Hornady Centerfire Rifle and Pistol Ammunition

TSA OUTDOORS

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5534-29**
Version No: **2.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **14/03/2022**
Print Date: **21/06/2022**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Hornady Centerfire Rifle and Pistol Ammunition
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	CARTRIDGES FOR WEAPONS, INERT PROJECTILE or CARTRIDGES, SMALL ARMS
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Firearm ammunition.

Details of the supplier of the safety data sheet

Registered company name	**TSA OUTDOORS**
Address	Unit 6/ 9 - 13 Winbourne Road Brookvale NSW 2100 Australia
Telephone	+61 2 9938 3244
Fax	+61 2 9939 2972
Website	Tsaoutdoors.com.au
Email	sales@tasco.com.au

Emergency telephone number

Association / Organisation	**Aaron Millard**
Emergency telephone numbers	+61 450 086 593 (Mon-Fri, 9 am-6pm)
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Explosives Division 1.4, Acute Toxicity (Oral) Category 3, Acute Toxicity (Dermal) Category 2, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2A, Acute Toxicity (Inhalation) Category 2, Carcinogenicity Category 1B, Reproductive Toxicity Category 1A, Reproductive Toxicity Effects on or via Lactation, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI*

Label elements

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

Hazard statement(s)

H204	Fire or projection hazard.
H301	Toxic if swallowed.
H310	Fatal in contact with skin.
H315	Causes skin irritation.
H319	Causes serious eye irritation.

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H330	Fatal if inhaled.
H350	May cause cancer.
H360Df	May damage the unborn child. Suspected of damaging fertility.
H362	May cause harm to breast-fed children.
H402	Harmful to aquatic life.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P234	Keep only in original packaging.
P250	Do not subject to grinding/shock/sources of friction.
P260	Do not breathe dust/fume.
P262	Do not get in eyes, on skin, or on clothing.
P263	Avoid contact during pregnancy and while nursing.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection, face protection and hearing protection.
P240	Ground and bond container and receiving equipment.
P273	Avoid release to the environment.
P284	[In case of inadequate ventilation] wear respiratory protection.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P330	Rinse mouth.
P361+P364	Take off immediately all contaminated clothing and wash it before reuse.
P370+P372+P380+P373	In case of fire: Explosion risk. Evacuate area. DO NOT fight fire when fire reaches explosives.
P370+P380+P375	In case of fire: Evacuate area. Fight fire remotely due to the risk of explosion.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P337+P313	If eye irritation persists: Get medical advice/attention.
P391	Collect spillage.
P332+P313	If skin irritation occurs: Get medical advice/attention.

Precautionary statement(s) Storage

P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.
P401	Store in accordance with local/regional/national/international regulations.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
P503	Refer to manufacturer or supplier for information on disposal/recovery/recycling.

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7440-50-8	5-<60	copper
7439-92-1	5-<50	lead
9004-70-0	5-<20	nitrocellulose
7440-66-6	1-<20	zinc
7429-90-5	0-3	aluminium
7440-36-0	0-2.5	antimony

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CAS No	%[weight]	Name
55-63-0	0-2.5	nitroglycerin
84-74-2	0-1.3	dibutyl phthalate
1345-04-6	<1	antimony trisulfide
10022-31-8	<1	barium nitrate
121-14-2	<1	2,4-dinitrotoluene
122-39-4	<1	diphenylamine
63918-97-8	<1	lead styphnate
7440-02-0	<1	nickel
78-11-5	<1	pentaerythritol tetranitrate
109-27-3	<1	tetrazene
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

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.
- **DO NOT** attempt to remove particles attached to or embedded in eye .
- Lay victim down, on stretcher if available and pad **BOTH** eyes, make sure dressing does not press on the injured eye by placing thick pads under dressing, above and below the eye.
- Seek urgent medical assistance, or transport to hospital.

Skin Contact

If skin or hair contact occurs:
- Immediately flush body and clothes with large amounts of water, using safety shower if available.
- Quickly remove all contaminated clothing, including footwear.
- Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre.
- Transport to hospital, or doctor.

In case of burns:
- Immediately apply cold water to burn either by immersion or wrapping with saturated clean cloth.
- **DO NOT** remove or cut away clothing over burnt areas. **DO NOT** pull away clothing which has adhered to the skin as this can cause further injury.
- **DO NOT** break blister or remove solidified material.
- Quickly cover wound with dressing or clean cloth to help prevent infection and to ease pain.
- For large burns, sheets, towels or pillow slips are ideal; leave holes for eyes, nose and mouth.
- **DO NOT** apply ointments, oils, butter, etc. to a burn under any circumstances.
- Water may be given in small quantities if the person is conscious.
- Alcohol is not to be given under any circumstances.
- Reassure.
- Treat for shock by keeping the person warm and in a lying position.
- Seek medical aid and advise medical personnel in advance of the cause and extent of the injury and the estimated time of arrival of the patient.
- Immediately wash contaminated skin with plenty of soap and water.
- Immediately seek medical advice.
- Wearing gloves, remove all contaminated clothing and loosen remaining clothing.
- Allow patient to assume comfortable position, keep warm.
- Keep at rest until fully recovered.
- If breathing has stopped or is shallow apply artificial respiration at once.
- In the event of cardiac arrest apply external cardiac massage.
- If breathing is laboured and patient cyanotic (blue), ensure airways are clear and have qualified person give oxygen through a face mask.

For thermal burns:
- Decontaminate area around burn.
- Consider the use of cold packs and topical antibiotics.

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

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

To prevent shock: (unless the person has a head, neck, or leg injury, or it would cause discomfort):
- Lay the person flat.
- Elevate feet about 12 inches.
- Elevate burn area above heart level, if possible.
- Cover the person with coat or blanket.
- Seek medical assistance.

For third-degree burns
Seek immediate medical or emergency assistance.
In the mean time:
- Protect burn area cover loosely with sterile, nonstick bandage or, for large areas, a sheet or other material that will not leave lint in wound.

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	▸ Separate burned toes and fingers with dry, sterile dressings. ▸ Do not soak burn in water or apply ointments or butter; this may cause infection. ▸ To prevent shock see above. ▸ For an airway burn, do not place pillow under the person's head when the person is lying down. This can close the airway. ▸ Have a person with a facial burn sit up. ▸ Check pulse and breathing to monitor for shock until emergency help arrives.
Inhalation	▸ 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. ▸ Remove victim from exposure - avoid becoming a casualty. ▸ Seek immediate medical advice and treat as for skin absorption.
Ingestion	▸ Give a slurry of activated charcoal in water to drink. **NEVER** GIVE AN UNCONSCIOUS PATIENT WATER TO DRINK. ▸ At least 3 tablespoons in a glass of water should be given. ▸ Although induction of vomiting may be recommended (**IN CONSCIOUS PERSONS ONLY**), such a first aid measure is dissuaded due to the risk of aspiration of stomach contents. (i) It is better to take the patient to a doctor who can decide on the necessity and method of emptying the stomach. (ii) Special circumstances may however exist; these include non-availability of charcoal and the ready availability of the doctor. **NOTE:** If vomiting is induced, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear protective gloves when inducing vomiting. ▸ REFER FOR MEDICAL ATTENTION WITHOUT DELAY. ▸ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▸ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. ▸ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. (ICSC20305/20307)

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
for copper intoxication:
- Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
- Administer egg white and other demulcents.
- Maintain electrolyte and fluid balances.
- Morphine or meperidine (Demerol) may be necessary for control of pain.
- If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
- Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
- If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
- It is unlikely that methylene blue would be effective against the occassional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
- Institute measures for impending renal and hepatic failure.

[GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]
- A role for activated charcoals for emesis is, as yet, unproven.
- In severe poisoning CaNa2EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]
- Chelation with British Anti-Lewisite (BAL) for serious antimony exposures should be employed.
- Dialyse as needed. The role of exchange diffusion is not clear.
- Be sure to monitor for dysrhythmias.

[Ellenhorn and Barceloux: Medical Toxicology]

Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce "metal fume fever" in workers from an acute or long term exposure.
- Onset occurs in 4-6 hours generally on the evening following exposure. Tolerance develops in workers but may be lost over the weekend. (Monday Morning Fever)
- Pulmonary function tests may indicate reduced lung volumes, small airway obstruction and decreased carbon monoxide diffusing capacity but these abnormalities resolve after several months.
- Although mildly elevated urinary levels of heavy metal may occur they do not correlate with clinical effects.
- The general approach to treatment is recognition of the disease, supportive care and prevention of exposure.
- Seriously symptomatic patients should receive chest x-rays, have arterial blood gases determined and be observed for the development of tracheobronchitis and pulmonary edema.

[Ellenhorn and Barceloux: Medical Toxicology]

Symptoms of vasodilation and reflex tachycardia may present following organic nitrate overdose; most organic nitrates are extensively metabolised by hydrolysis to inorganic nitrites. Organic nitrates and nitrites are readily absorbed through the skin, lungs, mucosa and gastro-intestinal tract.
- Gastric acids solubilise lead and its salts and lead absorption occurs in the small bowel.
- Particles of less than 1 um diameter are substantially absorbed by the alveoli following inhalation.
- Lead is distributed to the red blood cells and has a half-life of 35 days. It is subsequently redistributed to soft tissue & bone-stores or eliminated. The kidney accounts for 75% of daily lead loss; integumentary and alimentary losses account for the remainder.
- Neurasthenic symptoms are the most common symptoms of intoxication. Lead toxicity produces a classic motor neuropathy. Acute encephalopathy appears infrequently in adults. Diazepam is the best drug for seizures.
- Whole-blood lead is the best measure of recent exposure; free erythrocyte protoporphyrin (FEP) provides the best screening for chronic exposure. Obvious clinical symptoms occur in adults when whole-blood lead exceeds 80 ug/dL.
- British Anti-Lewisite is an effective antidote and enhances faecal and urinary excretion of lead. The onset of action of BAL is about 30 minutes and most of the chelated metal complex is excreted in 4-6 hours, primarily in the bile. Adverse reaction appears in up to 50% of patients given BAL in doses exceeding 5 mg/kg. CaNa2EDTA has also been used alone or in concert with BAL as an antidote. D-penicillamine is the usual oral agent for mobilisation of bone lead; its use in the treatment of lead poisoning remains investigational. 2,3-dimercapto-1-propanesulfonic acid (DMPS) and dimercaptosuccinic acid (DMSA) are water soluble analogues of BAL and their effectiveness is undergoing review. As a rule, stop BAL if lead decreases below 50 ug/dL; stop CaNa2EDTA if blood lead decreases below 40 ug/dL or urinary lead drops below 2 mg/24hrs.

