

SAFETY DATA SHEET

Porcelain Etch

1. MATERIAL AND SUPPLY COMPANY IDENTIFICATION

Product Name:	Porcelain Etch
Product Codes:	DL1511
Recommended Use:	Rapidly, chemically etches porcelain and ceramic restorations to facilitate bonding procedures and improve bond adhesion.
Contact Information:	Dentalife Australia Pty. Ltd. Factory 9/505 Maroondah Highway Ringwood, VIC, 3134, Australia Phone: +61 3 9879 1226
Emergency Telephone Number:	+61 3 9879 1226
Poisons Information Centre:	24 hour, 7 days a week in an emergency call: 13 11 26

2. HAZARD IDENTIFICATION

This material is hazardous according to health criteria of Safe Work Australia.

Signal Word: DANGER



Hazard Classifications: Acute Toxicity (Oral) Category 2, Acute Toxicity (Dermal) Category 1, Specific target organ toxicity - repeated exposure Category 2, Serious Eye Damage Category 1, Acute Toxicity (Inhalation) Category 1, Skin Sensitizer Category 1, Germ cell mutagenicity Category 2, Skin Corrosion/Irritation Category 1A

Hazard Statement: H300 Fatal if swallowed
H310 Fatal in contact with skin
H373 May cause damage to organs through prolonged or repeated exposure
H330 Fatal if inhaled
H317 May cause an allergic skin reaction

SAFETY DATA SHEET

H341 Suspected of causing genetic defects
H314 Causes severe skin burns and eye damage.

Prevention Precautionary Statements:

P201 Obtain special instructions before use.
P260 Do not breath mist/vapours/spray
P262 Do not get in eyes, on kin, or on clothing.
P270 Do not eat, drink or smoke when using this product.
P271 Use only outdoors or in a well-ventilated area
P280 Wear protective clothing, gloves, eye/face protection and suitable respirator.
P284 Wear respirator protection.
P272 Contaminated work clothing should not be allowed out of workplace

Response Precautionary Statements:

P301+P310 IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P301+P330+P331 IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353 IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P304+P340 IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.
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.
P320 Specific treatment is urgent (see advice on this label).
P322 Specific measures (see advice on this label)
P363 Wash contaminated clothing before use
P302+P350 IF ON SKIN: Gently wash with plenty of soap and water.
P302+P352 IF ON SKIN: Wash with plenty of water
P333+P313 if skin irritation or rash occurs: Get medical advice/attention.

Storage Precautionary Statements:

P403+P233 Store in a well-ventilated place. Keep container tightly closed.
P403+P235 Store in a well-ventilated place. Keep cool.

Disposal Precautionary Statements:

P501 Dispose of contents/container in accordance with local, regional, national, and international regulations.

Poison Schedule:

Not Applicable

3. COMPOSITION INFORMATION

CHEMICAL ENTITY	CAS NO.	PROPORTION %
Hydrofluoric Acid	7664-39-3	9-10%
Balance ingredients (non-hazardous)	-	> 5%
Water	7732-18-5	to 100 %

SAFETY DATA SHEET

4. FIRST AID MEASURES

If poisoning occurs, contact a doctor or Poisons Information Centre (Phone Australia 131 126, New Zealand 0800 764 766).

Inhalation:

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.

Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema.

Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs).

As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (yet) manifested.

Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered.

This must definitely be left to a doctor or person authorised by him/her.
(ICSC13719)

For massive exposures:

If dusts, vapours, aerosols, 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.

If victim is conscious, give six calcium gluconate or calcium carbonate tablets in water by mouth.

Transport to hospital, or doctor, urgently.

Skin Contact:

If there is evidence of severe skin irritation or skin burns:

Avoid further contact. Immediately remove contaminated clothing, including footwear.

Flush skin under running water for 15 minutes.

Avoiding contamination of the hands, massage calcium gluconate gel into affected areas, pay particular attention to creases in skin.

