



Riva Applicator Pro
SDI (North America) Inc.

Version No: 2.1
Safety Data Sheet according to OSHA HazCom Standard (2024) requirements

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L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Riva Applicator Pro
Chemical Name	Stainless Steel
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	For the application of capsulated dental materials by dental professionals. Note: Hazard statements relates to capsule applicator contents. Potential for exposure should not exist unless the capsule applicator broken. Use according to manufacturer's directions.
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Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	SDI (North America) Inc.	SDI Limited	SDI HOLDINGS PTY LTD DO
Address	1279 Hamilton Parkway Itasca IL 60143 United States	3-15 Brunsdon Street Bayswater VIC 3153 Australia	Rua Dr. Reinaldo Schmithausen 3141 – Cordeiros Itajaí – SC – CEP 88310-004 Brazil
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Emergency phone number

Association / Organisation	SDI Limited	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	131126 Poisons Information Centre	+1 855-237-5573 (ID#: 7941-93)
Other emergency telephone number(s)	+61 3 8727 7111	+61 3 9573 3188

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond





Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health Red = Fire Yellow = Reactivity White = Special (Oxidizer or water reactive substances)

Classification	Sensitisation (Skin) Category 1, Acute Toxicity (Inhalation) Category 4, Carcinogenicity Category 1A, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
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Label elements

Riva Applicator Pro

Hazard pictogram(s)	 
Signal word	Danger

Hazard statement(s)

H317	May cause an allergic skin reaction.
H332	Harmful if inhaled.
H350	May cause cancer.
H372	Causes damage to organs through prolonged or repeated exposure.
H412	Harmful to aquatic life with long lasting effects.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe dust/fume.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves and protective clothing.
P261	Avoid breathing dust/fumes.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing must not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P314	Get medical advice/attention if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		capsule applicator containing
12597-68-1	>99.9	<u>Stainless Steel</u>
Not Available		Stainless Steel grade 316L

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. ▶ Generally not applicable.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear.

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	<ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation. ▶ Generally not applicable.
Inhalation	<ul style="list-style-type: none"> ▶ 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. ▶ Generally not applicable.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice. ▶ Generally not applicable.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

- ▶ In cases of nickel poisoning, dimercaptol delivered by deep intramuscular injection may be a suitable antidote. (Patients should not exhibit renal or hepatic dysfunction.) The use of diethyldithiocarbamate is the subject of ongoing research.
- ▶ Irritant contact dermatoses or eczemas may respond to applications of weak antiseptic packs, antibiotic ointments (tetracycline or erythromycin) or inert pastes and ointments. Systemic antibiotics are advisable in the presence of lymphangitis or lymphadenitis.

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 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 Fire-fighting measures**Extinguishing media**

Metal dust fires need to be smothered with sand, inert dry powders.

DO NOT USE WATER, CO2 or FOAM.

- ▶ Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- ▶ Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- ▶ Chemical reaction with CO2 may produce flammable and explosive methane.
- ▶ If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.
- ▶ **DO NOT** use halogenated fire extinguishing agents.

Special hazards arising from the substrate or mixture

Fire Incompatibility	<ul style="list-style-type: none"> ▶ Reacts with acids producing flammable / explosive hydrogen (H2) gas None known.
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Special protective equipment and precautions for fire-fighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use. <p>Slight hazard when exposed to heat, flame and oxidisers.</p>
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ DO NOT disturb burning dust. Explosion may result if dust is stirred into a cloud, by providing oxygen to a large surface of hot metal. ▶ DO NOT use water or foam as generation of explosive hydrogen may result. <p>With the exception of the metals that burn in contact with air or water (for example, sodium), masses of combustible metals do not represent unusual fire risks because they have the ability to conduct heat away from hot spots so efficiently that the heat of combustion cannot be maintained - this means that it will require a lot of heat to ignite a mass of combustible metal. Generally, metal fire risks exist when sawdust, machine shavings and other metal 'fines' are present.</p> <p>Metal powders, while generally regarded as non-combustible:</p> <ul style="list-style-type: none"> ▶ May burn when metal is finely divided and energy input is high. ▶ May react explosively with water. ▶ May be ignited by friction, heat, sparks or flame. ▶ May REIGNITE after fire is extinguished. ▶ Will burn with intense heat. <p>Note:</p> <ul style="list-style-type: none"> ▶ Metal dust fires are slow moving but intense and difficult to extinguish. ▶ Containers may explode on heating. ▶ Dusts or fumes may form explosive mixtures with air. ▶ Gases generated in fire may be poisonous, corrosive or irritating. ▶ Hot or burning metals may react violently upon contact with other materials, such as oxidising agents and extinguishing agents used on fires involving ordinary combustibles or flammable liquids. ▶ Temperatures produced by burning metals can be higher than temperatures generated by burning flammable liquids

