

251408Y - Vario Ammonia Salicylate F10 ml

YSI Inc.

Part Number: 251408Y
Version No: 1.1
Safety Data Sheet according to OSHA HazCom Standard (2024) requirements

Initial Date: 03/15/2019
Revision Date: 03/15/2019
Print Date: 03/13/2026
S.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	251408Y - Vario Ammonia Salicylate F10 ml
Chemical Name	Not Applicable
Synonyms	251408Y
Chemical formula	Not Applicable
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Reagent for water analysis
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Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	YSI Inc.	GFS Chemicals, Inc.
Address	1700/1725 Brannum Ln Yellow Springs OH 45387 United States	PO Box 245 Powell OH 43065 United States
Telephone	(937) 767-7241	740-881-5501 800-858-9682
Fax	Not Available	740-881-5989
Website	www.ysi.com	www.gfschemicals.com
Email	MSDSinfo@ysi.com	service@gfschemicals.com

Emergency phone number

Association / Organisation	CHEMTREC	ChemTrec
Emergency telephone number(s)	(800) 424-9300	800-424-9300
Other emergency telephone number(s)	011 703-527-3887	Not Available

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

Considered a Hazardous Substance by the 2012 OSHA Hazard Communication Standard (29 CFR 1910.1200). Not classified as Dangerous Goods for transport purposes.

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 and White = Special (Oxidizer or water reactive substances)

Classification	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3
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Label elements

Hazard pictogram(s)	
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Signal word	Danger
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Hazard statement(s)

H302	Harmful if swallowed.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P271	Use only outdoors or in a well-ventilated area.
P261	Avoid breathing dust/fumes.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P280	Wear protective gloves, protective clothing, eye protection and face protection.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P330	Rinse mouth.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

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.
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No further product hazard information.

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
54-21-7	40-50	<u>sodium salicylate</u>
13755-38-9	<2.5	<u>sodium nitroprusside</u>

SECTION 4 First-aid measures**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▶ Quickly remove all contaminated clothing, including footwear. ▶ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▶ Transport to hospital, or doctor.
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, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ For advice, contact a Poisons Information Centre or a doctor at once. ▶ Urgent hospital treatment is likely to be needed. ▶ If swallowed do NOT induce vomiting.

Continued...

- ▶ 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.
- ▶ Transport to hospital or doctor without delay.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

for salicylate intoxication:

- Pending gastric lavage, use emetics such as syrup of Ipecac or delay gastric emptying and absorption by swallowing a slurry of activated charcoal. **Do not give ipecac after charcoal.**
- Gastric lavage with water or perhaps sodium bicarbonate solution (3%-5%). Mild alkali delays salicylate absorption from the stomach and perhaps slightly from the duodenum.
- Saline catharsis with sodium or magnesium sulfate (15-30 gm in water).
- Take an immediate blood sample for an appraisal of the patient's acid-base status. A pH determination on an anaerobic sample of arterial blood is best. An analysis of the plasma salicylate concentration should be made at the same time. Laboratory controls are almost essential for the proper management of severe salicylism.
- In the presence of an established acidosis, alkali therapy is essential, but at least in an adult, alkali should be withheld until its need is demonstrated by chemical analysis. The intensity of treatment depends on the intensity of acidosis. In the presence of vomiting, intravenous sodium bicarbonate is the most satisfactory of all alkali therapy.
- Correct dehydration and hypoglycaemia (if present) by the intravenous administration of glucose in water or in isotonic saline. The administration of glucose may also serve to remedy ketosis which is often seen in poisoned children.
- Even in patients without hypoglycaemia, infusions of glucose adequate to produce distinct hyperglycaemia are recommended to prevent glucose depletion in the brain. This recommendation is based on impressive experimental data in animals.
- Renal function should be supported by correcting dehydration and incipient shock. Overhydration is not justified. An alkaline urine should be maintained by the administration of alkali if necessary with care to prevent a severe systemic alkalosis. As long as urine remains alkaline (pH above 7.5), administration of an osmotic diuretic such as mannitol or perhaps THAM is useful, but one must be careful to avoid hypokalaemia. Supplements of potassium chloride should be included in parenteral fluids.
- Small doses of barbiturates, diazepam, paraldehyde, or perhaps other sedatives (but probably not morphine) may be required to suppress extreme restlessness and convulsions.
- For hyperpyrexia, use sponge baths.
- The presence of petechiae or other signs of haemorrhagic tendency calls for a large Vitamin K dose and perhaps ascorbic acid. Minor transfusions may be necessary since bleeding in salicylism is not always due to a prothrombin effect.
- Haemodialysis and haemoperfusion have proved useful in salicylate poisoning, as have peritoneal dialysis and exchange transfusions, but alkaline diuretic therapy is probably sufficient except in fulminating cases.