Contact the Poisons Information Centre.

SAFETY DATA SHEET

Continue gel application for at least 15 minutes after burning sensation ceases.

If pain recurs, repeat application of calcium gluconate gel or apply every 20 minutes.

If no gel is available, continue washing for at least 15 minutes, using soap if available. If patient is conscious, give six calcium gluconate or calcium carbonate tablets in water by mouth.

Transport to hospital, or doctor, urgently.

Eye contact:

If this product comes in contact with the eyes:

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.

Ingestion:

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.

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.

Indication of any immediate medical attention and special treatment needed

Following acute or short term repeated exposure to hydrofluoric acid:

Subcutaneous injections of Calcium Gluconate may be necessary around the burnt area. Continued application of Calcium Gluconate Gel or subcutaneous Calcium Gluconate should then continue for 3-4 days at a frequency of 4-6 times per day. If a "burning" sensation recurs, apply more frequently.

Systemic effects of extensive hydrofluoric acid burns include renal damage, hypocalcaemia and consequent cardiac arrhythmias. Monitor haematological, respiratory, renal, cardiac and electrolyte status at least daily. Tests should include FBE, blood gases, chest X-ray, creatinine and electrolytes, urine output, Ca ions, Mg ions and phosphate ions. Continuous ECG monitoring may be required.

Where serum calcium is low, or clinical, or ECG signs of hypocalcaemia develop, infusions of calcium gluconate, or if less serious, oral Sandocal, should be given. Hydrocortisone 500 mg in a four to six hourly infusion may help.

Antibiotics should not be given as a routine, but only when indicated.

Eye contact pain may be excruciating and 2-3 drops of 0.05% pentocaine hydrochloride may be instilled, followed by further irrigation

SAFETY DATA SHEET

For acute or short-term repeated exposures to fluorides:

Fluoride absorption from gastro-intestinal tract may be retarded by calcium salts, milk or antacids.

Fluoride particulates or fume may be absorbed through the respiratory tract with 20-30% deposited at alveolar level. Peak serum levels are reached 30 mins. post-exposure; 50% appears in the urine within 24 hours.

For acute poisoning (endotracheal intubation if inadequate tidal volume), monitor breathing and evaluate/monitor blood pressure and pulse frequently since shock may supervene with little warning. Monitor ECG immediately; watch for arrhythmias and evidence of Q-T prolongation or T-wave changes. Maintain monitor. Treat shock vigorously with isotonic saline (in 5% glucose) to restore blood volume and enhance renal excretion.

Where evidence of hypocalcaemic or normocalcaemic tetany exists, calcium gluconate (10 ml of a 10% solution) is injected to avoid tachycardia.

5. FIRE FIGHTING MEASURES

Hazchem Code:	2X
Suitable Extinguishing Media:	There is no restriction on the type of extinguisher which may be used. Use extinguishing media suitable for surrounding area.
Specific Hazards:	None known.
Firefighting Further Advice	Non combustible. Not considered as significant fire risk, however containers may burn. May emit corrosive fumes.

6. ACCIDENTAL RELEASE MEASURES

Personal Precautions, Protection Equipment and Emergency Procedures: See section 8

Environmental Precautions: See section 12

Methods and Material for Containment and Cleaning Up:

Minor Spills: Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. Check regularly for spills and leaks. Clean up all spills immediately. Avoid breathing vapours/ aerosols or dusts and avoid contact with skin and eyes. Place in a suitable, labelled container for waste disposal.

Major Spills: Clear area of all unprotected personnel. Slippery when spilt. Avoid accidents, clean up immediately. Wear protective equipment to prevent skin and eye contamination and the inhalation of vapours. Work up wind or increase ventilation. Contain - prevent run off into drains and waterways. Use absorbent (soil, sand, or other inert material). Collect and seal in properly labelled containers or drums for disposal. If contamination of crops, sewers or waterways has occurred advise local emergency services.