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- ▶ Some metals can continue to burn in carbon dioxide, nitrogen, water, or steam atmospheres in which ordinary combustibles or flammable liquids would be incapable of burning.

Decomposition may produce toxic fumes of:

metal oxides

May emit poisonous fumes.

May emit corrosive fumes.

Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.

Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.

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	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Secure load if safe to do so. ▶ Bundle/collect recoverable product. ▶ Collect remaining material in containers with covers for disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear 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. • Do not use compressed air to remove metal dusts 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. • Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations • Cover and reseal partially empty containers. • Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. If molten: <ul style="list-style-type: none"> ▶ Contain the flow using dry sand or salt flux as a dam. ▶ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. ▶ Allow the spill to cool before remelting scrap. ▶ Clean up all spills immediately. ▶ Wear protective clothing, safety glasses, dust mask, gloves. ▶ Secure load if safe to do so. Bundle/collect recoverable product. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▶ Water may be used to prevent dusting. ▶ Collect remaining material in containers with covers for disposal. ▶ Flush spill area with water. Minor hazard. <ul style="list-style-type: none"> ▶ Clear area of personnel. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Control personal contact with the substance, by using protective equipment as required. ▶ Prevent spillage from entering drains or water ways. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▶ Wash area and prevent runoff into drains or waterways. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<p>For molten metals:</p> <ul style="list-style-type: none"> • 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. <p>During melting operations, the following minimum guidelines should be observed:</p> <ul style="list-style-type: none"> • 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.
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	<ul style="list-style-type: none">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.<ul style="list-style-type: none">Avoid all personal contact, including inhalation.Wear protective clothing when risk of exposure occurs.Use in a well-ventilated area.Prevent concentration in hollows and sumps.DO NOT enter confined spaces until atmosphere has been checked.DO NOT allow material to contact humans, exposed food or food utensils.Avoid contact with incompatible materials.When handling, DO NOT eat, drink or smoke.Keep containers securely sealed when not in use.Avoid physical damage to containers.Always wash hands with soap and water after handling.Work clothes should be laundered separately. Launder contaminated clothing before re-use.Use good occupational work practice.Observe manufacturer's storage and handling recommendations contained within this SDS.Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none">Store away from incompatible materials.

Conditions for safe storage, including any incompatibilities

Suitable container	Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler. <ul style="list-style-type: none">Heavy gauge metal packages / Heavy gauge metal drums
Storage incompatibility	<ul style="list-style-type: none">Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
US OSHA Permissible Exposure Limits (PELs) Table Z-1	Stainless Steel	Particulates Not Otherwise Regulated (PNOR)- Total dust	15 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-1	Stainless Steel	Particulates Not Otherwise Regulated (PNOR)- Respirable fraction	5 mg/m3	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	Stainless Steel	Inert or Nuisance Dust: Respirable fraction	5 mg/m3 / 15 mppcf	Not Available	Not Available	Not Available
US OSHA Permissible Exposure Limits (PELs) Table Z-3	Stainless Steel	Inert or Nuisance Dust: Total Dust	15 mg/m3 / 50 mppcf	Not Available	Not Available	Not Available
US NIOSH Recommended Exposure Limits (RELs)	Stainless Steel	Particulates not otherwise regulated	Not Available	Not Available	Not Available	See Appendix D

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
Riva Applicator Pro	Not Available	Not Available	Not Available


Ingredient	Original IDLH	Revised IDLH
Stainless Steel	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none">Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.Open-vessel systems are prohibited.Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
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	<p>► Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.</p> <p>Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment.</p>
Individual protection measures, such as personal protective equipment	
Eye and face protection	<p>No special equipment required due to the physical form of the product.</p> <ul style="list-style-type: none"> ► Safety glasses with side shields. ► Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ► 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].
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ► Wear chemical protective gloves, e.g. PVC. ► Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ► 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 <p>No special equipment required due to the physical form of the product.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ► Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ► Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ► Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ► Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ► Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. <p>· 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.</p> <p>· 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.</p> <p>· 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).</p>

Respiratory protection

Respiratory protection not normally required due to the physical form of the product.

