Clearview Clear-Prin Liquid

Clearview Enterprises

Part Number: **Not Available**
Version No: **1.4**
Safety Data Sheet according to OSHA HazCom Standard (2012) requirements

Issue Date: **31/08/2022**
Print Date: **31/08/2022**
L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	Clearview Clear-Prin Liquid
Synonyms	Not Available
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	Not Available

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Clearview Enterprises**
Address	451 Agnes Drive Tontitown AR 72770 United States
Telephone	866-361-4689
Fax	Not Available
Website	www.cvear.com
Email	Not Available

Emergency phone number

Association / Organisation	**Infortrac**
Emergency telephone numbers	**800-535-5053**
Other emergency telephone numbers	**+1-352-323-3500**

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	Serious Eye Damage/Eye Irritation Category 1, Skin Corrosion/Irritation Category 2, Reproductive Toxicity Category 2, Carcinogenicity Category 2

Label elements

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

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Hazard statement(s)

H318	Causes serious eye damage.
H315	Causes skin irritation.
H361	Suspected of damaging fertility or the unborn child.
H351	Suspected of causing cancer.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.

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.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
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.

Precautionary statement(s) Disposal

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

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
54-21-7	12	sodium salicylate

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	‣ Generally not applicable.
Skin Contact	If skin contact occurs: ‣ Immediately remove all contaminated clothing, including footwear. ‣ Flush skin and hair with running water (and soap if available). ‣ Seek medical attention in event of irritation. ‣ Generally not applicable.
Inhalation	‣ Generally not applicable.
Ingestion	‣ 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.

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- 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

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 http://www.ozemail.com.au/-ouad/SALI0001.HTA
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

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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.

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	‣ 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. Slight hazard when exposed to heat, flame and oxidisers.
Fire/Explosion Hazard	Combustible. Will burn if ignited. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes. 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	‣ 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	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves.

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	‣ 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. Minor hazard. ‣ 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. ‣ 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.

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

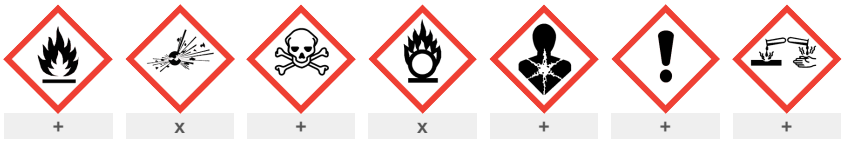
SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ 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	‣ 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.
Storage incompatibility	‣ Avoid reaction with oxidising agents



X — *Must not be stored together*

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0 *— May be stored together with specific preventions*
+ *— May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
Clearview Clear-Prin Liquid	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
sodium salicylate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium salicylate	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Airborne particulate or vapour must be kept to levels as low as is practicably achievable given access to modern engineering controls and monitoring hardware. Biologically active compounds may produce idiosyncratic effects which are entirely unpredictable on the basis of literature searches and prior clinical experience (both recent and past).

Exposure controls

Appropriate engineering controls

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.
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:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
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.
Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing 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, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, 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.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

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	Within each range the appropriate value depends on: Lower end of the range / Upper end of the range 1: Room air currents minimal or favourable to capture / 1: Disturbing room air currents 2: Contaminants of low toxicity or of nuisance value only. / 2: Contaminants of high toxicity 3: Intermittent, low production. / 3: High production, heavy use 4: Large hood or large air mass in motion / 4: Small hood-local control only Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	No special equipment required due to the physical form of the product. ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	Wear general protective gloves, eg. light weight rubber gloves.
Body protection	See Other protection below
Other protection	▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Coloured

Physical state	article	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	6.5	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Applicable	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	Not Available	**Taste**	Not Available