[Ellenhorn & Barceloux: Medical Toxicology]

BIOLOGICAL EXPOSURE INDEX - BEI

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These represent the determinants observed in specimens collected from a healthy worker who has been exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Lead in blood	30 ug/100 ml	Not Critical	
2. Lead in urine	150 ug/gm creatinine	Not Critical	B
3. Zinc protoporphyrin in blood	250 ug/100 ml erythrocytes OR 100 ug/100 ml blood	After 1 month exposure	B

B: Background levels occur in specimens collected from subjects **NOT** exposed.

SECTION 5 Firefighting measures

Extinguishing media

- Do NOT direct a solid stream of water or foam into burning molten material; this may cause spattering and spread the fire.
- **DO NOT** use halogenated fire extinguishing agents.

Metal dust fires need to be smothered with sand, inert dry powders.
DO NOT USE WATER, CO2 or FOAM.

- Use DRY sand, graphite powder, dry sodium chloride based extinguishers, G-1 or Met L-X to smother fire.
- Confining or smothering material is preferable to applying water as chemical reaction may produce flammable and explosive hydrogen gas.
- Chemical reaction with CO2 may produce flammable and explosive methane.
- If impossible to extinguish, withdraw, protect surroundings and allow fire to burn itself out.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	**WARNING**: EXPLOSIVE MATERIALS / ARTICLES PRESENT! ▸ Evacuate all personnel and move upwind. ▸ Prevent re-entry. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May detonate and burning material may be propelled from fire. ▸ Wear full-body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage and fire effluent from entering drains and water courses. ▸ Fight fire from safe distances and from protected locations. ▸ Use flooding quantities of water. ▸ **DO NOT** approach containers or packages suspected to be hot. ▸ Cool any exposed containers not involved in fire from a protected location. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	Division 1.4 Substances, mixtures and articles which present no significant hazard: substances, mixtures and articles which present only a small hazard in the event of ignition or initiation. The effects are largely confined to the package and no projection of fragments of appreciable size or range is to be expected. An external fire shall not cause virtually instantaneous explosion of almost the entire contents of the package. Combustion products include: carbon monoxide (CO) carbon dioxide (CO2) nitrogen oxides (NOx) metal oxides other pyrolysis products typical of burning organic material.
HAZCHEM	1YE

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	Environmental hazard - contain spillage. **WARNING!: EXPLOSIVE**. BLAST and/or PROJECTION and/or FIRE HAZARD ▸ Clean up all spills immediately. ▸ Avoid inhalation of the material and avoid contact with eyes and skin. ▸ Wear impervious gloves and safety glasses. ▸ Remove all ignition sources. ▸ Use spark-free tools when handling. ▸ Sweep into non-sparking containers or barrels and moisten with water. ▸ Place spilled material in clean, sealable, labelled container for disposal. ▸ Flush area with large amounts of water.
Major Spills	Environmental hazard - contain spillage. · Do not use compressed air to remove metal dusts from floors, beams or equipment · Vacuum cleaners, of flame-proof design, should be used to minimise dust accumulation. · Use non-sparking handling equipment, tools and natural bristle brushes. · Provide grounding and bonding where necessary to prevent accumulation of static charges during metal dust handling and transfer operations · Cover and reseal partially empty containers. · Do not allow chips, fines or dusts to contact water, particularly in enclosed areas. If molten: ▸ Contain the flow using dry sand or salt flux as a dam. ▸ All tooling (e.g., shovels or hand tools) and containers which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use.

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	▸ Allow the spill to cool before remelting scrap. **WARNING!**: EXPLOSIVE. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ May be violently or explosively reactive. ▸ Wear full body protective clothing with breathing apparatus. ▸ Consider evacuation (or protect in place). ▸ In case of transport accident notify Police, Emergency Authority, Competent Explosives Authority or Manufacturer. ▸ No smoking, naked lights, heat or ignition sources. ▸ Increase ventilation. ▸ Use extreme caution to prevent physical shock. ▸ Use only spark-free shovels and explosion-proof equipment. ▸ Collect recoverable material and segregate from spilled material. ▸ Wash spill area with large quantities of water.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	For molten metals: · Molten metal and water can be an explosive combination. The risk is greatest when there is sufficient molten metal to entrap or seal off water. Water and other forms of contamination on or contained in scrap or remelt ingot are known to have caused explosions in melting operations. While the products may have minimal surface roughness and internal voids, there remains the possibility of moisture contamination or entrapment. If confined, even a few drops can lead to violent explosions. · All tooling, containers, molds and ladles, which come in contact with molten metal must be preheated or specially coated, rust free and approved for such use. · Any surfaces that may contact molten metal (e.g. concrete) should be specially coated · Drops of molten metal in water (e.g. from plasma arc cutting), while not normally an explosion hazard, can generate enough flammable hydrogen gas to present an explosion hazard. Vigorous circulation of the water and removal of the particles minimise the hazard. During melting operations, the following minimum guidelines should be observed: · Inspect all materials prior to furnace charging and completely remove surface contamination such as water, ice, snow, deposits of grease and oil or other surface contamination resulting from weather exposure, shipment, or storage. · Store materials in dry, heated areas with any cracks or cavities pointed downwards. · Preheat and dry large objects adequately before charging in to a furnace containing molten metal. This is typically done by the use of a drying oven or homogenising furnace. The dry cycle should bring the metal temperature of the coldest item of the batch to 200 degree C (400 deg F) and then hold at that temperature for 6 hours. ▸ The greatest potential for injury caused by molten materials occurs during purging of machinery (moulders, extruders etc.) ▸ It is essential that workers in the immediate area of the machinery wear eye and skin protection (such as full face, safety glasses, heat resistant gloves, overalls and safety boots) as protection from thermal burns. ▸ Fumes or vapours emitted from hot melted materials, during converting operations, may condense on overhead metal surfaces or exhaust ducts. The condensate may contain substances which are irritating or toxic. Avoid contact of that material with the skin. Wear rubber or other impermeable gloves when cleaning contaminated areas. ▸ Avoid process temperatures above decomposition temperatures. Overheating may occur at excessively high cylinder heats, overworking of the melt by wrong screw configuration, or by long dwell time in the machine. Under such conditions, thermal emissions and heat-degradation products might, without proper ventilation, reach hazardous concentrations in the converting area. Hot purgings should be collected only as thin flat strands to allow for rapid cooling. Hot purgings should be cooled by quenching in water in a well-ventilated area. ▸ Handle gently. Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Avoid all personal contact, including inhalation. ▸ Avoid smoking, naked lights, heat or ignition sources. ▸ Explosives must not be struck with metal implements. ▸ Avoid mechanical and thermal shock and friction. ▸ Use in a well ventilated area. ▸ Avoid contact with incompatible materials. ▸ When handling **DO NOT** eat, drink or smoke. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Organic powders when finely divided over a range of concentrations regardless of particulate size or shape and suspended in air or some other oxidizing medium may form explosive dust-air mixtures and result in a fire or dust explosion (including secondary explosions) ▸ Minimise airborne dust and eliminate all ignition sources. Keep away from heat, hot surfaces, sparks, and flame. ▸ Establish good housekeeping practices. ▸ Remove dust accumulations on a regular basis by vacuuming or gentle sweeping to avoid creating dust clouds. ▸ Use continuous suction at points of dust generation to capture and minimise the accumulation of dusts. Particular attention should be given to overhead and hidden horizontal surfaces to minimise the probability of a "secondary" explosion. According to NFPA Standard 654, dust layers 1/32 in.(0.8 mm) thick can be sufficient to warrant immediate cleaning of the area. ▸ Do not use air hoses for cleaning. ▸ Minimise dry sweeping to avoid generation of dust clouds. Vacuum dust-accumulating surfaces and remove to a chemical disposal area. Vacuums with explosion-proof motors should be used. ▸ Control sources of static electricity. Dusts or their packages may accumulate static charges, and static discharge can be a source of ignition. ▸ Solids handling systems must be designed in accordance with applicable standards (e.g. NFPA including 654 and 77) and other national guidance. ▸ Do not empty directly into flammable solvents or in the presence of flammable vapors. ▸ The operator, the packaging container and all equipment must be grounded with electrical bonding and grounding systems. Plastic bags and plastics cannot be grounded, and antistatic bags do not completely protect against development of static charges. Empty containers may contain residual dust which has the potential to accumulate following settling. Such dusts may explode in the presence of an appropriate ignition source. ▸ Do NOT cut, drill, grind or weld such containers. ▸ In addition ensure such activity is not performed near full, partially empty or empty containers without appropriate workplace safety authorisation or permit.
Other information	▸ **DO NOT** store near acids, or oxidising agents ▸ Store cases in a well ventilated magazine licensed for the appropriate Class, Division and Compatibility Group. ▸ Rotate stock to prevent ageing. Use on FIFO (first in-first out) basis. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