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.

Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Solid; insoluble in water.		
Physical state	Manufactured	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 Applicable
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	Not Available

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Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	<p>The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation, of the material, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress. Bronchial and alveolar exudate are apparent in animals exposed to molybdenum by inhalation. Molybdenum fume may produce bronchial irritation and moderate fatty changes in liver and kidney.</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p> <p>Manganese fume is toxic and produces nervous system effects characterised by tiredness. Acute poisoning is rare although acute inflammation of the lungs may occur. A chemical pneumonia may also result from frequent exposure. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.</p> <p>Regular exposure to nickel fume, as the oxide, may result in "metal fume fever" a sometimes debilitating upper respiratory tract condition resembling influenza.</p> <p>Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in closed or poorly ventilated areas. Pulmonary oedema, pulmonary fibrosis and asthma has been reported in welders using nickel alloys; level of exposure are generally not available and case reports are often confounded by mixed exposures to other agents.</p> <p>Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental</p>
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Riva Applicator Pro

	activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	<p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>Chrome fume, as the chrome VI oxide, is corrosive to the skin and may aggravate pre-existing skin conditions such as dermatitis and eczema. As a potential skin sensitiser, the fume may cause dermatoses to appear suddenly and without warning. Absorption of chrome VI compounds through the skin can cause systemic poisoning affecting the kidneys and liver.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	Although the material is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	<p>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.</p> <p>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.</p> <p>Substances that can cause 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 cause 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.</p> <p>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.</p> <p>On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature.</p> <p>Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC).</p> <p>Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).</p> <p>Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.</p> <p>Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.</p> <p>The most common toxic reaction to nickel is skin sensitisation which may produce a chronic eczema called "nickel itch". The first symptom is itching which occurs up to 7 days prior to the appearance of skin eruption. The primary skin eruption is erythematous or follicular and may be followed by superficial discrete ulcers (which discharge and become crusted), or eczema. In the chronic stages, pigmented or depigmented plaques may be formed. Recovery from the dermatitis usually occurs within 7 days but may take several weeks.</p> <p>Nickel dusts and several specific compounds are carcinogenic in animals following inhalation or parenteral administration (but not by ingestion or skin contact). Increases in lung and nasal cavity cancers have been observed amongst nickel workers in smelters and refineries. Respiratory cancer risks primarily relate to chronic exposure to soluble nickels at concentrations in excess of 1 mg Ni/m³ and exposure to the less soluble forms at concentrations greater than 10 mg Ni/m³. Metallic nickel does not appear to pose such a threat.</p> <p>Persons, exposed for long periods to molybdenum oxides, suffer from anaemia. Animals exposed to certain insoluble molybdenum compounds show anorexia, diarrhoea, weight loss, listlessness, and liver and kidney damage. Molybdenum disturbs bone metabolism, giving rise to lameness, bone joint abnormalities, osteoporosis and high serum phosphatase levels. Cattle, rabbits, and chicks on high dietary levels of molybdenum exhibited deformities of joints of the extremities. Low molybdenum intake has been attributed to the high incidence of oesophageal cancer in South Africa among the Bantu of Transkei, in China and in Russia.</p> <p>Chronic exposure of workers in Russian molybdenum-copper plant resulted in a fall in the albumin/globulin ratio owing to a rise in globulins (particularly alpha-globulins) which is interpreted as evidence of liver dysfunction with hyperbilirubinaemia. Hepatotoxic effects are also found in animals given molybdenum salts with a rise in alpha-globulin levels, hypoalbuminaemia and increased serum bilirubin reported. Other reported biochemical effects include an early depletive effect on tissue nicotinamide nucleotides, hyperaminoaciduria, reduction in red blood cell life-span and hyper-thyroidism. Industrial exposure to some insoluble molybdenum compounds is thought to have resulted in an increased incidence of weakness, fatigue, anorexia, headache and joint and muscular pain. Under the conditions of a 2-year inhalation study* there was equivocal evidence of carcinogenic activity of molybdenum trioxide in male rats, male mice and female mice based on a marginally significant positive trend of alveolar/bronchiolar adenoma or carcinoma. There was no evidence of carcinogenic activity in female rats exposed to 10, 30 and 100 mg/m³. Exposure of male and female rats to molybdenum trioxide by inhalation resulted in increased incidences of chronic alveolar inflammation, hyaline degeneration of the olfactory epithelium (females), hyaline degeneration of the respiratory epithelium and squamous metaplasia of the epiglottis. Exposure of female and male mice to molybdenum trioxide resulted in an increased incidence of metaplasia of the alveolar epithelium, histiocyte cellular inflammation (males), hyaline degeneration of the respiratory epithelium, hyaline degeneration of the olfactory epithelium (females), squamous metaplasia of the epiglottis, and hyperplasia of the larynx.</p> <p>Guinea pigs exposed to molybdenum trioxide dust for 1 hour daily at 250 mg/m³ showed extreme irritation. Symptoms include loss of appetite, weight loss, diarrhoea, muscular incoordination and loss of hair. Of the 51 animals exposed, 26 died after the tenth exposure. Exposure to freshly generated MoO₂ fume under about the same exposure conditions proved unexpectedly less toxic, with only 8.3% mortality compared with 51% mortality with the dust, and no mortality when the exposure level was reduced to about one-third (57 mg Mo/m³). Explanation for this unexpected finding was felt to reside in the more rapid solution and elimination of the large surface area fume particle. [Patty's]</p>