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Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Applicable
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (Not Available%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	Product is considered stable and hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. At sufficiently high doses the material may be hepatotoxic (i.e. poisonous to the liver). Signs may include nausea, stomach pains, low fever, loss of appetite, dark urine, clay-coloured stools, jaundice (yellowing of the skin or eyes) Large oral doses of salicylates may cause mild burning pain in the throat, stomach and usually prompt vomiting. Several hours may elapse before the development of deep and rapid breathing, lassitude, anorexia, nausea, vomiting, thirst and occasional diarrhoea. Common derivatives of salicylic acid produce substantially the same toxic syndrome, ("salicylism"). Major signs and symptoms arise from stimulation and terminal depression of the central nervous system. Stimulation produces vomiting, hyperpnea (abnormal increase in rate and depth of respiration), headache, tinnitus (ringing in the ears) confusion, bizarre behaviour or mania, generalised convulsions. Death is due to respiratory failure or cardiovascular collapse. Severe sensory disturbances such as deafness and dimness of vision are common. Less common features include sweating, skin eruptions, gastrointestinal and other hemorrhages, renal failure and pancreatitis. A tendency to bleed may be manifest by blood in the vomitus (haematemesis), bloody stools (melena) or purplish-red spots (petechiae) on the skin. Many of the toxic effects detailed here are due to or aggravated by severe disturbance of acid-base balance with the chief cause being prolonged hyperventilation from central stimulation. An assessment of acute salicylate intoxication based on dose suggests; 500 mg/kg: Potentially lethal Non-steroidal anti-inflammatory drugs (NSAID) can cause serious gastrointestinal (GI) adverse events including inflammation, bleeding, ulceration, and perforation of the stomach, small intestine, or large intestine, which can be fatal. These serious adverse events can occur at any time, with or without warning symptoms, in patients treated with NSAIDs. Only one in five patients, who develop a serious upper GI adverse event on NSAID therapy, is symptomatic. Upper GI ulcers, gross bleeding, or perforation caused by NSAIDs occur in approximately 1% of patients treated for 3-6 months, and in about 2-4% of patients treated for one year. These trends continue with longer duration of use, increasing the likelihood of developing a serious GI event at some time during the course of therapy. Anaphylactoid (allergic) reactions may occur. This typically occurs in asthmatic patients who experience rhinitis with or without nasal polyps, or who exhibit severe, potentially fatal bronchospasm after taking aspirin or other NSAIDs. NSAIDs, can cause serious skin adverse events such as exfoliative dermatitis, Stevens-Johnson Syndrome (SJS), and toxic epidermal necrolysis (TEN), which can be fatal Non-steroidal anti-inflammatory drug (NSAID) overdose may produce nausea, vomiting, indigestion and epigastric pain. Central nervous system effects may include drowsiness, dizziness, mental confusion, disorientation, lethargy, paraesthesia, numbness, intense headache, blurred vision, tinnitus, decreased auditory acuity, ataxia, muscle twitching, convulsions, stupor and coma. Other reported effects include sweating, oliguria or anuria, tachycardia and hypo- or hypertension. Renal damage may also occur.