SAFETY DATA SHEET

7. HANDLING AND STORAGE

Safe Handling:

Avoid all personal contact, including inhalation.

Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area.

WARNING: To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material. Avoid smoking, naked lights or ignition sources.

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 in original containers. Keep containers securely sealed.

Store in a cool, dry, well-ventilated area.

Store away from incompatible materials and foodstuff containers.

Protect containers against physical damage and check regularly for leaks.

Observe manufacturer's storage and handling recommendations contained within this SDS.

Storage incompatibility:

Inorganic acids are generally soluble in water with the release of hydrogen ions. The resulting solutions have pH's of less than 7.0.

Inorganic acids neutralise chemical bases (for example: amines and inorganic hydroxides) to form salts - neutralisation can generate dangerously large amounts of heat in small spaces.

The dissolution of inorganic acids in water or the dilution of their concentrated solutions with additional water may generate significant heat.

The addition of water to inorganic acids often generates sufficient heat in the small region of mixing to cause some of the water to boil explosively. The resulting "bumping" can spatter the acid.

Inorganic acids react with active metals, including such structural metals as aluminum and iron, to release hydrogen, a flammable gas.

Inorganic acids can initiate the polymerisation of certain classes of organic compounds. Inorganic acids react with cyanide compounds to release gaseous hydrogen cyanide.

Inorganic acids generate flammable and/or toxic gases in contact with dithiocarbamates, isocyanates, mercaptans, nitrides, nitriles, sulfides, and strong reducing agents. Additional gas-generating reactions occur

SAFETY DATA SHEET

with sulfites, nitrites, thiosulfates (to give H₂S and SO₃), dithionites (SO₂), and even carbonates.

Acids often catalyse (increase the rate of) chemical reactions.

Salts of inorganic fluoride:

- react with water forming acidic solutions.
- are violent reactive with boron, bromine pentafluoride, bromine trifluoride, calcium disilicide, calcium hydride, oxygen difluoride, platinum, potassium.
- in aqueous solutions are incompatible with sulfuric acid, alkalis, ammonia, aliphatic amines, alkanolamines, alkylene oxides, amides, epichlorohydrin, isocyanates, nitromethane, organic anhydrides, vinylacetate.
- corrode metals in presence of moisture
- may be incompatible with glass and porcelain
- Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air.

Hydrogen fluoride:

- reacts violently with strong oxidisers, acetic anhydride, alkalis, 2-aminoethanol, arsenic trioxide (with generation of heat), bismuthic acid, calcium oxide, chlorosulfonic acid, cyanogen fluoride, ethylenediamine, ethyleneimine, fluorine (fluorine gas reacts vigorously with a 50% hydrofluoric acid solution and may burst into flame), nitrogen trifluoride, N-phenylazopiperidine, oleum, oxygen difluoride, phosphorus pentoxide, potassium permanganate, potassium tetrafluorosilicate(2-),
- beta-propiolactone, propylene oxide, sodium, sodium tetrafluorosilicate, sulfuric acid, vinyl acetate
- reacts (possibly violently) with aliphatic amines, alcohols, alkanolamines, alkylene oxides, aromatic amines, amides, ammonia, ammonium hydroxide, epichlorohydrin, isocyanates, metal acetylides, metal silicides, methanesulfonic acid, nitrogen compounds, organic anhydrides, oxides, silicon compounds, vinylidene fluoride
- attacks glass and siliceous materials, concrete, ceramics, metals (flammable hydrogen gas may be produced), metal alloys, some plastics, rubber coatings, leather, and most other materials with the exception of lead, platinum, polyethylene, wax.