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Exposure of male and female rats to molybdenum trioxide resulted in the development of respiratory system lesions. In the lung, the incidence and severity of chronic alveolar inflammation increased with increasing exposure concentration in male and female rats. In some male rats, exposure to the material resulted in alveolar/ bronchiolar adenomas or carcinomas. Lesions in the nose (hyaline degeneration) and larynx (squamous metaplasia) were considered to be a non-specific defensive or adaptive response to chronic inhalation exposure. Inhalation exposure of mice to molybdenum trioxide was associated with the development of lung neoplasms and an increased incidence of alveolar/ bronchiolar carcinoma or adenoma in both sexes. Chronic inflammatory lesions were not present in the lungs. Lesions of the nose and larynx were similar to those observed in rats.

Molybdenum trioxide was not mutagenic in any of five strains of Salmonella typhimurium and did not induce sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells in vitro. All tests were conducted with or without S9 metabolic activation enzymes.

Pneumoconiosis has been described in experimental animals exposed sub-chronically to molybdenum trioxide.

The mechanism of molybdenum trioxide action in lung carcinogenicity is not known; the material is not mutagenic. Non-neoplastic lesions of the nose and larynx of rats and in the nose, larynx and lungs of mice were apparently due to the development of a more durable epithelium in response to chronic exposure.

The US Department of Health and Human Services (1) concluded that there was equivocal evidence of carcinogenic activity in male F344/N rats based on a marginally significant positive trend of alveolar/ bronchiolar adenoma or carcinoma; that there was no evidence of carcinogenic activity in female F344/N rats; that there was some evidence of carcinogenic activity in male B6C3F1 mice and that there was evidence of carcinogenic activity in female B6C3F1 mice

National Toxicology Program: Technical Report Series 462, April 1997

Exposure to fume containing high concentrations of water-soluble chromium (VI) during the welding of stainless steels in confined spaces has been reported to result in chronic chrome intoxication, dermatitis and asthma. Certain insoluble chromium (VI) compounds have been named as carcinogens (by the ACGIH) in other work environments. Chromium may also appear in welding fumes as Cr2O3 or double oxides with iron. These chromium (III) compounds are generally biologically inert.

Riva Applicator Pro	TOXICITY	IRRITATION
	Not Available	Not Available
Stainless Steel	TOXICITY	IRRITATION
	Not Available	Not Available

Legend:

1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

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 eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the 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.

For chrome(III) and other valence states (except hexavalent):

For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases.

The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells.

enter cells

The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter through these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals

Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures.

The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage.

Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms.

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 airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal 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.