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	▸ Store in a cool place in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights, heat or ignition sources. ▸ Store in an isolated area away from other materials. ▸ Keep storage area free of debris, waste and combustibles. ▸ Protect containers against physical damage. ▸ Check regularly for spills and leaks **NOTE**: If explosives need to be destroyed contact the Competent Authority.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Bulk bags: Reinforced bags required for dense materials. ▸ **CARE**: Packing of high density product in light weight metal or plastic packages may result in container collapse with product release ▸ All packaging for Class 1 Goods shall be in accordance with the requirements of the relevant Code for the transport of Dangerous Goods. ▸ Class 1 is unique in that the type of packaging used frequently has a very decisive effect on the hazard and therefore on the assignment to a particular division ▸ Heavy gauge metal packages / Heavy gauge metal drums
Storage incompatibility	Inorganic derivative of Group 11 metal. For aluminas (aluminium oxide): Incompatible with hot chlorinated rubber. In the presence of chlorine trifluoride may react violently and ignite. -May initiate explosive polymerisation of olefin oxides including ethylene oxide. -Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals. -Produces exothermic reaction with oxygen difluoride. -May form explosive mixture with oxygen difluoride. -Forms explosive mixtures with sodium nitrate. -Reacts vigorously with vinyl acetate. Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt. The substance may be or contains a "metalloid" The following elements are considered to be metalloids; boron,silicon, germanium, arsenic, antimony, tellurium and (possibly) polonium The electronegativities and ionisation energies of the metalloids are between those of the metals and nonmetals, so the metalloids exhibit characteristics of both classes. The reactivity of the metalloids depends on the element with which they are reacting. For example, boron acts as a nonmetal when reacting with sodium yet as a metal when reacting with fluorine. Unlike most metals, most metalloids are amphoteric- that is they can act as both an acid and a base. For instance, arsenic forms not only salts such as arsenic halides, by the reaction with certain strong acid, but it also forms arsenites by reactions with strong bases. Most metalloids have a multiplicity of oxidation states or valences. For instance, tellurium has the oxidation states +2, -2, +4, and +6. Metalloids react like non-metals when they react with metals and act like metals when they react with non-metals. The material is described as an electropositive metal. The activity or electromotive series of metals is a listing of the metals in decreasing order of their reactivity with hydrogen-ion sources such as water and acids. In the reaction with a hydrogen-ion source, the metal is oxidised to a metal ion, and the hydrogen ion is reduced to H2. The ordering of the activity series can be related to the standard reduction potential of a metal cation. The more positive the standard reduction potential of the cation, the more difficult it is to oxidise the metal to a hydrated metal cation and the later that metal falls in the series Three notable groups comprise the series ▸ very electropositive metals ▸ electropositive metals ▸ electronegative metals Electropositive metals.have electronegativities that fall between 1.4 and 1.9 Cations of these metals generally have standard reduction potentials between 0.0 and -1.6 V They: ▸ do not react very readily with water to release hydrogen ▸ react with H+ (acids) Electropositive metals do not burn in air as readily as do very electropositive metals. The surfaces of these metals will tarnish in the presence of oxygen forming a protective oxide coating. This coating protects the bulk of the metal against further oxidation (the metal is passivated). Reaction is reduced in the massive form (sheet, rod, or drop), compared with finely divided forms. The less active metals will not burn in air but: ▸ can react exothermically with oxidising acids to form noxious gases. ▸ catalyse polymerisation and other reactions, particularly when finely divided ▸ react with halogenated hydrocarbons (for example, copper dissolves when heated in carbon tetrachloride), sometimes forming explosive compounds. ▸ Elemental metals may react with azo/diazo compounds to form explosive products ▸ Finely divided metal powders develop pyrophoricity when a critical specific surface area is exceeded; this is ascribed to high heat of oxide formation on exposure to air. ▸ Safe handling is possible in relatively low concentrations of oxygen in an inert gas ▸ Several pyrophoric metals, stored in glass bottles have ignited when the container is broken on impact. Storage of these materials moist and in metal containers is recommended. ▸ The reaction residues from various metal syntheses (involving vacuum evaporation and co-deposition with a ligand) are often pyrophoric If the surface of the metal is in contact with both oxygen and water, corrosion can occur. In corrosion, the metal acts as an anode and is oxidised. Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid. Some electropositive metals do not react with nitric acid because they are passivated. http://www.wou.edu/las/physci/ch412/activity.htm Phthalates: ▸ react with strong acids, strong oxidisers, permanganates and nitrates ▸ attack some form of plastics Metal nitrites: ▸ are incompatible with chlorates, hypophosphites, iodides, mercury salts, permanganates, sulfites, primary amines and amides, secondary amines and amides, ammonium salts, activated carbon, cyanogen compounds, thiocyanates, thiosulfates, cyanides, sodium amide, boron, acetanilide, antipyrine, tannic acid and cellulose ▸ react explosively with hydrazine and liquid ammonia . ▸ react explosively following fusion with metal cyanides ▸ react (often) with salts of nitrogenous bases to produce an unstable corresponding nitrite salt.

Continued...

- WARNING: Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.
- The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono-or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
- Avoid reaction with borohydrides or cyanoborohydrides
- Segregate from alcohol, water.
- Avoid strong acids, bases.
- Explosion hazard may follow contact with incompatible materials
- Avoid reaction with oxidising agents
- Many metals may incandesce, react violently, ignite or react explosively upon addition of concentrated nitric acid.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	lead	Lead, inorganic dusts & fumes (as Pb)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (metal dust)	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium (welding fumes) (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	aluminium	Aluminium, pyro powders (as Al)	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	antimony	Antimony & compounds (as Sb)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nitroglycerin	Nitroglycerine (NG)	0.05 ppm / 0.46 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	dibutyl phthalate	Dibutyl phthalate	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	antimony trisulfide	Antimony & compounds (as Sb)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	barium nitrate	Barium, soluble compounds (as Ba)	0.5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	diphenylamine	Diphenylamine	10 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, metal	1 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	nickel	Nickel, powder	1 mg/m3	Not Available	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
copper	3 mg/m3	33 mg/m3	200 mg/m3
lead	0.15 mg/m3	120 mg/m3	700 mg/m3
zinc	6 mg/m3	21 mg/m3	120 mg/m3
antimony	1.5 mg/m3	13 mg/m3	80 mg/m3
nitroglycerin	0.1 mg/m3	2 mg/m3	75 mg/m3
dibutyl phthalate	15 mg/m3	1,600 mg/m3	9300* mg/m3
barium nitrate	2.9 mg/m3	350 mg/m3	2,100 mg/m3
2,4-dinitrotoluene	0.6 mg/m3	12 mg/m3	200 mg/m3
diphenylamine	30 mg/m3	180 mg/m3	220 mg/m3
nickel	4.5 mg/m3	50 mg/m3	99 mg/m3
pentaerythritol tetranitrate	5 mg/m3	55 mg/m3	330 mg/m3

Ingredient	Original IDLH	Revised IDLH
copper	100 mg/m3	Not Available
lead	Not Available	Not Available
nitrocellulose	Not Available	Not Available
zinc	Not Available	Not Available
aluminium	Not Available	Not Available
antimony	Not Available	Not Available
nitroglycerin	75 mg/m3	Not Available
dibutyl phthalate	4,000 mg/m3	Not Available
antimony trisulfide	50 mg/m3	Not Available
barium nitrate	50 mg/m3	Not Available
2,4-dinitrotoluene	Not Available	Not Available
diphenylamine	Not Available	Not Available
lead styphnate	100 mg/m3	Not Available
nickel	10 mg/m3	Not Available
pentaerythritol tetranitrate	Not Available	Not Available
tetrazene	Not Available	Not Available

Continued...

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
2,4-dinitrotoluene	E	≤ 0.01 mg/m³
lead styphnate	E	≤ 0.01 mg/m³
pentaerythritol tetranitrate	E	≤ 0.01 mg/m³
tetrazene	E	≤ 0.01 mg/m³
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

MATERIAL DATA

Exposure controls

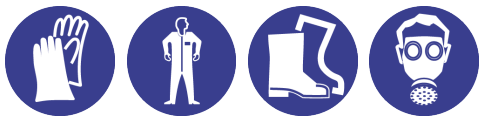
Appropriate engineering controls

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.

- Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- Work should be undertaken in an isolated system such as a "glove-box" . Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.
- Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within.
- Open-vessel systems are prohibited.
- Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation.
- Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system.
- For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas).
- Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air.
- Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.

For molten materials:
Provide mechanical ventilation; in general such ventilation should be provided at compounding/ converting areas and at fabricating/ filling work stations where the material is heated. Local exhaust ventilation should be used over and in the vicinity of machinery involved in handling the molten material.
Keep dry!!
Processing temperatures may be well above boiling point of water, so wet or damp material may cause a serious steam explosion if used in unvented equipment.

Personal protection



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]

Skin protection

See Hand protection below

Hands/feet protection

- Elbow length PVC gloves

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,

Continued...

	· glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Protective gloves eg. Leather gloves or gloves with Leather facing · Non-sparking or conductive footwear essential. Conductive footwear describes a boot or shoe with a sole made from a conductive compound chemically bound to the bottom components, for permanent control to electrically ground the foot an shall dissipate static electricity from the body to reduce the possibility of ignition of volatile compounds. Electrical resistance must range between 0 to 500,000 ohms. Conductive shoes should be stored in lockers close to the room in which they are worn. Personnel who have been issued conductive footwear should not wear them from their place of work to their homes and return.
Body protection	See Other protection below
Other protection	▸ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▸ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▸ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▸ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▸ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. For handling explosives or explosive compositions: ▸ Wear close-fitting flame-protection treated clothing closed at the neck and sleeves. ▸ Cotton underwear, socks and conductive shoes are recommended to avoid human static discharge. · During repair or maintenance activities the potential exists for exposures to toxic metal particulate in excess of the occupational standards. Under these circumstances, protecting workers can require the use of specific work practices or procedures involving the combined use of ventilation, wet and vacuum cleaning methods, respiratory protection, decontamination, special protective clothing, and when necessary, restricted work zones. · Protective over-garments or work clothing must be worn by persons who may become contaminated with particulate during activities such as machining, furnace rebuilding, air cleaning equipment filter changes, maintenance, furnace tending, etc. Contaminated work clothing and over-garments must be managed in a controlled manner to prevent secondary exposure to workers of third parties, to prevent the spread of particulate to other areas, and to prevent particulate from being taken home by workers. · Personnel who handle and work with molten metal should utilise primary protective clothing like polycarbonate face shields, fire resistant tapper's jackets, neck shades (snoods), leggings, spats and similar equipment to prevent burn injuries. In addition to primary protection, secondary or day-to-day work clothing that is fire resistant and sheds metal splash is recommended for use with molten metal. Synthetic materials should never be worn even as secondary clothing (undergarments). Manufacture may require: ▸ Non-static flame retardant treated clothing ▸ Access to deluge Safety shower ▸ Barrier cream.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Hornady Centerfire Rifle and Pistol Ammunition

Material	CPI
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C

Respiratory protection

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

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

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

Continued...