[GOSSELIN, et.al.: *Clinical Toxicology of Commercial Products*]

The mechanism of the toxic effect involves metabolic acidosis, respiratory alkalosis, hypoglycaemia, and potassium depletion. Salicylate poisoning is characterised by extreme acid-base disturbances, electrolyte disturbances and decreased levels of consciousness. There are differences between acute and chronic toxicity and a varying clinical picture which is dependent on the age of the patient and their kidney function. The major feature of poisoning is metabolic acidosis due to "uncoupling of oxidative phosphorylation" which produces an increased metabolic rate, increased oxygen consumption, increased formation of carbon dioxide, increased heat production and increased utilisation of glucose. Direct stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. This leads to compensatory increased renal excretion of bicarbonate which contributes to the metabolic acidosis which may coexist or develop subsequently. Hypoglycaemia may occur as a result of increased glucose demand, increased rates of tissue glycolysis, and impaired rate of glucose synthesis. **NOTE:** Tissue glucose levels may be lower than plasma levels. Hyperglycaemia may occur due to increased glycogenolysis. Potassium depletion occurs as a result of increased renal excretion as well as intracellular movement of potassium. Salicylates competitively inhibit vitamin K dependent synthesis of factors II, VII, IX, X and in addition, may produce a mild dose dependent hepatitis. Salicylates are bound to albumin. The extent of protein binding is concentration dependent (and falls with higher blood levels). This, and the effects of acidosis, decreasing ionisation, means that the volume of distribution increases markedly in overdose as does CNS penetration. The extent of protein binding (50-80%) and the rate of metabolism are concentration dependent. Hepatic clearance has zero order kinetics and thus the therapeutic half-life of 2-4.5 hours but the half-life in overdose is 18-36 hours. Renal excretion is the most important route in overdose. Thus when the salicylate concentrations are in the toxic range there is increased tissue distribution and impaired clearance of the drug.

HyperTox 3.0 <https://www.ozemail.com.au/-ouad/SAL10001.HTA>

For cyanide intoxication (and for certain nitriles which produce cyanide ion)

- ▶ Signs symptoms of acute cyanide poisoning reflect cellular hypoxia and are often non-specific.
- ▶ Cyanosis may be a late finding.
- ▶ A *bradycardic*, hypertensive and tachypneic patient suggests poisoning especially if CNS and cardiovascular depression subsequently occurs.
- ▶ Immediate attention should be directed towards assisted ventilation, administration of 100% oxygen, insertion of intravenous lines and institution of cardiac monitoring.
- ▶ Obtain an arterial blood gas immediately and correct any severe metabolic acidosis (pH below 7.15).
- ▶ Mildly symptomatic patients generally require supportive care alone. Nitrites should not be given indiscriminately - in all cases of moderate to severe poisoning, they should be given in conjunction with thiosulfate. As a temporizing measure supply amyl nitrite perles (0.2ml inhaled 30 seconds every minute) until intravenous lines for sodium nitrite are established. 10 ml of a 3% solution is administered over 4 minutes to produce 20% methaemoglobin in adults. Follow directly with 50 ml of 25% sodium thiosulfate, at the same rate, IV. If symptoms reappear or persist within 1/2-1 hour, repeat nitrite and thiosulfate at 50% of initial dose. As the mode of action involves the metabolic conversion of the thiosulfate to thiocyanate, renal failure may enhance thiocyanate toxicity.
- ▶ Methylene blue is not an antidote. [Ellenhorn and Barceloux: Medical Toxicology]

If amyl nitrite intervention is employed then Medical Treatment Kits should contain the following:

- ▶ One box containing one dozen amyl nitrite ampoules
- ▶ Two sterile ampoules of sodium nitrite solution (10 mL of a 3% solution in each)
- ▶ Two sterile ampoules of sodium thiosulfate solution (50 mL of a 25% solution in each)
- ▶ One 10 mL sterile syringe. One 50 mL sterile syringe. Two sterile intravenous needles. One tourniquet.
- ▶ One dozen gauze pads.
- ▶ Latex gloves
- ▶ A "Biohazard" bag for disposal of bloody/contaminated equipment.
- ▶ A set of cyanide instructions on first aid and medical treatment.