No significant acute toxicological data identified in literature search.

Riva Applicator Pro

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

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

SECTION 12 Ecological information

Toxicity

Riva Applicator Pro	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Stainless Steel	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
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 allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For Metal:

Atmospheric Fate - Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air.

Environmental Fate: Environmental processes, such as oxidation, the presence of acids or bases and microbiological processes, may transform insoluble metals to more soluble ionic forms. Environmental processes may enhance bioavailability and may also be important in changing solubilities.

Aquatic/Terrestrial Fate: When released to dry soil, most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. A metal ion is considered infinitely persistent because it cannot degrade further. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms. Ionic species may bind to dissolved ligands or sorb to solid particles in water.

Ecotoxicity: Even though many metals show few toxic effects at physiological pH levels, transformation may introduce new or magnified effects.

Chromium in the oxidation state +3 (the trivalent form) is poorly absorbed by cells found in microorganisms, plants and animals. Chromate anions (CrO4-, oxidation state +6, the hexavalent form) are readily transported into cells and toxicity is closely linked to the higher oxidation state.

Chromium Ecotoxicology:

Toxicity in Aquatic Organisms:

Chromium is harmful to aquatic organisms in very low concentrations. Fish food organisms are very sensitive to low levels of chromium. Chromium is toxic to fish although less so in warm water. Marked decreases in toxicity are found with increasing pH or water hardness; changes in salinity have little if any effect. Chromium appears to make fish more susceptible to infection. High concentrations can damage and/or accumulate in various fish tissues and in invertebrates such as snails and worms.

Reproduction of Daphnia is affected by exposure to 0.01 mg/kg hexavalent chromium/litre

Toxicity of chromium in fresh-water organisms (50% mortality)*				
Compound	Category	Exposure	Toxicity Range (mg/litre)	Most sensitive species
hexavalent chrome	invertebrate	acute	0.067-59.9	scud
		long-term	-	-
	vertebrate	acute	17.6-249	fathead minnow
		long-term	0.265-2.0	rainbow trout
trivalent chrome	invertebrate	acute	2.0-64.0	cladoceran
		long-term	0.066	cladoceran
	vertebrate	acute	33.0-71.9	guppy
		long-term	1.0	fathead minnow

* from Environmental Health Criteria 61: WHO Publication.

Toxicity in Microorganisms:

In general, toxicity for most microorganisms occurs in the range of 0.05-5 mg chromium/kg of medium. Trivalent chromium is less toxic than the hexavalent form. The main signs of toxicity are inhibition of growth and the inhibition of various metabolic processes such as photosynthesis or protein synthesis. Gram-negative soil bacteria are generally more sensitive to hexavalent chromium (1-12 mg/kg) than the gram-positive types. Toxicity to trivalent chromium is not observed at similar levels. The toxicity of low levels of hexavalent chromium (1 mg/kg) indicates that soil microbial transformation, such as nitrification, may be affected. Chromium should not be introduced to municipal sewage treatment facilities.

Toxicity in Plants: Chromium in high concentrations can be toxic to plants. The main feature of chromium intoxication is chlorosis, which is similar to iron deficiency. Chromium affects carbohydrate metabolism and leaf chlorophyll concentration decreases with hexavalent chromium concentration (0.01-1 mg/l). The hexavalent form appears to more toxic than the trivalent species.

Biological half-life: The elimination curve for chromium, as measured by whole-body counting, has an exponential form. In rats, three different components of the curve have been identified, with half-lives of 0.5, 5.9 and 83.4 days, respectively.

Water Standards: Chromium is identified as a hazardous substance in the Federal (U.S.) Water Pollution Control Act and further regulated by Clean Air Water Act Amendments (US). These regulations apply to discharge. The US Primary drinking water Maximum Contaminant Level (MCL), for chromium, is 0.05 mg/l (total chromium). Since chromium compounds cannot volatilize from water, transport of chromium from water to the atmosphere is not likely, except by transport in windblown sea sprays. Most of the chromium released into water will ultimately be deposited in the sediment. A very small percentage of chromium can be present in water in both soluble and insoluble forms. Soluble chromium generally accounts for a very small percentage of the total chromium. Most of the soluble chromium is present as chromium(VI) and soluble chromium(III)