NITRILE	C
PE/EVAL/PE	C
PVA	C
SARANEX-23	C
VITON	C

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

· Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
· The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
· Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
· Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
· Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
· Use approved positive flow mask if significant quantities of dust becomes airborne.
· Try to avoid creating dust conditions.
Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.
Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.
Filtration rate: Filters at least 99.95% of airborne particles
Suitable for:
· Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
· Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
· Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
· Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos
Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.
For molten materials:
76a-p()

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Solid; insoluble in water.

Physical state	Manufactured	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	Not Applicable	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Applicable
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Applicable	**Gas group**	Not Available
Solubility in water	Immiscible	**pH as a solution (Not Available%)**	Not Applicable
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Presence of shock and friction ▸ Presence of heat source and ignition source ▸ Product is considered stable under normal handling conditions. ▸ Stable under normal storage conditions. ▸ Hazardous polymerization will not occur. Avoid contact with other chemicals.

Continued...

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	Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of dusts, generated by the material during the course of normal handling, may produce severe damage to the health of the individual. Relatively small amounts absorbed from the lungs may prove fatal. Inhalation hazard is increased at higher temperatures. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of fume may aggravate a pre-existing respiratory condition such as asthma, bronchitis, emphysema Copper poisoning following exposure to copper dusts and fume may result in headache, cold sweat and weak pulse. Capillary, kidney, liver and brain damage are the longer term manifestations of such poisoning. Inhalation of freshly formed metal oxide particles sized below 1.5 microns and generally between 0.02 to 0.05 microns may result in "metal fume fever". Symptoms may be delayed for up to 12 hours and begin with the sudden onset of thirst, and a sweet, metallic or foul taste in the mouth. Other symptoms include upper respiratory tract irritation accompanied by coughing and a dryness of the mucous membranes, lassitude and a generalised feeling of malaise. Mild to severe headache, nausea, occasional vomiting, fever or chills, exaggerated mental activity, profuse sweating, diarrhoea, excessive urination and prostration may also occur. Tolerance to the fumes develops rapidly, but is quickly lost. All symptoms usually subside within 24-36 hours following removal from exposure. Inhalation of antimony and its compounds may produce respiratory and gastrointestinal tract discomfort with sore throat, shallow respiration, coughing, headaches, breathing difficulties, dizziness, weight loss, gingivitis, anaemia, eosinophilia and enzyme inhibition. Inflammation of the upper and lower respiratory tract may occur. Pulmonary congestion and oedema may also occur. Other symptoms include rhinitis, eye irritation, vomiting and diarrhoea, weight loss, dysomnia, hair loss and haematological disorders. Death due to circulatory failure has been described, with pathology showing acute congestion of the heart (myocardial failure), liver and kidneys.
Ingestion	Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract Not normally a hazard due to physical form of product.
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 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	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	On the basis, primarily, of animal experiments, the material may be regarded as carcinogenic to humans. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in cancer on the basis of: - appropriate long-term animal studies - other relevant information There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility There is sufficient evidence to establish a causal relationship between human exposure to the material and subsequent developmental toxic effects in the off-spring. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects. There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. The various phthalates have different uses, chemical structures and toxicity profiles. It is therefore difficult to generalise about the safety of all phthalates as a group. The main health concern associated with some phthalates is that animal studies have shown that high regular doses can affect the reproductive system in developing young, particularly males. While there is no significant risk to the general population, young children may experience higher exposures than the general population if they chew or suck on phthalate-containing toys, or if they ingest phthalates over a long period from other products containing high levels of phthalates. In animal tests, phthalates have been shown to "feminise" male animals, increasing the likelihood of small or undeveloped testes, undescended testicles, and low sperm counts. A 2005 study also linked higher foetal exposure to phthalates through the mother's blood with increased risk of

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developmental abnormalities in male infants. Higher phthalate levels are also associated with lower testosterone production and reduced sperm count in men.
One study suggested that high levels of phthalates may be connected to the current obesity epidemic in children. It was found that obese children show greater exposure to phthalates than non-obese children. It was reported that the obesity risk increases according to the level of the chemical found in the children's bloodstream. in a national cross-section of U.S. men, concentrations of several prevalent phthalate metabolites showed statistically significant correlations with abnormal obesity and insulin resistance. A further study found that people with elevated phthalate levels had roughly twice the risk of developing diabetes compared with those with lower levels. This study also found that phthalates were associated with disrupted insulin production.
Much of the current research on effects of phthalate exposure has been focused towards children and men's health, however, women may be at higher risk for potential adverse health effects of phthalates due to increased cosmetic use. According to in vivo and observational studies there is an association between phthalate exposure and endocrine disruption leading to development of breast cancer. This finding may be associated with the demethylation of the oestrogen receptor complex in breast cancer cells.
A Russian study describes exposure by workers to mixed phthalates (and other plasticisers) - pain, numbness and spasms in the upper and lower extremities were related to duration of exposures. Symptoms usually developed after the sixth or seventh year of work. Neurological studies revealed the development of polyneuritis in about 30% of the workers involved in this study. About 30% of the workforce showed depression of the vestibular receptors. Because the study described mixed exposures it is difficult to determine what, if any, unique role was played by the phthalates. Increased incidences of anovulatory reproductive cycles and low oestrogen concentrations were reported among Russian women working with phthalate plasticisers; the abnormal cycles were associated with spontaneous abortion. The specific phthalates implicated, dose levels and other data were not reported. It has been alleged that the phthalates mimic or interfere with sex packaging) and are used as ingredients in paints, inks and adhesives. Their potential for entering the human body is marked. They have been added to a list of chemicals (including alkyl phenolics, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and dioxins) which are implicated in reducing sperm counts and fertility in males a phenomenon which has apparently arisen since the mid 1960s.
Phthalates are generally considered to be in a class of endocrine disruptors known as "xenoestrogens," for their ability to mimic the effect of oestrogen on the body.
Although the human foetus is "bathed" in naturally occurring oestrogens during pregnancy it is suggested that it has developed a protective mechanism against natural oestrogens but is not safe from synthetic variants. These tend to accumulate in body fats which sets them apart from the natural product. During early pregnancy, fats are broken down and may flood the body with concentrated pollutants
Human phthalate exposure during pregnancy results in decreased anogenital distance among baby boys.Boys born to mothers with the highest levels of phthalates were 7 times more likely to have a shortened anogenital distance.
While anogenital distance is routinely used as a measure of foetal exposure to endocrine disruptors in animals, this parameter is rarely assessed in humans, and its significance is unknown
One study also found that female animals exposed to higher levels of phthalates experienced increased risk of miscarriage, a common symptom of excessive estrogen levels in human women, and stillbirth. Prematurity may also be linked to phthalate exposure.
Another study found a link between exposure to phthalates and increased rates of childhood obesity.
In adult human men, phthalates have been linked to greater waist circumference and higher insulin resistance, a common precursor to type 2 (adult onset) diabetes. They have been linked to thyroid irregularities, asthma, and skin allergies in both sexes. Though the exact mechanism is unclear, studies have linked higher rates of respiratory infections and other symptoms in children living in houses with vinyl floors. One possible explanation is inhalation of dust tainted by phthalates, which are used in cosmetics such as nail polishes and hand creams precisely because of their ability to bind to human tissues.
Animal studies have shown increased risks of certain birth defects (including the genital abnormalities and, in rats, extra ribs) and low birth rates in rats whose mothers were fed higher levels of phthalates.
These effects on foetal development are of particular concern because young women of childbearing age often have higher than average phthalate levels in the body thanks to their use of cosmetics, many of which contain phthalates.
The EU has applied limitations to the use of several phthalates in general food contact applications (packaging and closures) and medical device applications. The USA has introduced regulation of phthalate esters as components of children's toys and childcare articles for children under the age of 12 that could be 'placed in the mouth'.
Endocrine disruptors such as phthalates can be add to the effects of other endocrine disruptors, so even very small amounts can interact with other chemicals to have cumulative, adverse "cocktail effects"
Large amounts of specific phthalates fed to rodents have been shown to damage their liver and testes, and initial rodent studies also indicated hepatocarcinogenicity. Later studies on primates showed that the mechanism is specific to rodents - humans are resistant to the effect
Studies conducted on mice exposed to phthalates in utero did not result in metabolic disorder in adults. However, "At least one phthalate, monoethyhexyl phthalate (MEHP) has been found to interact with all three peroxisome proliferator-activated receptors (PPARs) PPARs are members of the nuclear receptor superfamily involved in lipid and carbohdrate metabolism.
Prenatal exposure to phthalates may affect children's mental, motor and behavioral development during the preschool year.
A 2009 study found that prenatal phthalate exposure was related to low birth weight in infants. Low birth weight is the leading cause of death in children under 5 years of age and increases the risk of cardiovascular and metabolic disease in adulthood. Another study found that women who deliver prematurely have, on average, up to three times the phthalate level in their urine compared to women who carry to term.
Several findings point to a statistically significant correlation between urine phthalate concentrations in children and symptoms of attention deficit hyperactivity disorder (ADHD)
Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.
Very fine Al_2O_3 powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed
at periods up to 12 months following the last exposure.
When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.
The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis(aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.
Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.
There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveolii (sub 5 um) are able to produce pathogenic effects in the lungs.