- Notes on the use of amyl nitrite:-

- ▶ AN is highly volatile and flammable - do not smoke or use around a source of ignition.
- ▶ If treating patient in a windy or draughty area provide some shelter or protection (shirt, wall, drum, cupped hand etc.) to prevent amyl nitrite vapour from being blown away. Keep ampoule upwind from the nose, the objective is to get amyl nitrite into the patients lungs.
- ▶ Rescuers should avoid AN inhalation to avoid becoming dizzy and losing competence.
- ▶ Lay the patient down. Since AN dilates blood vessels and lowers blood pressure, lying down will help keep patient conscious.
- ▶ **DO NOT overuse - excessive use might put the patient into shock.** Experience at DuPont plants has not shown any serious after-effects from treatment with amyl nitrite.

ADDITIONAL NOTES:

- ▶ Major medical treatment procedures may vary e.g. US (FDA method as recommended by DuPont) uses amyl nitrite as a methaemoglobin generator, followed by treatment with sodium nitrite and then sodium thiosulfate.

MODES OF ACTION: Amyl nitrite (AN) reacts with haemoglobin (HB) to form about 5% methaemoglobin (MHB). Sodium nitrite (NaNO₂) reacts with haemoglobin to form approximately 20-30% methaemoglobin. Methaemoglobin attracts cyanide ions (CN) from tissue and binds with them to become cyanmethaemoglobin (CNMHB). Sodium thiosulfate (Na₂S₂O₃) converts cyanmethaemoglobin to thiocyanate (HSCN) which is excreted by the kidneys. i.e. AN + HB = MHB NaNO₂ + HB = MHB CN + MHB = CNMHB Na₂S₂O₃ + CNMHB + O₂ = HSCN

- ▶ The administration of the antidote salts is intravenous in normal saline, Ringers lactate or other available IV fluid.
- ▶ European practice may use 4-dimethylaminophenol (DMAP) as a methaemoglobin generator. Also hydroxycobalamin (Vitamin B12a) is used. Hydroxycobalamin works by reacting with cyanide to form cyanocobalamin (Vitamin B12) which is excreted in the urine.

- ▶ European and Australian NOHSC (ASCC) propose dicobalt edetate (Kelocyanor) as antidote. This acts by chelating cyanide to form stable cobalticyanide, which is excreted in the urine. In all cases hyperbaric therapy may increase the efficiency of a cyanide antidote kit.

for non-steroidal anti-inflammatories (NSAIDs)

- ▶ Symptoms following acute NSAIDs overdoses are usually limited to lethargy, drowsiness, nausea, vomiting, and epigastric pain, which are generally reversible with supportive care. Gastrointestinal bleeding can occur. Hypertension, acute renal failure, respiratory depression, and coma may occur, but are rare. Anaphylactoid reactions have been reported with therapeutic ingestion of NSAIDs, and may occur following an overdose.
- ▶ Patients should be managed by symptomatic and supportive care following a NSAIDs overdose.
- ▶ There are no specific antidotes.
- ▶ Emesis and/or activated charcoal (60 to 100 grams in adults, 1 to 2 g/kg in children), and/or osmotic cathartic may be indicated in patients seen within 4 hours of ingestion with symptoms or following a large overdose (5 to 10 times the usual dose).
- ▶ Forced diuresis, alkalinisation of urine, hemodialysis, or haemoperfusion may not be useful due to high protein binding.
- ▶ For gastrointestinal haemorrhage, monitor stool guaiac and administer antacids or sucralfate.
- ▶ For mild/moderate allergic reactions, administer antihistamines with or without inhaled beta agonists, corticosteroids, or epinephrine.
- ▶ For severe allergic reactions, administer oxygen, antihistamines, epinephrine, or corticosteroids. Nephritis or nephrotic syndrome, thrombocytopenia, or haemolytic anemia may respond to glucocorticoid administration.
- ▶ For severe acidosis, administer sodium bicarbonate.
- ▶ Administer as required: plasma volume expanders for severe hypotension; diazepam or other benzodiazepine for convulsions; vitamin K1 for hypoprothrombinaemia; and/or dopamine plus dobutamine intravenously to prevent or reverse early indications of renal failure.

