fish	0.005-0.06mg/l	4

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	EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	17.17mg/l	2
maleic acid	EC50	48h	Crustacea	42.81mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	4.15mg/l	2
	LC50	96h	Fish	>300mg/l	1
Legend:	Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment. DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
rosin-colophony	HIGH	HIGH
terpineol mixed isomers	HIGH	HIGH
maleic acid	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
rosin-colophony	HIGH (LogKOW = 6.4607)
terpineol mixed isomers	LOW (LogKOW = 3.28)
maleic acid	LOW (BCF = 11)

Mobility in soil

Ingredient	Mobility
rosin-colophony	LOW (KOC = 21990)
terpineol mixed isomers	LOW (KOC = 57.85)
maleic acid	LOW (KOC = 6.314)

SECTION 13 Disposal considerations

Waste treatment methods

- Recycle wherever possible or consult manufacturer for recycling options.
- ▶ Consult State Land Waste Management Authority for disposal

Metal scrap recycling operations present a wide variety of hazards, including health hazards associated with chemical exposures and safety hazards associated with material processing operations and the equipment used in these tasks. Many of these metals do not pose any hazard to people who handle objects containing the metal in everyday use. In cases where employees could be exposed to multiple hazardous metals or other hazardous substances at the same time or during the same workday, employers must consider the combined effects of the exposure in determining safe exposure levels.

The recycling of scrap metals is associated with illness and injury The most common causes of illness were poisoning (e.g., lead or cadmium poisoning), disorders associated with repeated trauma, skin diseases or disorders, and respiratory conditions due to inhalation of, or other contact with, toxic agents.

The most common events or exposures leading to these cases were contact with an object or piece of equipment; overextension; and exposure to a harmful substance. The most common types of these injuries were sprains and strains; heat burns; and cuts, lacerations, and punctures. Any combustible material can burn rapidly when in a finely divided form. If such a dust is suspended in air in the right concentration, under certain conditions, it can become explosible. Even materials that do not burn in larger pieces (such as aluminum or iron), given the proper conditions, can be explosible in dust form. The force from such an explosion can cause employee deaths, injuries, and destruction of entire buildings. Breaking apart large metal pieces may involve the use of gas cutting torch. Classic cutting torches use gas, while other torches use plasma or powder, or even water. Thermal (gas) torches expose employees to sprays of sparks and metal dust particles, to high temperatures, to bright light that could damage eves (light both inside and outside of the visible spectrum), and to various gases.

Materials that require higher temperatures to cut, such as pig iron and heat-resistant alloyed scrap, or materials that conduct heat too well to be cut with thermal torches, such as copper and bronze, may be cut with non-thermal methods such as plasma torches or powder cutting torches. Plasma torches are often used for superconductors of heat or heat-resistant metals, such as alloy steels containing nickel and/or chromium. Plasma torches generate a large amount of smoke and noise, as well as ultraviolet (UV) and infrared(IR) light. Depending on the metal, this smoke could contain toxic fumes or dusts.

Other hazards common to cutting operations (as well as to welding and brazing) include burns, fires, explosions, electric shock, and heat stress. Even chemicals that are generally not flammable may burn readily when vapourised.

Larger scrap metal objects are often broken apart using stationary shears, such as alligator shears used to cut apart short steel for foundries or to cut nonferrous metals. These machines can send small pieces of metal flying.

Many scrap metal recycling operations heat scrap pieces to high temperatures to separate different metal components, increase the purity of scrap, bake out non-metal substances, burn off contaminants, remove insulation from wire, or otherwise process the metal scrap. This may be done using furnaces or ovens that use fuel or electrical heating sources. Furnaces generate smoke, dust, and metal furnes, depending on temperature and content. Combustion by-products may include sulfur and nitrogen oxides, and carbon monoxide and carbon dioxide. Organic compounds may be emitted as heating vapourises oil and grease on scraps. In addition, heating or burning of certain plastics (such as plastic-coated wiring) may release phosgene or other hazardous substances. Emissions from fluxing typically include chlorides and fluorides. The highest concentrations of 'fugitive emissions (i.e., gases and vapours that escape from equipment) occur when the lids and doors of a furnace are opened during charging, alloying, and other operations.

Chemical processes are also used in a wide range of metal scrap recycling industries as a means to separate scrap into its component metals, to clean scrap metal prior to using physical processes, to remove contaminants (such as paint) from scrap material, or to extract selected metals from a batch of scrap containing many metal types. Chemical processes may include high-temperature chlorination, electrorefining, plating,

Product / Packaging disposal

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leaching, chemical separation, dissolution, reduction, or galvanizing. The most probable emissions from these processes include metal fumes and vapours, organic vapours, and acid gases. Other potential hazards may include high amounts of heat, splashing of caustic or other-wise hazardous chemicals, or combustion hazards.

The recycling of scrap metals or metals found in e-waste (such as printed circuit boards) may present a significant environmental and human health risk. These may contain heavy metals such as cadmium, cobalt, chrome, copper, nickel, lead and zinc. Roads and premises of nearby public facilities such as a school-yard and outdoor food market have been shown to be adversely impacted by the uncontrolled recycling activity. Heavy metal concentrations, especially lead and copper, in workshop and road dusts were found to be severely enriched, posing potential health risks, especially to children.

- ▶ Recycle wherever possible or consult manufacturer for recycling options.
- Consult State Land Waste Authority for disposal
- Bury or incinerate residue at an approved site.
- · Recycle containers if possible, or dispose of in an authorised landfill.
- 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.

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

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
tin	Not Available
rosin-colophony	Not Available
silver	Not Available
terpineol mixed isomers	Not Available
copper	Not Available
maleic acid	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
tin	Not Available
rosin-colophony	Not Available
silver	Not Available
terpineol mixed isomers	Not Available
copper	Not Available
maleic acid	Not Available

SECTION 15 Regulatory information

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

tin is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

rosin-colophony is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

silver is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2

Australian Inventory of Industrial Chemicals (AIIC)

terpineol mixed isomers is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

copper is found on the following regulatory lists

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

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Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) -

Schedule 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) -

Schedule 5

maleic acid is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) -

Australian Inventory of Industrial Chemicals (AIIC)

International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (tin; rosin-colophony; silver; terpineol mixed isomers; copper; maleic acid)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (tin; rosin-colophony; silver; copper)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	20/08/2021
Initial Date	11/12/2017

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification
4.1	20/08/2021	Classification change due to full database hazard calculation/update.

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 Cancel

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

ES: Exposure Standard 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

AIIC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

IECSC: Inventory of Existing Chemical Substance in China

EINECS: European INventory of Existing Commercial chemical Substances

ELINCS: European List of Notified Chemical Substances

NLP: No-Longer Polymers

ENCS: Existing and New Chemical Substances Inventory

KECI: Korea Existing Chemicals Inventory

NZIoC: New Zealand Inventory of Chemicals

PICCS: Philippine Inventory of Chemicals and Chemical Substances

TSCA: Toxic Substances Control Act

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TCSI: Taiwan Chemical Substance Inventory

INSQ: Inventario Nacional de Sustancias Químicas

NCI: National Chemical Inventory

FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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