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Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. NSAIDs may cause an increased risk of serious cardiovascular thrombotic events, myocardial infarction, and stroke, which can be fatal. NSAIDs cause an increased risk of serious gastrointestinal adverse events including bleeding, ulceration, and perforation of the stomach or intestines, which can be fatal. Both cyclooxygenase-1 and cyclooxygenase-2 (COX-1 and COX-2) inhibit the production of prostaglandins in the stomach and intestines responsible for maintaining the mucous lining of the gastrointestinal tract. These events can occur at any time during use and without warning symptoms. All NSAIDs increase plasma renin activity and aldosterone levels, and increase sodium and potassium retention. Vasopressin activity is also enhanced. Together these may lead to: · oedema (swelling due to fluid retention) · hyperkalaemia (high potassium levels) · hypernatraemia (high sodium levels) · hypertension Elevations of serum creatinine and more serious renal damage such as acute renal failure, chronic nephritis and nephrotic syndrome, are also possible. These conditions also often begin with edema and hyperkalemia. Many NSAIDs cause lithium retention by reducing its excretion by the kidneys; users have an elevated risk of lithium toxicity. Prolonged treatment with non-steroidal anti-inflammatory drugs (NSAIDs) has been associated with gastrointestinal irritation, erosion, ulceration, perforation, frank or occult bleeding, diarrhoea, constipation, and blood in the vomit or stool. Kidney damage may result in haematuria (blood in the urine), pyuria (white blood cells in the urine), proteinuria (protein in the urine), urinary casts (cylindrical aggregations of particles that form in the distal nephron, dislodge, and pass into the urine), nocturia (excessive night time urination) , polyuria (production of large volumes of pale urea), dysuria (painful or difficult urination), oliguria (production of abnormally small volumes of urea), or anuria (inability to urinate), renal insufficiency (insufficient excretion of wastes by the kidney), nephrosis and nephrotic syndrome (conditions characterized by oedema and large amounts of protein in the urine and usually increased blood cholesterol), and glomerular and interstitial nephritis. Liver effects, although rare, include jaundice, hepatocellular injury, possible fatal hepatitis, and abnormal liver function tests. Aspirin and other non-steroidal anti-inflammatory drugs, causes foetotoxicity, minor skeletal malformations, e.g., supernumerary ribs, and delayed ossification in rodent reproduction trials, but no major teratogenicity. Similarly, NSAIDs prolong gestation and interfere with parturition and with normal development of young before weaning. Therapeutic use of NSAIDs during the second half of pregnancy is associated with adverse effects in the foetus such as premature closure of the ductus arteriosus, which may lead to persistent pulmonary hypertension in the newborn. In rat studies with NSAIDs, as with other drugs known to inhibit prostaglandin synthesis, an increased incidence of dystocia, delayed parturition, and decreased pup survival occurred. Because of the known effects of NSAIDs on the foetal cardiovascular system (closure of ductus arteriosus), use during pregnancy (particularly late pregnancy) should be avoided.. Animal studies have shown that NSAIDs administered during late pregnancy can cause prolonged gestation, difficult labour, delayed birth, and decreased pup survival rates, In October 2020, the U.S. Food and Drug Administration (FDA) required the drug label to be updated for all nonsteroidal anti-inflammatory medications to describe the risk of kidney problems in fetuses that result in low amniotic fluid. They recommend avoiding NSAIDs in pregnant women at 20 weeks or later in pregnancy Clinical trials of several COX-2 selective and nonselective NSAIDS of up to three years duration have shown an increased risk of serious cardiovascular (CV) thrombotic events, myocardial infarction, and stroke, which can be fatal. All NSAIDs, both COX-2 selective and nonselective, may have a similar risk. NSAIDs, can lead to onset of new hypertension or worsening of preexisting hypertension, either of which may contribute to the increased incidence of CV events. Long-term administration of NSAIDs has resulted in renal papillary necrosis and other renal injury. Acute interstitial nephritis with haematuria, proteinuria, and occasionally nephritic syndrome have been reported.

Continued...