Serious gastrointestinal toxicity, such as bleeding, ulceration, and perforation, can occur at any time, with or without warning symptoms, in patients treated chronically with NSAID therapy. Although minor upper gastrointestinal problems, such as dyspepsia, are common, usually developing early in therapy, physicians should remain alert for ulceration and bleeding in patients treated chronically with NSAIDs even in the absence of previous GI tract symptoms. In patients observed in clinical trials of several months to two years duration, symptomatic upper GI ulcers, gross bleeding or perforation appear to occur in approximately 1% of patients treated for 3 to 6 months, and in about 2% to 4% of patients treated for one year. Physicians should inform patients about the signs and/or symptoms of serious GI toxicity and what steps to take if they occur. Studies to date have not identified any subset of patients not at risk of developing peptic ulceration and bleeding. Except for a prior history of serious GI events and other risk factors known to be associated with peptic ulcer disease, such as alcoholism, smoking, etc., no risk factors (e.g., age, sex) have been associated with increased risk. Elderly or debilitated patients seem to tolerate ulceration or bleeding less well than other individuals, and most spontaneous reports of fatal GI events are in this population. Studies to date are inconclusive concerning the relative risk of various NSAIDs in causing such reactions. High doses of any NSAID probably carry a greater risk of these reactions, although controlled clinical trials showing this do not exist in most cases. In considering the use of relatively large doses (within the recommended dosage range), sufficient benefit should be anticipated to offset the potential increased risk of GI toxicity.

For acute or short-term repeated exposures to highly alkaline materials:

- ▶ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- ▶ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.

* Activated charcoal does not absorb alkali.

* Gastric lavage should not be used.

Supportive care involves the following:

- ▶ Withhold oral feedings initially.
- ▶ If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- ▶ Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- ▶ Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

SECTION 5 Fire-fighting measures

Extinguishing media

- ▶ Water spray or fog.
- ▶ Foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility

- ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

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. ▶ 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	<p>Combustible. Will burn if ignited. Combustion products include:</p> <ul style="list-style-type: none"> ▶ carbon monoxide (CO) ▶ carbon dioxide (CO₂) ▶ other pyrolysis products typical of burning organic material. <p>May emit poisonous fumes. May emit corrosive fumes.</p>

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	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> ▶ Clean up waste regularly and abnormal spills immediately. ▶ Avoid breathing dust and contact with skin and eyes. ▶ Wear protective clothing, gloves, safety glasses and dust respirator. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (H-Class HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). H-Class HEPA filtered industrial vacuum cleaners should NOT be used on wet materials or surfaces. ▶ Dampen with water to prevent dusting before sweeping. ▶ Place in suitable containers for disposal.
Major Spills	<p>Environmental hazard - contain spillage. Moderate hazard.</p> <ul style="list-style-type: none"> ▶ CAUTION: Advise personnel in area. ▶ Alert Emergency Services and tell them location and nature of hazard. ▶ Control personal contact by wearing protective clothing. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Recover product wherever possible. ▶ IF DRY: Use dry clean up procedures and avoid generating dust. Collect residues and place in sealed plastic bags or other containers for disposal. IF WET: Vacuum/shovel up and place in labelled containers for disposal. ▶ ALWAYS: Wash area down with large amounts of water and prevent runoff into drains. ▶ If contamination of drains or waterways occurs, advise Emergency Services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid skin 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 come in direct contact with human skin or eyes. ▶ DO NOT allow material to come in contact with exposed food or food contact surfaces. ▶ Suitable PPE must be worn at all times. ▶ 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 in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry area protected from environmental extremes. ▶ 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. <p>For major quantities:</p> <ul style="list-style-type: none"> ▶ Consider storage in bunded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams). ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Glass container is suitable for laboratory quantities ▶ Polyethylene or polypropylene container. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid strong acids, bases. ▶ 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	sodium nitroprusside	Cyanides (as CN)	5 mg/m3	Not Available	Not Available	Skin designation
US NIOSH Recommended Exposure Limits (RELs)	sodium nitroprusside	Iron salts (soluble, as Fe)	1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Continued...