Excessive exposure to lead can affect the blood, the nervous system, heart, endocrine organs and the immune system and the digestive system. The synthesis of haemoglobin is inhibited and can result in anaemia. If left untreated, neuromuscular dysfunction, possible paralysis and encephalopathy (brain tissue damage) may result. Other symptoms of overexposure include joint and muscle pain, weakness of the extensor muscles (frequently the hand and wrist), headache, dizziness, abdominal pain, diarrhoea, constipation, nausea, vomiting, blue line on the gums, insomnia and metallic taste. High body levels produce cerebrospinal pressure, brain damage with stupor leading to coma and, in some cases, death. Early symptoms of lead poisoning ("plumbism") include anorexia and loss of weight, constipation, apathy or irritability, occasional vomiting, fatigue, headache, weakness, and a metallic taste in the mouth. Advanced poisonings are characterised by intermittent vomiting, irritability,

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nervousness, myalgia of the arms and legs (often with wrist and foot drop). Severe poisonings may produce persistent vomiting, ataxia, stupor or lethargy, visual disturbances progressing to optic neuritis and atrophy, hyper- tension, papilloedema, cranial nerve paralysis, delirium, convulsions and coma. Neurological effects include mental retardation, seizures, cerebral palsy and marked muscular contractions that distort the spine, limbs, hips and sometimes the cranial inervated muscles (dystonia musculorum deformans). Industrial exposure has been associated with irreversible kidney damage.
Lead is a cumulative poison with adverse effects in pregnancy [NIOSHTIC]
Lead salts have been reported to cross the placenta and induce embryo- and foeto-mortality. They also may have a teratogenic effect (causing birth deformities) in certain animal species. Organometallic lead may not produce these effects. Adverse effects of lead on human reproduction, embryonic and foetal development and postnatal mental development have also been recorded. Foetal exposure to lead may result in birth defects, mental retardation, behavioural disorders and death during the first year of childhood. Paternal effects may include reduced sex drive, impotence, sterility and adverse effects on the sperm which in turn may increase the potential for increased birth defects. Maternal effects may include miscarriage and stillbirth in exposed women, or women whose husbands might be exposed, sterility or decreased fertility, and abnormal menses. Exposure by both parents to lead may exacerbate the reproductive effects.
Oral or intraperitoneal administration of dibutyl phthalate, at high doses relative to the TLV, produced a number of resorptions, neural tube defects, skeletal abnormalities and increased foetal deaths.
For copper and its compounds (typically copper chloride):
Acute toxicity: There are no reliable acute oral toxicity results available. Animal testing shows that skin in exposure to copper may lead to hardness of the skin, scar formation, exudation and reddish changes. Inflammation, irritation and injury of the skin were noted.
Repeat dose toxicity: Animal testing shows that very high levels of copper monochloride may cause anaemia.
Genetic toxicity: Copper monochloride does not appear to cause mutations in vivo, although chromosomal aberrations were seen at very high concentrations in vitro.
Cancer-causing potential: There was insufficient information to evaluate the cancer-causing activity of copper monochloride.
The major concern of possible long-term effects of exposure to nitrate and nitrite is associated with formation of nitroso compounds, many of which are carcinogenic. This formation may take place wherever nitrite and nitrosable compounds are present, but it is favoured by acidic conditions or the presence of some bacteria. The gastrointestinal tract and especially the stomach is regarded as the main formation site, but nitrosation reactions can also take place in an infected urinary bladder.
Nitrite is mutagenic in a number of in vitro assays against microorganisms or cultured mammalian cells. Nitrates show no mutagenic activity in microbial tests under aerobic conditions. Activity has been reported under anaerobic conditions, probably due to reduction of nitrate into nitrite
The mutagenic effects of nitrites were observed in an in vivo and in vitro experiment using Syrian hamsters. In vivo assays have been equivocal, both positive and negative results having been reported
Exposure to sodium nitrite in drinking water resulted in an increased incidence of epithelial hyperplasia in the forestomach of male and female rats and in the glandular stomach of male mice.
There was equivocal evidence of carcinogenic activity of sodium nitrite in female B6C3F1 mice based on the positive trend in the incidences of squamous cell papillomas or carcinomas (combined) of the forestomach. There was no evidence of carcinogenic activity in male and female F344/N rats or B6C3F1 male mice exposed to 750, 1500 or 3000 ppm.
NTP Technical Report Series No. 495, May 2001
Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines.
Sodium nitrite (60 mg/kg) administered in drinking water to pregnant guinea pigs produced maternal anaemia and increased the incidences of abortion and foetal mortality. Administration of 2000-3000 mg/l sodium nitrite in drinking water, to pregnant rats, produced 30-53% foetal mortality. In rat dams given 0.025-0.5% in feed, sodium nitrite caused an increase in foetal and pup mortality and decreases in pre-weanling body weights.
Repeated or prolonged exposure to antimony and its compounds may produce stomatitis, dry throat, metallic taste, gingivitis, septal and laryngeal perforation, laryngitis, headache, dyspnea, indigestion, nausea, vomiting, diarrhoea, anorexia, anaemia, weight loss, pain and chest tightness, sleeplessness, muscular pain and weakness, dizziness, pharyngitis,tracheitis, bronchitis, pneumonitis, benign pneumoconiosis (with obstructive lung disease and emphysema) and haematological disorders. Degenerative changes of the liver and kidney may occur. Symptoms can be variable, and may including fatigue, myopathy (muscle aches and inflammation), hypotension, angina and immune dysregulation and hypertrophy of splenic follicles.
Antimony's deposition in body tissues and its detrimental effects depend upon the oxidation state of the element. The trivalent antimony compounds are cardiotoxic. Trivalent antimony affects liver functions, impairs enzymes, and may interfere with sulfur chemistry. If antimony impairs phosphofructokinase (PFK), then purine metabolism may be disrupted, resulting in elevated blood and/or urine levels of hypoxanthine, uric acid and possibly ammonia. Pentavalent antimony deposits in bone, kidney, and in organs of the endocrine system.
Chronic exposure to antimony compounds may result in itchiness, papules and pustules around sweat and sebaceous glands ("antimony spots"), but rarely around the face, and dermatitis.
Collapse and sudden death due to anaphylactic-type reactions have occurred. Therapeutic doses given intravenously cause nausea, vomiting, cough and abdominal pain and diarrhoea. Other side-effects include anorexia, chest, muscle and joint pains, pruritus, skin rashes, dizziness and oedema. Renal and hepatic damage occur rarely and haemolytic anaemia has been reported. Continuous treatment with small doses of antimony may give rise to subacute poisoning similar to chronic arsenic poisoning.
Smelter workers often show skin rashes on the forearms and thighs resembling chicken pox pustules.
Workers exposed to inorganic antimony compounds show a benign pneumoconiosis and obstructive lung disease - these are probably non-specific. Woman appear to more susceptible to systemic effects following exposure. Antimony crosses the placenta, is present in amniotic fluids, and is excreted in breast milk. There are suggestions that exposure may produce an increased incidence of spontaneous late abortions, premature births, and gynecological problems among female antimony smelter workers. An excess of deaths from lung cancer has been reported in smelter workers with more than 7 years exposure to relatively high levels of dust and fume. Animal studies demonstrate that the dust may produce pathological changes in cardiac muscle and may produce an interstitial pneumonitis and endogenous pneumonia. One animal study has also suggested that inhalation of the dust by rats induced a significantly increased incidence of carcinogenic tumours of the lungs and thorax.
Increased chromosome defects occur when human lymphocytes are incubated with a soluble antimony salt.
The inhalation data suggests that the myocardium is a target of antimony toxicity. It is possible that antimony affects circulating glucose by interfering with enzymes of the glycogenolysis and gluconeogenesis pathways. The mechanism of action of antimony remains unclear. However, some studies suggest that antimony combines with sulfhydryl groups including those in several enzymes important for tissue respiration
Following an oral intake of extremely high doses of zinc (where 300 mg Zn/d – 20 times the US Recommended Dietary Allowance (RDA) – is a "low intake" overdose), nausea, vomiting, pain, cramps and diarrhea may occur. There is evidence of induced copper deficiency, alterations of blood lipoprotein levels, increased levels of LDL, and decreased levels of HDL at long-term intakes of 100 mg Zn/d. The USDA RDA is 15 mg Zn/d.
There is also a condition called the "zinc shakes" or "zinc chills" or metal fume fever that can be induced by the inhalation of freshly formed zinc oxide formed during the welding of galvanized materials.
Supplemental zinc can prevent iron absorption, leading to iron deficiency and possible peripheral neuropathy, with loss of sensation in extremities.
Zinc is necessary for normal fetal growth and development. Fetal damage may result from zinc deficiency. Only one report in the literature suggested adverse developmental effects in humans due to exposure to excessive levels of zinc. Four women were given zinc supplements of 0.6 mg zinc/kg/day as zinc sulfate during the third trimester of pregnancy. Three of the women had premature deliveries, and one delivered a stillborn infant. However, the significance of these results cannot be determined because very few details were given regarding the study protocol, reproductive histories, and the nutritional status of the women. Other human studies have found no developmental effects in the newborns of mothers consuming 0.3 mg zinc/kg/day as zinc sulfate or zinc citrate or 0.06 mg zinc/kg/day as zinc aspartate during the last two trimesters. There has been a suggestion that increased serum zinc levels in pregnant women may be associated with an increase in neural tube defects, but others have failed to confirm this association. The developmental toxicity of zinc in experimental animals has been evaluated in a number of investigations. Exposure to high levels of zinc in the diet prior to and/or during gestation has been associated with increased fetal resorptions, reduced fetal weights, altered tissue concentrations of fetal iron and copper, and reduced growth in the offspring.
Animal studies suggest that exposure to very high levels of dietary zinc is associated with reduced fetal weight, alopecia, decreased hematocrit, and copper deficiency in offspring. For example, second generation mice exposed to zinc carbonate during gestation and lactation (260