Anaphylactoid reactions may occur in patients with known prior exposure to other NSAIDs.
NSAIDs have produced ocular changes in animals and there have been reports of adverse eye findings in patients.
Anaemia is sometimes seen in patients receiving NSAIDs.. This may be due to fluid retention, occult or gross GI blood loss, or an incompletely described effect upon erythropoiesis.
NSAIDs inhibit enzymes collectively described as "COXs". In the course of the early search for a specific inhibitor of the negative effects of prostaglandins which spared the positive effects, it was discovered that prostaglandins could indeed be separated into two general classes which could loosely be regarded as "good prostaglandins" and "bad prostaglandins", according to the structure of a particular enzyme involved in their biosynthesis, cyclooxygenase (COX).
Prostaglandins whose synthesis involves the cyclooxygenase-I enzyme, or COX-1, are responsible for maintenance and protection of the gastrointestinal tract, while prostaglandins whose synthesis involves the cyclooxygenase-II enzyme, or COX-2, are responsible for inflammation and pain.
The existing non-steroidal anti-inflammatory drugs (NSAIDs) differ in their relative specificities for COX-2 and COX-1
There has been much concern about the possibility of increased risk for heart attack and stroke in users of NSAID drugs, particularly COX-2 selective NSAIDs. The cardiovascular risks associated with NSAIDs are controversial, with apparently contradictory data produced from different clinical trials and in published meta-analyses. Cardiovascular risk of COX-2 specific inhibitors is not surprising since prostaglandins are involved in regulation of blood pressure by the kidneys. COX-inhibitors produce blood dyscrasias (abnormal conditions of the blood), and interfere with platelet function.
Phototoxic or photoallergic skin reactions may also occur. Anaphylactoid reactions characterised by maculopapular rash, urticaria, pruritus, bronchospasm, and syncope have been described. Other effects include oedema, metabolic acidosis, hyperkalaemia, azotemia, cystitis and urinary tract infections, visual and hearing disturbances, conjunctivitis, corneal deposits, retinal degeneration, ear pain and occasionally, deafness. Idiosyncratic responses include asthma, allergic interstitial nephritis, hypersensitivity hepatitis, aplastic anaemia and exfoliative dermatitis.
Non-steroidal anti-inflammatory drugs with an inhibitory effect on prostaglandin synthesis, when given during the latter stages of pregnancy, cause premature closure of the foetal ductus arteriosus (1). When given at term they prolong labour and delay parturition. Evidence (1) from animal experimental studies, clinical investigations in humans, and epidemiological studies supports the hypothesis that NSAIDs are chemopreventative agents against colon cancer. This is corroborated by knowledge of the underlying pathophysiological mechanisms and the effects of arachidonic metabolites, i.e prostaglandins, on the carcinogenic process and the influence of cyclooxygenase (COX) inhibitors such as NSAIDs on these metabolites. 1. Berkel et al; Epidemiol Rev., Vol 18, No. 2, 1996
Because of the known effects of NSAIDs drugs on the foetal cardiovascular system (closure of ductus arteriosus), use during pregnancy (particularly late pregnancy) should be avoided. In rat studies with NSAIDs, as with other drugs known to inhibit prostaglandin synthesis, an increased incidence of dystocia, delayed parturition, and decreased pup survival occurred
Aspirin and NSAIDs may cause anaphylactic or anaphylactoid reactions. Constitutively-expressed cyclooxygenase (COX-1) inhibition is likely to be responsible for the cross-reactions and side effects associated with these drugs, as well as the anaphylactoid reactions sometimes seen in aspirin-sensitive respiratory disease. Though anaphylactic and anaphylactoid reactions may be clinically indistinguishable, they involve different mechanisms. Anaphylactic reactions are due to immediate hypersensitivity involving cross-linking of drug-specific IgE. Regardless of COX selectivity pattern, NSAIDs may function as haptens capable of inducing allergic sensitization. Unlike anaphylaxis, anaphylactoid reactions are most likely related to inhibition of COX-1 by NSAIDS. Thus, an anaphylactoid reaction caused by a particular COX-1 inhibiting NSAID will occur with a chemically unrelated NSAID which also inhibits COX-1 enzymes. Selective COX-2 inhibitors appear to be safe in patients with a history of NSAID-related anaphylactoid reactions but can function as haptens, with resulting sensitisation and anaphylaxis upon next exposure. Eva A Berkes Clinical Reviews in Allergy and Immunology 24, pp 137-147 2003.
COX-2 inhibitors reduce inflammation (and pain) while minimising gastrointestinal adverse drug reactions (e.g. stomach ulcers) that are common with non-selective NSAIDs. COX-1 is involved in synthesis of prostaglandins and thromboxane, but COX-2 is only involved in the synthesis of prostaglandin. Therefore, inhibition of COX-2 inhibits only prostaglandin synthesis without affecting thromboxane and thus has no effect on platelet aggregation or blood clotting.
Chronic abuse of analgesics has been associated with nephropathy. Patients invariably have a history of regular ingestion of substantial or excessive doses over a period of years. In mild cases the condition is reversible. The initial renal lesion is papillary necrosis proceeding to secondary atrophic changes in the renal cortex body. An abnormally high incidence of transitional cell carcinoma of the renal pelvis and bladders has been reported in patients with analgesic nephropathy.
Mild chronic salicylate intoxication, or "salicylism", may occur after repeated exposures to large doses. Symptoms include dizziness, tinnitus, deafness, sweating, nausea and vomiting, headache and mental confusion. Symptoms of more severe intoxication include hyperventilation, fever, restlessness, ketosis, and respiratory alkalosis and metabolic acidosis. Depression of the central nervous system may lead to coma, cardiovascular collapse and respiratory failure.
Chronic exposure to the salicylates (o-hydroxybenzoates) may produce metabolic and central system disturbances or damage to the kidneys. Persons with pre-existing skin disorders, eye problems or impaired kidney function may be more susceptible to the effects of these substances. Certain individuals (atopics), notably asthmatics, exhibit significant hyper- sensitivity to salicylic acid derivatives. Reactions include urticaria and other skin eruptions, rhinitis and severe (even fatal) bronchospasm and dyspnea.
Chronic exposure to the p-hydroxybenzoates (parabens) is associated with hypersensitivity reactions following application of these to the skin. Hypersensitivity reactions have also been reported following parenteral or oral administration. Cross-sensitivity occurs between the p-hydroxybenzoates Hypersensitivity reactions may include by acute bronchospasm, hives (urticaria), deep dermal wheals (angioneurotic oedema), running nose (rhinitis) and blurred vision. Anaphylactic shock and skin rash (non-thrombocytopenic purpura) may also occur. Any individual may be predisposed to such anti-body mediated reaction if other chemical agents have caused prior sensitisation (cross-sensitivity).