Ingredient	TEEL-1	TEEL-2	TEEL-3
251408Y - Vario Ammonia Salicylate F10 ml	Not Available	Not Available	Not Available
Ingredient	Original IDLH	Revised IDLH	
sodium salicylate	Not Available	Not Available	
sodium nitroprusside	25 mg/m3	Not Available	

Exposure controls

Appropriate engineering controls	<p>Enclosed local exhaust ventilation is required at points of dust, fume or vapour generation. HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. Barrier protection or laminar flow cabinets should be considered for laboratory scale handling. A fume hood or vented balance enclosure is recommended for weighing/ transferring quantities exceeding 500 mg. When handling quantities up to 500 gram in either a standard laboratory with general dilution ventilation (e.g. 6-12 air changes per hour) is preferred. Quantities up to 1 kilogram may require a designated laboratory using fume hood, biological safety cabinet, or approved vented enclosures. Quantities exceeding 1 kilogram should be handled in a designated laboratory or containment laboratory using appropriate barrier/ containment technology. Manufacturing and pilot plant operations require barrier/ containment and direct coupling technologies. Barrier/ containment technology and direct coupling (totally enclosed processes that create a barrier between the equipment and the room) typically use double or split butterfly valves and hybrid unidirectional airflow/ local exhaust ventilation solutions (e.g. powder containment booths). Glove bags, isolator glove box systems are optional. HEPA filtration of exhaust from dry product handling areas is required. Fume-hoods and other open-face containment devices are acceptable when face velocities of at least 1 m/s (200 feet/minute) are achieved. Partitions, barriers, and other partial containment technologies are required to prevent migration of the material to uncontrolled areas. For non-routine emergencies maximum local and general exhaust are necessary. 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.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 80%;">Type of Contaminant:</th> <th style="width: 20%;">Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, etc. evaporating from tank (in still air)</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 50%;">Lower end of the range</th> <th style="width: 50%;">Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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.</p> <p>The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated: Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated.</p> <p>The following protective devices are recommended where exposures exceed the recommended exposure control guidelines by factors of:</p> <ul style="list-style-type: none"> 10; high efficiency particulate (HEPA) filters or cartridges 10-25; loose-fitting (Tyvek or helmet type) HEPA powered-air purifying respirator. 25-50; a full face-piece negative pressure respirator with HEPA filters 50-100; tight-fitting, full face-piece HEPA PAPR 100-1000; a hood-shroud HEPA PAPR or full face-piece supplied air respirator operated in pressure demand or other positive pressure mode. 	Type of Contaminant:	Air Speed:	solvent, vapours, etc. evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min.)	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)	direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)	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
	Type of Contaminant:	Air Speed:																	
	solvent, vapours, etc. evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min.)																	
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)																	
direct spray, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)																		
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																		
Individual protection measures, such as personal protective equipment																			
Eye and face protection	<p>When handling very small quantities of the material eye protection may not be required. For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs:</p> <ul style="list-style-type: none"> ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Face shield. Full face shield may be required for supplementary but never for primary protection of eyes. ▶ 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"> ▶ Elbow length PVC gloves <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. ▶ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▶ Double gloving should be considered. ▶ PVC gloves. ▶ Change gloves frequently and when contaminated, punctured or torn. ▶ Wash hands immediately after removing gloves. ▶ Protective shoe covers. [AS/NZS 2210] 																		

	▶ Head covering.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ For quantities up to 500 grams a laboratory coat may be suitable. ▶ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▶ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▶ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▶ Eye wash unit. ▶ Ensure there is ready access to an emergency shower. ▶ For Emergencies: Vinyl suit

Respiratory protection

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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	White		
Physical state	Divided Solid Powder	Relative density (Water = 1)	1.2
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	8.1	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 Available
Flash point (°C)	250	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	41.2 g/l
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	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information**Information on toxicological effects**

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.