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mg/kg/day in the maternal diet), and then continued on that diet for 8 weeks, had reduced body weight, alopecia, and signs of copper deficiency (e.g., lowered hematocrit and occasional achromotrichia [loss of hair colour]. Similarly, mink kits from dams that ingested a time-weighted-average dose of 20.8 mg zinc/kg/day as zinc sulfate also had alopecia and achromotrichia. It is likely that the alopecia resulted from zinc-induced copper deficiency, which is known to cause alopecia in monkeys. However, no adverse effects were observed in parental mice or mink. No effects on reproduction were reported in rats exposed to 50 mg zinc/kg/day as zinc carbonate; however, increased stillbirths were observed in rats exposed to 250 mg zinc/kg/day.
Welding or flame cutting of metals with zinc or zinc dust coatings may result in inhalation of zinc oxide fume; high concentrations of zinc oxide fume may result in "metal fume fever"; also known as "brass chills", an industrial disease of short duration. [I.L.O] Symptoms include malaise, fever, weakness, nausea and may appear quickly if operations occur in enclosed or poorly ventilated areas.
Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.
Metallic dusts generated by the industrial process give rise to a number of potential health problems. The larger particles, above 5 micron, are nose and throat irritants. Smaller particles however, may cause lung deterioration. Particles of less than 1.5 micron can be trapped in the lungs and, dependent on the nature of the particle, may give rise to further serious health consequences.
Metals are widely distributed in the environment and are not biodegradable. Biologically, many metals are essential to living systems and are involved in a variety of cellular, physiological, and structural functions. They often are cofactors of enzymes, and play a role in transcriptional control, muscle contraction, nerve transmission, blood clotting, and oxygen transport and delivery. Although all metals are potentially toxic at some level, some are highly toxic at relatively low levels. Moreover, in some cases the same metal can be essential at low levels and toxic at higher levels, or it may be toxic via one route of entry but not another. Toxic effects of some metals are associated with disruption of functions of essential metals. Metals may have a range of effects, including cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenicity, and genotoxicity. Biological half lives of metals vary greatly, from hours to years. Furthermore, the half life of a given metal varies in different tissues. Lead has a half life of 14 days in soft tissues and 20 years in bone.
In considering how to evaluate the toxicity of metals of potential concern, a number of aspects of metal toxicity should be kept in mind:
Different species vary in their responses to different metals; in some cases, humans are more sensitive than rodents. Thus, there is a need for broad-based testing of metals;

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

Hornady Centerfire Rifle and Pistol Ammunition	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

copper	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; 0.733 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; 0.7 mg/kg[2]	

lead	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation(Rat) LC50; >5.05 mg/l4h[1]	
	Oral (Rat) LD50; >2000 mg/kg[1]	

nitrocellulose	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; >5000 mg/kg[2]	Not Available

zinc	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1130 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

aluminium	**TOXICITY**	**IRRITATION**
	Inhalation(Rat) LC50; >2.3 mg/l4h[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >2000 mg/kg[1]	Skin: no adverse effect observed (not irritating)[1]

antimony	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >8000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >5.2 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 7000 mg/kg[2]	

nitroglycerin	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >9560 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 105 mg/kg[2]	Skin: adverse effect observed (irritating)[1]

dibutyl phthalate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >=15.68 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; 8000 mg/kg[2]	

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antimony trisulfide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation(Rat) LC50; >5.04 mg/l4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50; >2000 mg/kg[1]	
barium nitrate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye (rabbit):100 mg/24h - moderate
	Oral (Rat) LD50; >50<300 mg/kg[1]	Skin (rabbit): 500 mg/24h - mild
2,4-dinitrotoluene	**TOXICITY**	**IRRITATION**
	dermal (guinea pig) LD50: >1000 mg/kg[2]	Skin (rabbit): 500 mg/24h - mild
	Oral (Rat) LD50; 268 mg/kg[2]	
diphenylamine	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral (Guinea) LD50; 300 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
lead styphnate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Inhalation(Rat) LC50; >5.05 mg/l4h[1]	
	Oral (Rat) LD50; >2000 mg/kg[1]	
nickel	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 5000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
pentaerythritol tetranitrate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 1660 mg/kg[2]	Not Available
tetrazene	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

COPPER	WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever. for copper and its compounds (typically copper chloride): **Acute toxicity:** There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs. No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation. **Repeat dose toxicity:** In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride. **Genotoxicity:** An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen. **Carcinogenicity:** there was insufficient information to evaluate the carcinogenic activity of copper monochloride. Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).
LEAD	WARNING: Lead is a cumulative poison and has the potential to cause abortion and intellectual impairment to unborn children of pregnant workers.

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NITROGLYCERIN	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Substance has been investigated as a tumorigen, mutagen and reproductive effector. Equivocal tumorigen by RTECS criteria. Reproductive effector in rats.
DIBUTYL PHTHALATE	For dibutyl phthalate (DBP): In studies on rats, DBP is absorbed through the skin, although in *in vitro* studies human skin has been found to be less permeable than rat skin to this compound. Studies in laboratory animals indicate that DBP is rapidly absorbed from the gastrointestinal tract, distributed primarily to the liver and kidneys of rats and excreted in urine as metabolites following oral or intravenous administration. Following inhalation, it was consistently detected at low concentrations in the brain. Available data indicate that in rats, following ingestion, DBP is metabolised by nonspecific esterases mainly in the small intestine to yield mono- *n*-butyl phthalate (MBP) with limited subsequent biochemical oxidation of the alkyl side chain of MBP. MBP is stable and resistant to hydrolysis of the second ester group. Accumulation has not been observed in any organ. The profile of effects following exposure to DBP is similar to that of other phthalate esters, which, in susceptible species, can induce hepatomegaly, increased numbers of hepatic peroxisomes, foetotoxicity, teratogenicity and testicular damage. **Acute toxicity:** The acute toxicity of DBP in rats and mice is low. Signs of acute toxicity in laboratory animals include depression of activity, laboured breathing and lack of coordination. In a case of accidental poisoning of a worker who ingested approximately 10 grams of DBP, recovery was gradual within two weeks and complete after 1 month. On the basis of limited available data in animal species, DBP appears to have little potential to irritate skin or eyes or to induce sensitization. In humans, a few cases of sensitization after exposure to DBP have been reported, although this was not confirmed in controlled studies of larger numbers of individuals reported only in secondary accounts **Repeat dose toxicity:** In short-term repeated-dose toxicity studies, effects at lowest levels in rats after oral administration for 5 to 21 days included peroxisome proliferation and hepatomegaly at doses of 420 mg/kg body weight per day or more. In longer-term studies, the effects in rats observed following ingestion of DBP for periods up to 7 months included reduced rate of weight gain at doses of 250 mg/kg body weight per day or more. Increase in relative liver weight has been observed at doses of 120 mg/kg body weight or more. Peroxisomal proliferation with increased peroxisomal enzyme activity has been observed at doses of 279 mg/kg body weight per day or more. Necrotic hepatic changes in Wistar rats have been reported at doses of 250 mg/kg body weight per day or more but not in F-344 or Sprague-Dawley rats exposed to up to 2500 mg/kg body weight per day. Alteration in testicular enzymes and degeneration of testicular germinal cells of rats have been observed at doses of 250 and 571 mg/kg body weight per day. There are considerable species differences in effects on the testes following exposure to DBP, minimal effects being observed in mice and hamsters at doses as high as 2000 mg/kg body weight per day. In mice, effects on body and organ weights and histological alterations in the liver indicative of metabolic stress have been reported in a recent subchronic bioassay, for which the no-observed-effect-level (NOEL) was 353 mg/kg body weight per day. **Developmental toxicity:** . In a continuous breeding protocol, which included cross-over mating and offspring assessment phases, rats were exposed to 0, 1000, 5000 or 10 000 mg DBP/kg in the diet (equivalent to 0, 66, 320 and 651 mg/kg body weight per day). In the first generation the reduction in pup weight in the mid-dose group, in the absence of any adverse effect on maternal weight, could be regarded as a developmental toxicity effect. There was also a significant reduction of live litter numbers at all three dose levels. The effects in the second generation were more severe, with reduced pup weight in all groups including the low-dose group, structural defects (such as prepucial/ penile malformations, seminiferous tubule degeneration, and absence or underdevelopment of the epididymides) in the mid- and high-dose groups, and severe effects on spermatogenesis in the high-dose group that were not seen in the parent animals. These results suggest that the adverse effects of DBP are more marked in animals exposed during development and maturation than in animals exposed as adults only. No clear NOEL was established in this study. The lowest-observed- adverse-effect-level (LOAEL) was considered to be 66 mg/kg body weight per day. The available studies show that DBP generally induces foetotoxic effects in the absence of maternal toxicity. Available data also indicate that DBP is teratogenic at high doses and that susceptibility to teratogenesis varies with developmental stage and period of administration. In mice, DBP caused dose-dependent increases in the number of resorptions and dead fetuses at oral doses of 400 mg/kg body weight per day or more. Dose-dependent decreases in fetal weights and number of viable litters were also observed in mice at these doses. Adequate carcinogenesis bioassays for DBP have not been conducted. The weight of the available evidence indicates that DBP is not genotoxic. The material may produce peroxisome proliferation. Peroxisomes are single, membrane limited, cytoplasmic organelles that are found in the cells of animals, plants, fungi and protozoa. Peroxisome proliferators include certain hypolipidaemic drugs, phthalate ester plasticisers, industrial solvents, herbicides, food flavours, leukotriene D4 antagonists and hormones. Numerous studies in rats and mice have demonstrated the hepatocarcinogenic effects of peroxisome proliferators, and these compounds have been unequivocally established as carcinogens. However it is generally conceded that compounds inducing proliferation in rats and mice have little, if any, effect on human liver except at very high doses or extreme conditions of exposure. **Transitional Phthalate Esters:** produced from alcohols with straight-chain carbon backbones of C4 to C6. This subcategory also includes a phthalate produced from benzyl alcohol as one ester group with the second ester composed of an alkyl group with a C5 carbon backbone and butyrate group. Phthalate esters containing >10% C4 to C6 molecules were conservatively included in this subcategory. Branched C7 and C8 isomers (di-iso-heptyl, di-iso-octyl and diethylhexyl phthalates) in contrast to linear dihexyl and dioctyl phthalate are members of this family. Transitional phthalates have varied uses, but are largely used as plasticisers for PVC. Physicochemical properties also vary in that the lower molecular weight transitional phthalates are more water-soluble than higher molecular weight transitional phthalates, but none would be characterised as highly water soluble. Transitional phthalates have lower water solubility than the low molecular weight phthalates and except for butylbenzyl phthalate (BBP), existing data suggest they do not exhibit acute or chronic aquatic toxicity. What distinguishes some of the transitional phthalates from others is their greater mammalian toxicity potential, particularly with regard to reproductive and developmental effects, compared to either the low or high molecular weight phthalate subcategories **Acute Toxicity.** The available data on phthalates spanning the carbon range from C4 to C6 indicate that phthalate esters in the transitional subcategory are minimally toxic by acute oral and dermal administration. The oral LD50 value for BBP exceeds 2 g/ kg, and for materials with higher molecular weights, the LD50 values exceed the maximum amounts which can be administered to the animals in a manner consistent with the principles of responsible animal use. One member of this subcategory, diethylhexyl phthalate (DEHP), has been tested for acute inhalation toxicity. It did not cause an effect at the highest concentration tested. Further, considering the low volatility of these substances, inhalation exposure at toxicologically significant levels is not anticipated. **Repeated Dose Toxicity.** Several substances in the C4 to C6 range, including BBP, have been tested for repeated dose toxicity in studies ranging from 3 weeks to 2 years . The principal effects found in these studies were those associated with peroxisome proliferation including liver enlargement and induction of peroxisomal enzymes. As shown in a comparative study of liver effects, the strongest inducers of peroxisome proliferation are diisononyl phthalate (DINP) and di-iso-decyl phthalate (DIDP) with substances of shorter chain length (e.g., BBP) showing much less pronounced effects. Thus it is reasonable to conclude that other members of this subcategory would show effects similar to BBP and less pronounced than DINP or DIDP. It should also be noted that the relevance of these findings to human health is, at best, questionable. It has been shown that these effects are mediated through the peroxisome proliferation-activated receptor alpha (PPARa) and that levels of PPARa are much higher in rodents than they are in humans. Thus one would expect humans to be substantially less responsive than rodents to peroxisome proliferating agents. Empirical evidence that this is true is provided by studies in primates in which repeated administration of DINP had no effects on liver, kidney or testicular parameters. Several of the substances in the transitional phthalate esters subcategory, however, have been shown to produce testicular atrophy when given to juvenile rats at high levels. Testicular atrophy has been associated with BBP and other substances with C4 to C6 linear carbon chains. However, molecules with fewer than 4 or more than 6 carbons did not produce testicular atrophy in these studies. Although the relevance of these data are uncertain, as the testes is not a target organ for diethylhexyl phthalate (DEHP) in primates, these data do provide one of the distinguishing toxicological characteristics of this subcategory and are one of the underlying reasons supporting the differentiation of phthalate esters on the basis of length of the linear region of the carbon chain. **Genetic Toxicity (Salmonella)**. A number of the substances in this subcategory including the reference substance BBP has been assessed in the Salmonella and mouse lymphoma assays. All of these substances were inactive in these assays. **Chromosomal Aberrations**. BBP and dihexyl phthalate (DHP) were inactive in micronucleus assays in mice. DEHP was inactive in a