complexes. In the aquatic phase, chromium(III) occurs mostly as suspended solids adsorbed onto clayish materials, organics, or iron oxide (Fe2O3) present in water. Soluble forms and suspended chromium can undergo intramedia transport. Chromium(VI) in water will eventually be reduced to chromium(III) by organic matter in the water. The reduction of chromium(VI) and the oxidation of chromium(III) in water has been investigated. The reduction of chromium(VI) by S-2 or Fe+2 ions under anaerobic conditions was fast, and the reduction half-life ranged from instantaneous to a few days. However, the reduction of chromium(VI) by organic sediments and soils was much slower and depended on the type and amount of organic material and on the redox condition of the water. The reaction was generally faster under anaerobic than aerobic conditions. The reduction half-life of chromium(VI) in water with soil and sediment ranged from 4 to 140 day. Dissolved oxygen by itself in natural waters did not cause any measurable oxidation of chromium(III) to chromium(VI) in 128 days. When chromium(III) was added to lake water, a slow oxidation of chromium(III) to chromium(VI) occurred, corresponding to an oxidation half-life of nine years. The oxidation of chromium(III) to chromium(VI) during chlorination of water was highest in the pH range of 5.5-6.0. However, the process would rarely occur during chlorination of drinking water because of the low concentrations of chromium(III) in these waters, and the presence of naturally occurring organics that may protect chromium(III) from oxidation, either by forming strong complexes with chromium(III) or by acting as a reducing agent to free available chlorine. The bioconcentration factor (BCF) for chromium(VI) in rainbow trout (*Salmo gairdneri*) is 1. In bottom feeder bivalves, such as the oyster (*Crassostrea virginica*), blue mussel (*Mytilus edulis*), and soft shell clam (*Mya arenaria*), the BCF values for chromium(III) and chromium(VI) may range from 86 to 192. The bioavailability of chromium(III) to freshwater invertebrates (*Daphnia pulex*) decreased with the addition of humic acid. This decrease in bioavailability was attributed to lower availability of the free form of the metal due to its complexation with humic acid. Based on this information, chromium is not expected to biomagnify in the aquatic food chain. Although higher concentrations of chromium have been reported in plants growing in high chromium-containing soils (e.g., soil near ore deposits or chromium-emitting industries and soil fertilized by sewage sludge) compared with plants growing in normal soils, most of the increased uptake in plants is retained in roots, and only a small fraction is translocated in the aboveground part of edible plants. Therefore, bioaccumulation of chromium from soil to above-ground parts of plants is unlikely. There is no indication of biomagnification of chromium along the terrestrial food chain (soil-plant-animal). The fate of chromium in soil is greatly dependent upon the speciation of chromium, which is a function of redox potential and the pH of the soil. In most soils, chromium will be present predominantly in the chromium(III) state. This form has very low solubility and low reactivity resulting in low mobility in the environment and low toxicity in living organisms. Under oxidizing conditions chromium(VI) may be present in soil as CrO4²⁻ and HCrO4⁻. In this form, chromium is relatively soluble, mobile, and toxic to living organisms. In deeper soil where anaerobic conditions exist, chromium(VI) will be reduced to chromium(III) by S-2 and Fe+2 present in soil. The reduction of chromium(VI) to chromium(III) is possible in aerobic soils that contain appropriate organic energy sources to carry out the redox reaction. The reduction of chromium(VI) to chromium(III) is facilitated by low pH. From thermodynamic considerations, chromium(VI) may exist in the aerobic zone of some natural soil. The oxidation of chromium(III) to chromium(VI) in soil is facilitated by the presence of low oxidisable organic substances, oxygen, manganese dioxide, and moisture. Organic forms of chromium(III) (e.g., humic acid complexes) are more easily oxidised than insoluble oxides. Because most chromium(III) in soil is immobilized due to adsorption and complexation with soil materials, the barrier to this oxidation process is the lack of availability of mobile chromium(III) to immobile manganese dioxide in soil surfaces. Due to this lack of availability of mobile chromium(III) to manganese dioxide surfaces, a large portion of chromium in soil will not be oxidized to chromium(VI), even in the presence of manganese dioxide and favorable pH conditions. The microbial reduction of chromium(VI) to chromium(III) has been discussed as a possible remediation technique in heavily contaminated environmental media or wastes. Factors affecting the microbial reduction of chromium(VI) to chromium(III) include biomass concentration, initial chromium(VI) concentration, temperature, pH, carbon source, oxidation-reduction potential and the presence of both oxyanions and metal cations. Although high levels of chromium(VI) are toxic to most microbes, several resistant bacterial species have been identified