251408Y - Vario Ammonia Salicylate F10 ml

h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	The material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage. Inhaling corrosive bases may irritate the respiratory tract. Symptoms include cough, choking, pain and damage to the mucous membrane.
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. Ingestion of alkaline corrosives may produce burns around the mouth, ulcerations and swellings of the mucous membranes, profuse saliva production, with an inability to speak or swallow. Both the oesophagus and stomach may experience burning pain; vomiting and diarrhoea may follow. High oral doses of salicylates, such as aspirin, may cause a mild burning pain in the throat and stomach, causing vomiting. This is followed (within hours) by deep, rapid breathing, tiredness, nausea and further vomiting, thirst and diarrhoea. Non-steroidal anti-inflammatory drug (NSAID) overdose may produce nausea, vomiting, indigestion and upper abdominal pain. Other effects may include drowsiness, dizziness, confusion, disorientation, lethargy, "pins and needles", intense headache, blurred vision, ringing in the ears, muscle twitching, convulsions, stupor and coma.
Skin Contact	The material can produce severe chemical burns following direct contact with the skin. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions 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. This material can cause inflammation of the skin on contact in some persons.
Eye	If applied to the eyes, this material causes severe eye damage. Direct eye contact with corrosive bases can cause pain and burns. There may be swelling, epithelium destruction, clouding of the cornea and inflammation of the iris. Mild cases often resolve; severe cases can be prolonged with complications such as persistent swelling, scarring, permanent cloudiness, bulging of the eye, cataracts, eyelids glued to the eyeball and blindness.
Chronic	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Long-term exposure to respiratory irritants may result in airways disease, involving difficulty breathing and related whole-body problems. Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure. There is some evidence that inhaling this product is more likely to cause a sensitisation reaction in some persons compared to the general population. There is limited evidence that, skin contact with this product is more likely to cause a sensitisation reaction in some persons compared to the general population. There is some evidence from animal testing that exposure to this material may result in toxic effects to the unborn baby. Prolonged use of non-steroidal analgesics damages the lining of the gastrointestinal tract, causing ulcers and bleeding. There may be diarrhoea or constipation, perforations causing serious infection, and blood in the vomit or stools. Chronic exposure to salicylates produce problems with metabolism, central nervous system disturbances, or kidney damage. Those with pre-existing damage to the eye, skin or kidney are especially at risk. Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).

251408Y - Vario Ammonia Salicylate F10 ml	TOXICITY	IRRITATION
	Not Available	Not Available
sodium salicylate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (Rodent - rabbit): 100mg
	Oral (Rat) LD50: 1200 mg/kg ^[2]	Eye (Rodent - rabbit): 105mg/7D - Severe
		Eye: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
sodium nitroprusside	TOXICITY	IRRITATION
	Oral (Mouse) LD50: 61 mg/kg ^[2]	Not Available

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

SODIUM SALICYLATE	<p>Pregnant female rats were treated with the test chemical by oral gavage at 0, 20, 80 and 200 mg/kg bw/day from day 15 to 21 of gestation. Twenty-five rats were used per dose level up to 80 mg/kg whereas sixteen rats were used at 200 mg/kg. No significant effects were observed on gestational body weight gain and no signs of toxicity were observed until onset of delivery. Treatment at 200 mg/kg resulted in a significant increase in labor duration compared to control data (mean, ~3.5 hours at 200 mg/kg vs. mean, ~1.2 hours at 0 mg/kg). Gestation length was unaffected by treatment. A non-statistical increase in fetal peripartum deaths was reported at 200 mg/kg compared to the control group (9.7% fetuses affected at 200 mg/kg vs 3.1% of fetuses affected at 0 mg/kg). No significant effects were observed on mean pups per litter, live pups per litter, mean pup weight, or sex ratio. No external visible abnormalities were observed in any of the pups that survived delivery. Gross examination was not performed on fetuses that died peripartum. The incidence of maternal perinatal death was significantly increased at 200 mg/kg compared to the control group. That is, 4 of 10 animals at 200 mg/kg died or had to be sacrificed due to extreme distress whilst only 1 of 21 animals treated at 0 mg/kg died perinatally. NOAEL was considered to be 80 mg/kg/day when pregnant female Sprague Dawley rats were treated with the test chemical by oral gavage. LOAEL was considered at 200 mg/kg bw/day based on increased labor duration and increased maternal perinatal lethality. The test chemical tested negative for mutagenicity in CHO cells in the absence of metabolic activation. No conclusions could be drawn regarding the mutagenicity of the chemical in CHO in the presence of metabolic activation due to invalid positive control data.</p> <p>Inhibition of NF-κB in vivo can be detrimental. NF-κB controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF-κB activity.</p> <p>The same functions that make NF-κB attractive for developing inhibitors for treating disease also play a role in homeostasis, and disruption of the NF-κB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.</p> <p>NF-κB plays a role in multiple homeostatic cellular processes including response to stimuli, cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such as p38 and JNK. In addition to mediating proinflammatory responses, NF-κB may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF-κB can have adverse consequences such as immune suppression and tissue damage.</p> <p>Understanding the consequences of lack of NF-κB activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF-κB resulting in defects in</p>
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251408Y - Vario Ammonia Salicylate F10 ml