8. EXPOSURE CONTROLS / PERSONAL PROTECTION

Occupational Exposure Limits (OEL):

Ingredient Data:

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	Hydrofluoric acid	Hydrogen fluoride	Not available	Not available	3 ppm / 2.6 mg/m ³	Not available



SAFETY DATA SHEET

Emergency Limits:

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
Hydrofluoric acid	Hydrogen fluoride (Hydrofluoric acid)	Not available	Not available	Not available

Ingredient	Original IDLH	Revised IDLH
Hydrofluoric acid	30 ppm	Not available

Material Data:

Odour Threshold for hydrogen fluoride: 0.042 ppm

NOTE: Detector tubes for hydrogen fluoride, measuring in excess of 1.5 ppm, are available commercially. Long-term measurements (8 hrs) may be conducted to detect concentrations exceeding 0.25 ppm.

Hydrogen fluoride is a primary irritant which as a gas causes severe respiratory irritation and as a liquid which causes severe and painful burns to the skin and eyes. The recommendation for TLV-TWA is based on the results of controlled inhalation studies in human volunteers. The limit is thought to minimise the potential for occurrence of dental and/or osteofluorosis (systemic fluorosis) and to prevent the risk of primary irritation to the eyes, nose, throat and lower respiration system.

At concentrations exceeding 3 ppm there have been reports of skin reddening and burning of the nose and eyes.

Engineering Controls:

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.

SAFETY DATA SHEET

Personal Protection Equipment:

SAFETY SHOES, OVERALLS, GLOVES, CHEMICAL GOGGLES, RESPIRATOR.

Personal protective equipment (PPE) must be suitable for the nature of the work and any hazard associated with the work as identified by the risk assessment conducted.

Wear safety shoes, overalls, gloves, chemical goggles, respirator. Use with adequate ventilation. If inhalation risk exists wear organic vapour/particulate respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716. Available information suggests that gloves made from nitrile rubber should be suitable for intermittent contact. However, due to variations in glove construction and local conditions, the user should make a final assessment. Always wash hands before smoking, eating, drinking, or using the toilet. Wash contaminated clothing and other protective equipment before storing or re-using.

Eye and face protection:

Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk quantities, where there is a danger of splashing, or if the material may be under pressure.

Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted.

Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.

Alternatively, a gas mask may replace splash goggles and face shields.

Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience.

Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers

have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]



SAFETY DATA SHEET

Other Protection:	Overalls. PVC Apron. PVC protective suit may be required if exposure severe. Eyewash unit. Ensure there is ready access to a safety shower.
Hygiene Measures:	Keep away from food, drink and animal feedstuffs. When using do not eat, drink or smoke. Wash hands prior to eating, drinking or smoking. Avoid contact with clothing. Avoid eye contact and repeated or prolonged skin contact. Avoid inhalation of vapour, mist or aerosols. Ensure that eyewash stations and safety showers are close to the workstation location.

9. PHYSICAL AND CHEMICAL PROPERTIES

Form:	Gel
Colour:	Yellow
Odour:	Characteristic
Solubility:	Miscible with water
Specific gravity:	1.1
Vapour Pressure (20 °C):	Not available
Flash Point (°C):	Not available
Flammability Limits (%):	Not available
Autoignition Temperature (°C):	Not available
Decomposition Temperature(°C):	Not available
Melting Point/Range (°C):	Not available
Boiling Point/Range (°C):	Not available
Evaporation Rate:	Not available
pH:	< 2
Viscosity:	Not available
Total VOC (g/Litre):	Not available

10. STABILITY AND REACTIVITY

Reactivity:	See section 7
Chemical Stability:	Unstable in the presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.
Conditions to Avoid:	See section 7
Incompatible Materials:	See section 7
Hazardous Decomposition Products:	See section 5
Hazardous Reactions:	See section 7

11. TOXICOLOGICAL INFORMATION

Information on toxicological effects:

Inhaled:	Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may
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SAFETY DATA SHEET

include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema.