Clearview Clear-Prin Liquid

TOXICITY	IRRITATION
Not Available	Not Available

sodium salicylate

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]

Continued...

	Oral (Rat) LD50; 1200 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
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	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. NOAELwas 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. 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.
Clearview Clear-Prin Liquid & SODIUM SALICYLATE	Inhibition of NF-kB in vivo can be detrimental. NF-kB 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-kB activity. The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis,and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences. NF-kB 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 a p38 and JNK. In addition to mediating proinflammatory responses, NF-kB 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-kB can have adverse consequences such as immune suppression and tissue damage. Understanding the consequences of lack of NF-kB 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-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IkBalpha.The IKK gamma mutations result in a defective IKK complex and the IkBalpha mutation results in an IkBalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB 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-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF-kB 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-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB 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-kB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-kB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-kB through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF-kB 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-kB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-kB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-kB 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-kB activation in lymphocytes results in defects in

Continued...

growth, survival, and cytokine production and blocks multiple steps in germinal center formation.Given the diverse roles NF-kB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-kB pathway are susceptible to parasitic and bacterial infection.
The role of NF-kB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-kB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-kB 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-kB 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-lipooxygenase 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.
The Research Institute for Fragrance Materials (RIFM) Expert Panel study of fragrance salicylates concluded.

The salicylates are well absorbed by the oral route, and oral bioavailability is assumed to be 100%. Absorption by the dermal route in humans is more limited with bioavailability in the range of 11.8-30.7%.
The salicylates are expected to undergo extensive hydrolysis, primarily in the liver, to salicylic acid which is conjugated with either glycine or glucuronide and is excreted in the urine as salicyluric acid and acyl and phenolic glucuronides. The hydrolyzed side chains are metabolized by common and well-characterized metabolic pathways leading to the formation of innocuous end products. The expected metabolism of the salicylates does not present toxicological concerns.
The acute dermal toxicity of the salicylates is very low, with LD50 values in rabbits reported to be greater than 5000 mg/kg body weight. The acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group and with LD50‘s between 1000 and >5000 g/kg. In dermal subchronic toxicity studies, extreme doses of methyl salicylate (5 g/kg body weight/day) possibly were nephrotoxic but the data were minimal. The subchronic oral NOAEL is concluded to be 50 mg/kg body weight/day.
Genetic toxicity data, for methyl salicylate, a few other salicylates and for structurally related alkyl- and alkoxy-benzyl derivatives are negative for genotoxicity.
Given the metabolism of salicylate and the evidence that they are non-genotoxic, it can be concluded that the salicylates are without carcinogenic potential.
The reproductive and developmental toxicity data on methyl salicylate demonstrate that high, maternally toxic doses result in a pattern of embryotoxicity and teratogenesis similar to that characterized for salicylic acid.
At concentrations likely to be encountered by humans through the use of the salicylates as fragrance ingredients, these chemicals are considered to be non-irritating to the skin.
The salicylates (with the exception of benzyl salicylate) in general have no or very limited skin sensitization potential.
The salicylates are non-phototoxic and have no photoirritant or photoallergenic activity
The use of the salicylates in fragrances produces low levels of exposure relative to doses that elicit adverse systemic effects in laboratory animals exposed by the dermal or oral route. Based on NOAEL values of 50 mg/kg body weight/day identified in the subchronic and the chronic toxicity studies , a margin of safety for systemic exposure of humans to the individual salicylates in cosmetic products, may be calculated to range from 125 to 2,500,000 (depending upon the assumption of either 12–30% or 100% bioavailability following dermal application) times the maximum daily exposure.