development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-κB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IκBα. The IKK gamma mutations result in a defective IKK complex and the IκBα mutation results in an IκBα protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-κB activation and ectodermal dysplasia with immunodeficiency. In general patients with these genetic defects have multiple immunological defects including impaired innate immunity, impaired antibody production, and ultimately severe bacterial infections. Understanding the immune defects and susceptibilities in patients with genetic defects in the NF-κB pathway will help prepare for potential adverse effects of pharmacologic NF-κB inhibitors

The requirement for NF-κB in the development and maintenance of the immune system is well documented. NF-κB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-κB or the IKKβ gene results in death during fetal development primarily due to massive liver apoptosis

Fetal liver stem cells from p65 or IKKβ deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-κB for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF-κB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-κB have implications for therapeutic potential use in humans. The double-sided nature of NF-κB inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations. In addition to controlling lymphocyte development, NF-κB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-κB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-κB through phosphorylation of CARMA1 by PKC θ and PKC β respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF-κB plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs). TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF-κB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-κB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-κB is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF-κB activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation. Given the diverse roles NF-κB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-κB pathway are susceptible to parasitic and bacterial infection.

The role of NF-κB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-κB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-κB targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator. Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF-κB activation. Accumulated studies have proved that non-steroidal anti-inflammatory drugs (NSAIDs) which block inflammation by their actions on arachidonic acid (AA) metabolism have a potential role in cancer chemotherapy and chemoprevention.

There is a general acceptance that NSAIDs induce colon cancer in humans. One suggested reason is that the balance between COX and lipoxygenase (LOX) activity determines tumorigenesis critically. Under low COX activity, arachidonic acid released from cell membranes in response to external stimuli is preferentially metabolized by LOX enzymes. The oxygenated lipids (metabolites) produced by LOXs initiate subsequent biological reactions, activate cellular signaling mechanisms through specific cell surface receptors, or are further metabolized into potent lipid mediators.

There is evidence that a 15-LOX metabolite 13S-HPODE (13S-hydroperoxyoctadecaenoic acid) generated from linoleic acid induces apoptosis in colon cancer.

Ingestion of aspirin or other NSAIDs may elicit respiratory, nasal, and gastrointestinal symptoms, as well as dermal changes in a subset of patients with asthma. The sensitivity to cyclooxygenase (COX) inhibitors has led to the hypothesis that NSAIDs may be causing upregulation of the 5-lipoxygenase pathway and its attendant products, the leukotrienes, in these patients. It has been shown increase in urinary leukotriene E 4 (LTE4) after aspirin ingestion or inhalation of lysine-aspirin in aspirin-sensitive patients with asthma. It has also been demonstrated that pharmacologic blockade at the level of the cysteinyl leukotriene receptor(s) can blunt the bronchospastic response to aspirin. Cysteinyl leukotrienes are potent bronchoconstrictors, induce mucus secretion, and increase vascular permeability. Importantly, inhibition of 5-lipoxygenase blocks not only the respiratory but also the gastrointestinal and dermal reactions to aspirin in aspirin-sensitive patients with asthma. Although these results establish the importance of 5-lipoxygenase products in mediating reactions to aspirin, the cellular source and mechanism of release of these mediators remain unclear.