which could ultimately be employed in remediation strategies. Chromium in soil is present mainly as insoluble oxide Cr2O3. nH2O, and is not very mobile in soil. A leachability study was conducted to study the mobility of chromium in soil. Due to different pH values, a complicated adsorption process was observed and chromium moved only slightly in soil. Chromium was not found in the leachate from soil, possibly because it formed complexes with organic matter. These results support previous data finding that chromium is not very mobile in soil. These results are supported by leachability investigation in which chromium mobility was studied for a period of 4 years in a sandy loam. The vertical migration pattern of chromium in this soil indicated that after an initial period of mobility, chromium forms insoluble complexes and little leaching is observed. Flooding of soils and the subsequent anaerobic decomposition of plant detritus matters may increase the mobilization of chromium(III) in soils due to formation of soluble complexes. This complexation may be facilitated by a lower soil pH. A smaller percentage of total chromium in soil exists as soluble chromium(VI) and chromium(III), which are more mobile in soil. The mobility of soluble chromium in soil will depend on the sorption characteristics of the soil. The relative retention of metals by soil is in the order of lead > antimony > copper > chromium > zinc > nickel > cobalt > cadmium. The sorption of chromium to soil depends primarily on the clay content of the soil and, to a lesser extent, on Fe2O3 and the organic content of soil. Chromium that is irreversibly sorbed onto soil, for example, in the interstitial lattice of goethite, FeOOH, will not be bioavailable to plants and animals under any condition. Organic matter in soil is expected to convert soluble chromate, chromium(VI), to insoluble chromium(III) oxide, Cr2O3. Chromium in soil may be transported to the atmosphere as an aerosol. Surface runoff from soil can transport both soluble and bulk precipitate of chromium to surface water. Soluble and unadsorbed chromium(VI) and chromium(III) complexes in soil may leach into groundwater. The leachability of chromium(VI) in the soil increases as the pH of the soil increases. On the other hand, lower pH present in acid rain may facilitate leaching of acid-soluble chromium(III) and chromium(VI) compounds in soil. Chromium has a low mobility for translocation from roots to aboveground parts of plants. However, depending on the geographical areas where the plants are grown, the concentration of chromium in aerial parts of certain plants may differ by a factor of 2-3. In the atmosphere, chromium(VI) may be reduced to chromium(III) at a significant rate by vanadium (V2+, V3+, and VO2+), Fe2+, HSO3-, and As3+. Conversely, chromium(III), if present as a salt other than Cr2O3, may be oxidized to chromium(VI) in the atmosphere in the presence of at least 1% manganese oxide.. However, this reaction is unlikely under most environmental conditions. The estimated atmospheric half-life for chromium(VI) reduction to chromium(III) was reported in the range of 16 hours to about 5 days. Based on the high concentration of molybdenum in all analysed waste types, the exposure of the environment to molybdenum is regarded as significant. The limited amount of data regarding its toxicity makes it impossible to evaluate the potential for adverse environmental and health effects from molybdenum exposure. Molybdenum cause adverse effects in ruminant animals. Livestock have been injured by forage grown on soils with excessive geochemically-derived molybdenum. Soil molybdenum is a potentially toxic element, but no cases have been reported of molybdenum toxicity to animals from consumption of forage grown on sludge-amended soils. In pot studies, where clover was grown on alkaline soils containing up to 16 kg of molybdenum per ha, concentrations in the plant tissue reached levels that could be harmful to animals if the clover were to make up a substantial portion of the diet for an extended period of time. Molybdenum is generally found in two oxidation states in nature, Mo(IV) and Mo(VI). In oxidising environments Mo(VI) dominates and it is commonly present as the oxyanion molybdate (MoO4²⁻). In a laboratory experiment it was found that Mo was lost from solution under reducing conditions and remobilised under oxidizing conditions, and hypothesised that MoS2, a low-solubility mineral, formed in the system. In this study it was also found that Fe minerals were important sinks for Mo accumulation in reducing sediments. It has been proposed that under reducing conditions and with the reduction of sulfate, molybdate is converted to thiomolybdate (MoS4²⁻) which then binds to Fe, Al, and organic matter phases a via sulfur bridges. This mechanism could also account for decreased Mo solubility under reducing conditions. Another study in wetland found that Mo accumulated in the sediments with most of the accumulation occurring in the top 2 cm and decreasing with depth. It appears that Mo accumulation (as well as As and V accumulation) or retention in the surface sediments is dependent on the depth of the overlying water column and correspondingly on redox status.