Acute effects of fluoride inhalation include irritation of nose and throat, coughing, chest discomfort, chills, fever and cyanosis (blue lips and skin). Acute inhalation exposures to hydrogen fluoride (hydrofluoric acid) vapours produce severe eye, nose, and throat irritation; delayed fever, cyanosis, and pulmonary edema; and may cause death.

From accidental, occupational, and volunteer exposures, it is estimated that the lowest lethal concentration for a 5-minute human exposure to hydrogen fluoride is in the range of 50 to 250 ppm. Significant exposures by dermal or inhalation route may cause hypocalcaemia and hypomagnesaemia; cardiac arrhythmias may follow. Acute renal failure has also been documented after an ultimately fatal inhalation exposure. Fluorides are not bound to any extent to plasma proteins. In human serum the fluoride occurs equally as nonionic and ionic forms.

Repeated sublethal exposures to hydrogen fluoride produce liver and kidney damage.

Rats, rabbits, guinea pigs, and dogs subject to hydrogen fluoride inhalation experienced significant irritation of the conjunctivae, nasal tissues, and respiratory system after acute inhalation exposures at near-lethal levels. Pathological lesions were observed in the kidney and liver, and the severity of the lesions was dose related. The external nares and nasal vestibules were black, and, at dosages causing considerable mortality, those areas showed zones of mucosal and submucosal necrosis.

Skin Contact:

Skin contact with the material may produce severely toxic effects; systemic effects may result following absorption and these may be fatal. The skin is readily penetrated by the fluoride ion causing liquefaction necrosis of the soft tissues and decalcification and corrosion of bone. Healing is delayed and necrotic changes may continue to occur and spread beneath a layer of tough coagulated skin.

Percutaneous absorption of pure liquefied hydrogen fluoride gas produced severe hypocalcaemia, multiple attacks of ventricular fibrillation, and death 9.5 hours after exposure. Skin contact with hydrogen fluoride or solutions containing more than 30 percent hydrogen fluoride produces immediate pain; reactions to more dilute solutions may be delayed for many hours. The accompanying pain is excruciating and persistent due to delayed healing.

Contact of the skin with liquid hydrofluoric acid (hydrogen fluoride) may cause severe burns, erythema, and swelling, vesiculation, and serious crusting. With more serious burns, ulceration, blue-gray discoloration, and necrosis may occur. Solutions of hydrofluoric acid, as dilute as 2%, may cause severe skin burns.

Dermal burns may not be readily noticed or painful, unlike the warning

SAFETY DATA SHEET

properties of other acids. Skin contact with HF concentrations in the 20% to 50% range may not produce symptoms for one to eight hours. With concentrations less than 20%, the latency period may be up to twenty-four hours. A solution of only 1-2% HF exposed to greater than 10% of the body is fatal without medical attention; however dermal burns are not likely immediate.

Fluoride ions form insoluble salts with calcium and magnesium in bodily tissue. Soluble salts can form with other cations, which dissociate rapidly causing further disruption and damage to tissue. The severe, throbbing pain associated with HF burns is thought to result from nerve irritation due to potassium cations entering the extracellular space to compensate for reduced calcium ion concentrations. Fluoride poisoning is associated with hypocalcemia (low calcium levels), hyperkalemia (high potassium levels), hypomagnesemia (low magnesium levels), and sudden death.

Systemic hypocalcemia should be considered a risk whenever the body surface area of skin burns from concentrated hydrofluoric acid exceed 160 cm² (25 sq.in.), or about the size of the palm of your hand. Concentrated hydrogen fluoride burns can be fatal if only 2% of the body surface area is exposed.

Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue.

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.

Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact.

Ingestion:

Severely toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 5 gram may be fatal or may produce serious damage to the health of the individual.

Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure.

Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and

SAFETY DATA SHEET

pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal.

Fluoride is a general protoplasmic poison which appears to produce at least four major functional derangements;

(1) enzyme inhibition, (2) hypocalcaemia, (3) cardiovascular collapse and (4) specific organ damage.