Mast cells, which are a known source of leukotrienes, are activated in the nasal response to aspirin as demonstrated by the detection of nasal tryptase after aspirin challenge. Tryptase is an enzyme specific to mast cells and is an indicator of mast cell activation. Cysteinyl leukotrienes and histamine, which can be produced by mast cells, were detected as well. The occurrence of nasal symptoms, as well as activation of mast cells, in response to aspirin was blocked by zileuton, an inhibitor of 5-lipoxygenase. This confirms that 5-lipoxygenase products are critical to the development of aspirin-induced asthma (ASA-induced) reactions in the nose. It also suggests that 5-lipoxygenase products may have a role in the activation of mast cells during this reaction.

251408Y - Vario Ammonia Salicylate F10 ml & SODIUM SALICYLATE

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.

The salicylates are well absorbed by mouth, and oral bioavailability is assumed to be total. In humans, absorption through skin is more limited. The salicylates are expected to be broken down to salicylic acid, mostly in the liver, and then conjugated with glycine or glucuronide and excreted in the urine. The expected metabolism of the salicylates do not present toxicological concerns. Animal testing shows that acute toxicity by skin contact is very low, while acute toxicity by mouth is moderate. Salicylates do not possess genetic toxicity, and generally do not have the potential to cause cancer. The reproductive and developmental toxicity data on methyl salicylate shows that high doses which are toxic to the mother may cause toxicity to the embryo and birth defects. At concentrations likely to be encountered through their use as fragrance ingredients, salicylates are considered to be non-irritating to the skin. The salicylates in general have no, or very limited, potential to sensitize skin. They do not possess light-mediated toxicity and do not cause light-mediated irritation or allergies.

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

Continued...

251408Y - Vario Ammonia Salicylate F10 ml	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

sodium salicylate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	75.25mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	96h	Fish	1mg/l	4
	LC50	96h	Fish	>100mg/l	2

sodium nitroprusside	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	48h	Crustacea	<210mg/L	4

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

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

Soil Guidelines: Dutch Criteria:

free cyanide: 1 mg/kg (target)

20 mg/kg (intervention)

complex cyanide (pH 5): 5 mg/kg (target)

50 mg/kg (intervention)

Air Quality Standards: no safe guidelines recommended due to carcinogenic properties.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
sodium salicylate	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium salicylate	LOW (LogKOW = 2.2447)

Mobility in soil

Ingredient	Mobility
sodium salicylate	LOW (Log KOC = 23.96)

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"> ▶ Containers may still present a chemical hazard/ danger when empty. ▶ Return to supplier for reuse/ recycling if possible. Otherwise: <ul style="list-style-type: none"> ▶ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Treat and neutralise at an approved treatment plant. ▶ Treatment should involve: Mixing or slurring in water; Neutralisation with suitable dilute acid followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material). ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
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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
sodium salicylate	Not Applicable
sodium nitroprusside	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium salicylate	Not Applicable
sodium nitroprusside	Not Applicable

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****sodium salicylate is found on the following regulatory lists**

US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory

sodium nitroprusside is found on the following regulatory lists

US - California Hazardous Air Pollutants Identified as Toxic Air Contaminants

US - Pennsylvania - Hazardous Substance List

US CWA (Clean Water Act) - Priority Pollutants

US CWA (Clean Water Act) - Toxic Pollutants

US EPCRA Section 313 Chemical List

US New York City Community Right-to-Know: List of Hazardous Substances

US NIOSH Recommended Exposure Limits (RELs)

US OSHA Permissible Exposure Limits (PELs) Table Z-1

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	No
Acute toxicity (any route of exposure)	Yes
Reproductive toxicity	No
Skin Corrosion or Irritation	Yes
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	Yes
Specific target organ toxicity (single or repeated exposure)	No
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)

This product contains the following EPCRA section 313 chemicals subject to the reporting requirements of section 313 of the Emergency Planning and Community Right-To-Know-Act of 1986 (40 CFR 372):

CAS No	%[weight]	Name
13755-38-9	<2.5	sodium nitroprusside

This information must be included in all SDSs that are copied and distributed for this material.

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 - AIIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (sodium salicylate; sodium nitroprusside)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (sodium nitroprusside)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium salicylate; sodium nitroprusside)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (sodium salicylate)
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	03/15/2019
Initial Date	03/15/2019

Other information

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

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ 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

- ▶ AIIIC: 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

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