The acute dermal toxicity of the salicylates is very low. Rabbit dermal LD50 values have been reported to be >5000 mg/kg body weight for 15 of the 16 salicylates tested, findings likely related to the limited degree of dermal absorption, the retention of salicylate in the skin, and the relatively moderate toxicity of salicylic acid itself upon systemic exposure (i.e., oral LD50 value of 891 mg/kg body weight in rats) .
Overall, the acute oral toxicity of the salicylates is moderate, with toxicity generally decreasing with increasing size of the ester R-group. For the longer carbon chain salicylates, acute oral LD50 s range from 1320 to >5000 mg/kg body weight. The acute oral toxicity of the unsaturated salicylates is likewise low to moderate with rat oral LD50 s in the 3200 to >5000 mg/kg body weight range as are the acute oral toxicities of the aromatic salicylates (1300 to >5000 mg/kg body weight)

Continued...

	The 17 compounds assessed in this report include the core salicylate moiety that upon hydrolysis yield salicylic acid and the alcohol of the corresponding alkyl, alkenyl, benzyl, phenyl, phenethyl, etc. side chain. This is consistent with information on other alkyl- and alkoxy- benzyl derivatives whereby aromatic esters are hydrolyzed in vivo by carboxylesterases, or esterases, especially the A-esterases. Potential differences in the metabolism of the individual salicylates would be related to the manner in which the hydrolyzed side chain undergoes further oxidation/reduction and/or conjugation reactions. Salicylic acid undergoes metabolism primarily in the liver. At low, non-toxic doses, approximately 80% of salicylic acid is further metabolized in the liver via conjugation with glycine and subsequent formation of salicyluric acid. For each of the salicylates, following hydrolysis to salicylic acid, the resulting side chains, hydroxylated alkyl, alkenyl, and phenyl moieties, could be expected to be further metabolized. In the case of the alcohols formed following hydrolysis. Further metabolism would result in the formation of the corresponding aldehydes and acids, with eventual degradation to CO_2 by the fatty acid pathway and the tricarboxylic acid cycle. The secondary alcohols formed by hydrolysis of isobutyl and isoamyl salicylate, would primarily be conjugated with glucuronic acid and excreted. They could also interconvert to the corresponding ketones. Salicylates bearing alkenyl side chains, may undergo epoxidation and subsequent hydroxylation at points of unsaturation. However, since both the alkyl and alkenyl side chains would be hydroxylated at one terminus following hydrolysis of the corresponding salicylate, a significant proportion of these hydrolysis products would be excreted in the urine precluding further metabolism and epoxidation. In the case of hydrolysis of the salicylates containing aromatic side chains, phenyl salicylate and benzyl salicylate, phenol and benzyl alcohol, respectively, would be formed. Salicylates were potent and selective inhibitors for AKR1C1 enzymes , a family of aldo-keto reductases implicated in biosynthesis, intermediary metabolism and detoxification.

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

Clearview Clear-Prin Liquid	**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**
	NOEC(ECx)	96h	Fish	1mg/l	4
	EC50	72h	Algae or other aquatic plants	75.25mg/l	2
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2

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*

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

Continued...

Ingredient	Mobility
sodium salicylate	LOW (KOC = 23.96)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

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

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

Not Applicable

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

Product name	Group
sodium salicylate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
sodium salicylate	Not Available

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	US TSCA Chemical Substance Inventory - Interim List of Active Substances

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

Continued...

In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	Yes
Acute toxicity (any route of exposure)	No
Reproductive toxicity	Yes
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

State Regulations

US. California Proposition 65

None Reported

National Inventory Status

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

SECTION 16 Other information

Revision Date	31/08/2022
Initial Date	31/08/2022

Other information

Ingredients with multiple cas numbers

Name	CAS No
sodium salicylate	54-21-7, 18495-69-7, 94413-51-1

Classification of the preparation and its individual components has drawn on official and authoritative sources using available literature references.

Continued...

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
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