Hypocalcaemia which leads to severe reductions in plasma levels of both total calcium and ionic calcium, may appear several hours after exposure producing painful and involuntary muscular contractions (tetany) initially of the extremities (carpopedal spasm, twitching of limb muscles, laryngo-spasm, cardiospasm etc). Cardiovascular collapse is probably the principal cause of death in acute fluoride poisoning with sinus tachycardia the commonest cardiac finding and serious cardiac arrhythmias also common. Poisonings also cause major adverse effects on the brain and kidneys.

Toxic effects may include headache, excessive salivation, rapid movements of the eyeball (nystagmus) and dilated pupils. Convulsions may occur but lethargy, stupor and coma are more common. Renal pathology (acute congestion) has been described in human casualties.

Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis,

Eye Contact:

When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.

Irritation of the eyes may produce a heavy secretion of tears (lachrymation).

Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness.

Experiments in which a 20-percent aqueous solution of hydrofluoric acid (hydrogen fluoride) was instilled into the eyes of rabbits caused immediate damage in the form of total corneal opacification and conjunctival ischemia; within an hour, corneal stroma edema occurred, followed by necrosis of anterior ocular structures

Chronic:

Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.

Toxic: danger of serious damage to health by prolonged exposure

SAFETY DATA SHEET

through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Repeated or prolonged exposure to acids 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.

Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis.

The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects.

Long term exposure to vapour or dust with inorganic fluorides may result in fluorosis, with rheumatic symptoms, stiff joints, mottling of tooth enamel. Other signs may include nausea, vomiting, anorexia, diarrhoea or constipation, weight loss, anaemia, weakness and general ill-health. Polyuria and polydipsia may also occur. Exfoliative dermatitis, atopic dermatitis, stomatitis, gastrointestinal and respiratory allergy, and on occasions, central nervous system involvement have all been described.

Repeated human exposures to hydrogen fluoride (6 hours/day for 10-50 days) at concentrations as high as 4.7 ppm were tolerated without severe adverse reaction. At concentrations exceeding 3 ppm, researchers noted burning and irritation of the eyes and nose and burning of the skin. Three subjects who inhaled approximately 3 ppm had severe urinary excretions of 6.7-9.4 mg fluoride/day. One epidemiological study was able to demonstrate that there was no significant change in pulmonary function resulting from occupational exposure to average concentrations of 1.02 ppm hydrogen fluoride. A further study indicated a threshold for minimal increases (Grade 1) in bone density (fluorosis) at less than 3.38 mg/m³ (4.3 ppm) hydrogen fluoride. Grade 1 fluorosis resulted in no medically recognised disability.

SAFETY DATA SHEET

Hydrogen fluoride readily penetrates the skin and causes decalcification and corrosion of the bone and underlying tissue (hypocalcaemia). Deaths due to hypocalcaemia have been cited in the literature. Ingestion will cause severe pain and burns in the mouth and throat. Profound and possibly fatal hypocalcaemia is likely to occur unless medical treatment is promptly initiated. Symptoms include spasm and twitching of the muscles, high fever, convulsion and general extreme pain. Inhalation may cause corrosion of the throat, nose and lungs, leading to severe inflammation, pulmonary oedema or possible hypocalcaemia. [NIOSH TIC]

Carcinogenicity:	Data not available.
Reproductivity:	Data not available.
Aspiration Hazard:	Data not available.

12. ECOLOGICAL INFORMATION

DO NOT discharge into sewer or waterways.

Harmful to aquatic organisms.

Although small amounts of fluorides are conceded to have beneficial effects, two forms of chronic toxic effect, dental fluorosis and skeletal fluorosis may be caused by excessive intake over long periods. Fluorides are absorbed by humans following inhalation of workplace and ambient air that has been contaminated, ingestion of drinking water and foods and dermal contact.