Transport and distribution of nickel particulates between different environmental compartments, is strongly influenced by particle size. Fine particulate matter has a longer residence time in the environment and is carried a long distance from its source; larger particles are deposited near the emission source. Atmospheric residence time for nickel particulates is estimated to be 5.4-7.9 days. Water solubility and bioavailability is affected by soil pH; decrease in pH generally mobilises nickel, thus acid rain can mobilise nickel from the soil and increase nickel concentrations in ground water. Nickel bioaccumulates in the food chain but is not bioconcentrated.

Drinking Water Standards:

Nickel 50 ug/l (UK max.)

20 ug/l (WHO guideline)

Soil Guidelines:

Dutch Criteria: 35 mg/kg (target)

210 mg/kg (intervention)

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient		Bioaccumulation
		No Data available for all ingredients
Mobility in soil		
Ingredient		Mobility
		No Data available for all ingredients

Other adverse effects
No evidence of ozone depleting properties were found in the current literature.

SECTION 13 Disposal considerations

Waste treatment methods	
Product / Packaging disposal	<ul style="list-style-type: none">Recycle wherever possible or consult manufacturer for recycling options.Consult State Land Waste Management Authority for disposal.

SECTION 14 Transport information

Labels Required	
Marine Pollutant	NO

Land transport (DOT): 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
14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code
Not Applicable
14.7.2. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
Stainless Steel	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
Stainless Steel	Not Available

SECTION 15 Regulatory information

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

Stainless Steel is found on the following regulatory lists	
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)	
US - Alaska Air Quality Control - Concentrations Triggering an Air Quality Episode for Air Pollutants Other Than PM-2.5	
US NIOSH Recommended Exposure Limits (RELs)	
US OSHA Permissible Exposure Limits (PELs) Table Z-1	
US OSHA Permissible Exposure Limits (PELs) Table Z-3	
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory	

Additional Regulatory Information
Not Applicable

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories	
Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No
Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No

Combustible Dust	No
Carcinogenicity	Yes
Acute toxicity (any route of exposure)	Yes
Reproductive toxicity	No
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	Yes
Serious eye damage or eye irritation	No
Specific target organ toxicity (single or repeated exposure)	Yes
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)
None Reported

US. EPCRA Section 313 Toxic Release Inventory (TRI) (40 CFR 372)
None Reported

Additional Federal Regulatory Information
Not Applicable

State Regulations

US. California Proposition 65
None Reported

Additional State Regulatory Information
Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (Stainless Steel)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (Stainless Steel)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (Stainless Steel)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (Stainless Steel)
Vietnam - NCI	Yes
Russia - FBEPH	No (Stainless Steel)
Legend:	<i>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.</i>

SECTION 16 Other information

Revision Date	19/03/2025
Initial Date	19/03/2025

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	19/03/2025	Composition / information on ingredients - Ingredients

Other information

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

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

Definitions and abbreviations

- PC - TWA: Permissible Concentration-Time Weighted Average
- PC - STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists

Riva Applicator Pro

- 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
 - DNEL: Derived No-Effect Level
 - PNEC: Predicted no-effect concentration
 - MARPOL: International Convention for the Prevention of Pollution from Ships
 - IMSBC: International Maritime Solid Bulk Cargoes Code
 - IGC: International Gas Carrier Code
 - IBC: International Bulk Chemical Code
-
- 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
 - 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

The information contained in the Safety Data Sheet is based on data considered to be accurate, however, no warranty is expressed or implied regarding the accuracy of the data or the results to be obtained from the use thereof.

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