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	cytogenetics assay in rat bone marrow. Diisoheptyl phthalate was inactive in CHO cells, in vitro.. **Reproductive toxicity:** A series of studies assessed the structure-activity relationship of the effects of phthalate esters on fertility using a continuous breeding protocol . The test substances included in these studies were diethyl-, dipropyl-, dibutyl-, dipentyl-, d-n-hexyl-, di-2(ethylhexyl)-, and di-n-octyl phthalates. The most profound effects were on fertility (i.e., number of females delivering/ number mated) and number of live births. The substance showing the greatest activity was DEHP which produced effects at dietary levels of 0.1 % with a no effect level of 0.01 %. The next most active compounds were di-n-hexyl- and di-n-pentyl phthalate which showed effects in the range of 0.3 to 0.5 %; no effect levels were not experimentally defined. Dipropyl phthalate had an effect on live birth index at 2.5 % but produced no effects at 1.25 %. Diethyl phthalate and di-n-octyl phthalate were inactive at the highest levels tested, 2.5 % and 5.0 %, respectively. These data demonstrated that molecules with linear alkyl chains of 4 to **6** carbons profoundly affect fertility in rodents, with DEHP being the most active. Molecules with longer or shorter side chains are essentially inactive in these assays. These data were also a basis for the separation of phthalates into three categories based on length of side chain. In addition to these data there are reproductive toxicity studies on BBP and DEHP . A 2-generation reproductive study was conducted in rats in which BBP was administered via the diet. Parental effects were limited to changes in body weight, weight gain, and increased absolute and relative liver weights. In the F1 parents, treatment with BBP affected mating and fertility indices and sperm number and motility. The F1 male offspring exhibited shortened anogenital distance, delayed acquisition of puberty and retention of nipples and areolae as well as reproductive effects. The NOAEL of the study was reported to be 3750 mg/ kg for reproductive effects. However, for male F1 and F2 offspring, the NOEL for reproductive effects was reported to be 50 mg/ kg based on reductions in anogenital distance. These studies along with previous data provide a good basis to assess the reproductive effects of C4 to C6 phthalate esters. Although several substances (diheptyl, heptyl nonyl, heptyl undecyl) have ester side chain constituents that predominately fall in the high molecular weight subcategory, these substances are conservatively assumed to exhibit reproductive effects similar to other transitional phthalates . **Developmental toxicity:** There have been extensive studies of the developmental toxicity of BBP and DEHP. These substances produce structural malformations and also affect male reproductive development. No effect levels are in the range of 50 to 300 mg/ kg bw/ day. There is also an unpublished developmental toxicity study of di-isoheptyl phthalate (DIHP). The results of these studies are broadly consistent with the structure-activity relationships previously described, i.e., that phthalate esters with linear carbon chains of C4 to C6 carbons produce much more profound effects that either shorter or longer molecules. Phthalate esters with >10% C4 to C6 isomers were conservatively placed in the transitional subcategory. This conclusion is supported by developmental test data on "711P*" (which showed structural malformations in rats at 1000 mg/ kg/ day with a NOAEL of 200 mg/ kg/ day ."711P" is an equal composition mixture of six phthalate esters consisting of linear and methyl-branched C7, C9, and C11 ester side chains. This test substance is considered by EPA under the following CAS Numbers.: 68515-44-6 (di C7), 68515-45-7 (di C9), 3648-20-2 (di C1 I), 111381-89-6 (C7, C9), 111381-90-9 (C7, C11), and 111381-91-0 (C9, C11). The overall content of C4 to C6 isomers in "71 1P" is approximately 10%, based on the contribution from methyl-branched C7 isomers e.g., di C7 (30% C4-C6); C7, C9 (15% C4-C6); and C7, C11 (15 % C4-C6). Test data on 711P were used selectively as read-across data to the C7-containing substances in the mixture, based on the C4 to C6 content of each substance in the mixture.
ANTIMONY TRISULFIDE	The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. * REACH Dossier
BARIUM NITRATE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
2,4-DINITROTOLUENE	for dinitrotoluene (syn: dinitromethylbenzene; DNT) In humans, heavy DNT exposure causes signs of methaemoglobinaemia, which are reversible 2-3 days after removal from exposure. Signs of disturbances in liver function and exposure-dependent nephrotoxic effects directed to the tubular system were additionally found in exposed workers. Single findings in studies without reliable exposure data and/or only small numbers of significantly exposed workers indicating increased incidences of hepatobiliary or urothelial cancer in occupationally DNT exposed workers do not permit a conclusion on the carcinogenicity of DNT in humans. Preliminary observations pointing to an increased risk of ischemic heart disease or to an adverse effect on the human male reproductive system could not be confirmed by further studies In humans dinitrotoluene (DNT, technical grade) is absorbed following dermal and inhalative exposure and is rapidly metabolized and excreted in urine. **Acute toxicity**: There are no acute inhalation studies on technical grade DNT and the 2,4-isomer available. LC50 of the 2,6-isomer, is reported to be 0.36 mg/l, however, this isomer accounts only for about 18% of technical grade DNT. No acute dermal studies on technical grade DNT and the 2,6-isomer are available. The acute dermal toxicity of 2,4-DNT, the main component of technical grade DNT, is relatively low with an LD50 greater than 2500 mg/kg bw in rats. Technical grade DNT is moderately toxic following oral administration to rats, with LD50 values of 268 to 660 mg/kg bw reported. After a 24-hour occlusive application DNT is not irritating to the skin (interior side of the ear) of rabbits. Although DNT has been reported to induce slight irritation to the eye of one rabbit, the effect is reversible within 7 days, and therefore DNT is not considered to be an eye irritant in humans. There are no data available to evaluate the sensitising potential of technical grade DNT. The 2,4-DNT isomer showed no sensitising properties in a guinea pig maximization test, whereas 2,6-DNT gave a mild positive response. Patch tests or photo-patch tests in 10 or 5 healthy humans showed no allergic potential of DNT (unspecified isomer), whereas a single case of positive photo-patch test reaction was reported for a worker with skin problems. There are no inhalative or dermal repeated dose studies on technical grade DNT or on the 2,4-/2,6-DNT isomers available. **Repeat dose toxicity:** Chronic feeding of technical grade DNT to rats led to haematological changes (especially methaemoglobinaemia), and toxicity to liver, kidney, adrenal glands and testes in rats. At the lowest administered dose of 3.5 mg/kg bw/day signs of hepatotoxicity became obvious. No NOAEL can be derived for repeated dose toxicity. **Genotoxicity:** Technical grade DNT is mutagenic in bacterial test systems in the presence and absence of metabolic activation, but it shows no mutagenic or genotoxic activity in mammalian cells *in vitro*. Technical grade DNT shows no mutagenic activity in the mouse bone marrow micronucleus assay and in mouse dominant lethal and spot tests. However, a distinct activity of DNT to induce DNA repair in the liver of rats is reported. Additionally, DNA binding properties in various rat organs, mainly rat liver were demonstrated for 2,4-DNT and 2,6-DNT isomers. Gut flora may play an important role in activation of DNT to reactive metabolites. Overall, technical grade DNT shows the potential to induce genotoxic changes *in vivo*. **Carcinogenicity**: Technical grade DNT shows hepatocarcinogenic properties in rats. In a long-term feeding study liver tumours were dose dependently induced in male rats from the lowest administered dose (3.5 mg/kg bw/day) and in female rats from 14 mg/kg bw/day. In an initiation-promotion liver foci assay DNT showed tumor promoting activity, however, only weak initiating properties. The pure 2,4- and 2,6-DNT isomers also induced liver tumours in rats. Additionally, the 2,4-isomer was shown to induce tumour formation in the renal tubular epithelium of male mice. **Reproductive toxicity:** After long-term feeding of technical grade DNT to rats (104 weeks) increased incidences of abnormally small testes and increased ovary weights were observed at 14 mg/kg bw/day. A daily dose of 35 mg/kg bw DNT led to testicular degeneration and hypospermatogenesis after 52 weeks of exposure. The NOAEL for changes on reproductive organs was 3.5 mg/kg bw/day in this study. Technical grade DNT did not negatively affect fertility in dominant lethal assays. Overall, impairment of male rat fertility after chronic exposure of toxic doses cannot be excluded. **Developmental toxicity:** In pregnant rats, administration of technical grade DNT by gavage on gestation days 7-20 did not induce teratogenic/developmental effects even at dose levels, which produced significant maternal toxicity. The NOAEL for teratogenic/developmental toxicity can be determined to be 150 mg/kg bw/day.