Fluoride accumulates, food-dependently in skeletal tissues of both aquatic and terrestrial vertebrates and invertebrates. Bioaccumulation occurs in marine organisms and, to a lesser extent, fresh water organisms. Reported BCF-values for marine organisms range up to approximately 150 and 60 for fish and crustacea, respectively. The most important exposure route for plants is uptake from the atmosphere. Concentrations in plants in the vicinity of a HF production plant range up to approximately 200 mg/kg, with mean levels between 20 and 50 mg/kg dry weight. Generally, lowest fluoride levels are found in herbivores and (somewhat) higher levels in predators.

Fluorides have been shown to accumulate in animals that consume fluoride-containing foliage. However, accumulation is primarily in skeletal tissue and therefore, it is unlikely that fluoride will biomagnify up the food chain.

In water, the transport and transformation of inorganic fluorides are influenced by pH, water hardness and the presence of ion-exchange materials such as clays. In natural water, fluoride forms strong complexes with aluminum in water, and fluorine chemistry in water is largely regulated by aluminum concentration and pH. Below pH 5, fluoride is almost entirely complexed with aluminum and consequently, the concentration of free F⁻ is low. As the pH increases, Al-OH complexes dominate over Al-F complexes and the free F⁻ levels increase. Fluoride forms stable complexes with calcium and magnesium, which are present in sea water. Calcium carbonate precipitation dominates the removal of dissolved fluoride from sea water. The residence time for fluoride in ocean sediment is calculated to be 2-3 million years. Fluorosilicic acid and hydrofluoric acid in high aquatic concentrations such as may be found in industrial waste ponds may volatilise, releasing silicon tetrafluoride and hydrogen fluoride into the atmosphere.

SAFETY DATA SHEET

Persistence and Degradability:	No data available
Bioaccumulation Potential:	No data available
Mobility in Soil:	No data available

13. DISPOSAL CONSIDERATIONS

Waste treatment methods:

Product/Packaging Disposal:	Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. Otherwise: Where possible retain label warnings and SDS and observe all notices pertaining to the product. 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 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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14. TRANSPORT INFORMATION

Road and Rail Transport (ADG):

UN Number:	1790
UN Proper Shipping Name:	HYDROFLUORIC ACID, with not more than 60% hydrogen fluoride
Transport Hazard Class:	Class 8 Sub risk 6.1
Packing Group:	II
Environmental Hazard:	Not applicable
Special Precautions for User:	Special provisions - Not Applicable Limited quantity - 1 L

Air Transport (ICAO-IATA/DGR)

UN Number:	1790
UN Proper Shipping Name:	Hydrofluoric acid 60% or less hydrogen fluoride



SAFETY DATA SHEET

Transport Hazard Class:	ICAO/IATA Class - 8 ICAO/IATA Sub risk - 6.1 ERG Code - 8P
Packing Group:	II
Environmental Hazard:	Not Applicable
<u>Sea Transport (IMDG-Code / GGVSee)</u>	
UN Number:	1790
UN Proper Shipping Name:	Hydrofluoric acid 60% or less hydrogen fluoride
Transport Hazard Class:	IMDG Class - 8 IMDG Sub risk - 6.1
Packing Group:	II
Environmental Hazard:	Not Applicable

15. REGULATORY INFORMATION

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP):	Schedule 7
Prohibition/Licensing Requirements:	N/A
This material/constituent(s) is covered by the following requirements:	All components of this product are listed or exempt from the Australian Inventory of Industrial Chemicals (AIIIC)

16. OTHER INFORMATION

Product is considered safe if used as intended.
Product is intended for professional dental use only.

This information was prepared in good faith from the best information available at the time of issue. It is based on the present level of research and to this extent we believe it is accurate. However, no guarantee of accuracy is made or implied and since conditions of use are beyond our control, all information relevant to usage is offered without warranty. The manufacturer will not be held responsible for any unauthorised use of this information or for any modified or altered versions.

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