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DIPHENYLAMINE	For substituted diphenylamines: Based upon reviewed data the physicochemical and toxicological properties of the substituted diphenylamines are similar and follow a regular pattern as a result of that structural similarity. Because of their powerful antioxidant properties, Substituted Diphenylamines, along with their common starting material, Diphenylamine, are regulated for use in several food-contact applications by the Food and Drug Administration as Indirect Food Additives under the following sections of the Code of Federal Regulations (CFR): Heating may generate vapors which can irritate the eyes and respiratory passages. Drying of skin and mucous membranes leading to irritation may be possible from prolonged or repeated contact. Overexposure to vapors from heating the product may cause and/or skin irritation and respiratory tract irritation with symptoms such as, but not limited to, dizziness and flu-like symptoms **Acute toxicity:** As a group these materials do not produce significant acute toxicity in mammals. All show a slight to very low order of toxicity following oral administration, with LD50 values ranging from >5000 to > 34,000 mg/kg. Overall, the acute dermal LD50 for these materials was greater than the 2000 mg/kg limit dose indicating a very low order of toxicity. **Mammalian Toxicology - Mutagenicity**. Data from bacterial reverse mutation assays, in vitro and in vivo chromosome aberration studies, as well as additional supporting in vitro and in vivo genetic toxicity studies indicate a low concern for mutagenicity either for aryl or alkyl substituted materials. Similarly, the data for a mixed aryl/alkyl substituted molecule also indicates a lack of mutagenicity. **Acute toxicity:** Diphenylamine and its substituted derivatives all show a slight to moderate order of toxicity following oral administration, with LD50 values ranging from >500 to > 34,000 mg/kg. Overall, the acute dermal LD50 for these materials was greater than the 2000 mg/kg limit dose indicating a very low order of toxicity. **Mutagenicity:** Of five substituted diphenylamines tested, there was one weakly positive mutagenic response with in the bacterial mutagenicity test, with diphenylamine (122-39-4). Overall weight of evidence for this material, as well as the category indicates a negative evaluation for bacterial mutagenicity. Substituted diphenylamines have been tested for mutagenicity in tests for gene mutations and chromosomal aberrations. The assays included point mutations in bacterial cells, *in vitro* chromosomal aberrations in mammalian cells, and *in vivo* chromosomal aberrations. With one exception, the data consistently demonstrate no evidence of genotoxicity for this category of materials. This suggests that all members of the category lack genotoxicity due to their similarity in chemical structures and physicochemical properties **Repeat Dose Toxicity**: Diphenylamine (122-39-4) was tested in a 28 day oral study with rats. A NOAEL of 111 mg/kg/day was identified. Diphenylamine is not only the common precursor for the materials of this category, but also theoretically the most toxic of the class since it is the smallest member of the class. The addition of alkyl groups onto the diphenylamine molecule results in even lower water solubility and, therefore, becomes less bioavailable. Diphenylamine, styrenated (68442-68-2) was tested in a 28day oral gavage study in rats. A NOAEL of 100 mg/kg/day was identified. Diphenylamine styrenated was tested in a 28-day gavage study in rats; 100 mg/kg/day was selected as the NOAEL. Diphenylamine-, reaction products with styrene and 2,4,4-trimethylpentene (68921-45-9) was tested in a 64 week rat dietary study; a LOEL of 2500 ppm was identified. . **Reproductive and Developmental Toxicity:** Diphenylamine was administered in feed at 0.1, 0.25 or 0.5% (ca. 67, 167 or 333 mg/kg/day) to rats in a two-generation reproductive toxicity study. In general, the average size of the litters decreased as the concentration of dietary diphenylamine increased. A NOEL was not established. A developmental study was also conducted with diphenylamine in rabbits. The test article was administered by gavage at dose levels of 0, 33, 100 and 300 mg/kg/day for gestation days 7-19. The test article produced minimal effects (decreased food consumption and mean body weight) to maternal rats at 300 mg/kg during pregnancy; there were no other signs of maternal toxicity. NOAEL for maternal toxicity was established at 100 mg/kg/day. The NOAEL for teratogenicity/developmental effects was greater than 300 mg/kg/day. ADI: 0.02 mg/kg/day NOEL: 1.5 mg/kg/day
NICKEL	Oral (rat) TDLo: 500 mg/kg/5D-I Inhalation (rat) TCLo: 0.1 mg/m3/24H/17W-C Tenth Annual Report on Carcinogens: Substance anticipated to be Carcinogen [*National Toxicology Program: U.S. Dep. of Health & Human Services 2002*]
NITROCELLULOSE & ZINC & ALUMINIUM & LEAD STYPHNATE & TETRAZENE	No significant acute toxicological data identified in literature search.
ZINC & NITROGLYCERIN & BARIUM NITRATE & 2,4-DINITROTOLUENE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
2,4-DINITROTOLUENE & NICKEL	**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
DIPHENYLAMINE & TETRAZENE	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.
LEAD STYPHNATE & NICKEL	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

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

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Hornady Centerfire Rifle and Pistol Ammunition

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

copper

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	24h	Algae or other aquatic plants	<0.001mg/L	4
EC50	72h	Algae or other aquatic plants	0.011-0.017mg/L	4
EC50	48h	Crustacea	<0.001mg/L	4
EC50	96h	Algae or other aquatic plants	0.03-0.058mg/l	4
LC50	96h	Fish	0.005-0.06mg/l	4

lead

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	Not Available	Crustacea	0.051mg/L	5
EC50	72h	Algae or other aquatic plants	1.191mg/L	4
EC50	96h	Algae or other aquatic plants	0.282-0.864mg/l	4
LC50	96h	Fish	1.17mg/l	4

nitrocellulose

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

zinc

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	72h	Algae or other aquatic plants	0.005mg/l	4
EC50	72h	Algae or other aquatic plants	0.005mg/l	4
EC50	48h	Crustacea	1.4mg/l	2
EC50	96h	Algae or other aquatic plants	0.264-0.881mg/l	4
LC50	96h	Fish	0.16mg/L	4

aluminium

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	0.078-0.108mg/l	2
NOEC(ECx)	48h	Crustacea	>100mg/l	1
EC50	72h	Algae or other aquatic plants	0.2mg/l	2
EC50	48h	Crustacea	1.5mg/l	2
EC50	96h	Algae or other aquatic plants	0.024mg/l	2

antimony

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	2160h	Algae or other aquatic plants	0.032mg/l	2
EC50	72h	Algae or other aquatic plants	>2.4mg/l	2
EC50	48h	Crustacea	423.45mg/l	2
EC50	96h	Algae or other aquatic plants	0.61mg/l	2
LC50	96h	Fish	0.93mg/l	2

nitroglycerin

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	1440h	Fish	0.03mg/l	2
EC50	48h	Crustacea	4655mg/l	1
EC50	96h	Algae or other aquatic plants	0.1-1.3mg/l	4
LC50	96h	Fish	1.69-2.14mg/l	4

dibutyl phthalate

Endpoint	Test Duration (hr)	Species	Value	Source
ErC50	72h	Algae or other aquatic plants	1.2mg/l	1
BCF	1344h	Fish	3.1-21.2	7
NOEC(ECx)	72h	Algae or other aquatic plants	0.5mg/l	1
EC50	72h	Algae or other aquatic plants	1.2mg/l	1
LC50	96h	Fish	0.28-0.44mg/l	4
EC50	48h	Crustacea	3.4mg/l	1
EC50	96h	Algae or other aquatic plants	0.004-0.2mg/l	1

antimony trisulfide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>2.4mg/l	2
NOEC(ECx)	2160h	Algae or other aquatic plants	0.032mg/l	2
EC50	48h	Crustacea	423.45mg/l	2
EC50	96h	Algae or other aquatic plants	0.61mg/l	2

Continued...

	LC50	96h	Fish	0.93mg/l	2

barium nitrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>1.15mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>=1.15mg/l	2
EC50	48h	Crustacea	>=16<=18mg/l	2
LC50	96h	Fish	>3.5mg/l	2

2,4-dinitrotoluene

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	504h	Crustacea	0.02mg/L	5
EC50	48h	Crustacea	22mg/l	4
EC50	96h	Algae or other aquatic plants	1.912-3.497mg/L	4
LC50	96h	Fish	23.7mg/l	1

diphenylamine

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.048mg/l	1
EC50(ECx)	72h	Algae or other aquatic plants	0.048mg/l	1
BCF	1344h	Fish	51-253	7
EC50	48h	Crustacea	0.27-0.36mg/l	4
LC50	96h	Fish	2.088-3.596mg/L	4

lead styphnate

Endpoint	Test Duration (hr)	Species	Value	Source
EC10(ECx)	48h	Crustacea	0.5mg/l	2
EC50	48h	Crustacea	7.02mg/l	2

nickel

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	72h	Algae or other aquatic plants	0.18mg/l	1
EC50	72h	Algae or other aquatic plants	0.18mg/l	1
EC50	48h	Crustacea	>100mg/l	1
EC50	96h	Algae or other aquatic plants	0.36mg/l	2
LC50	96h	Fish	0.168mg/L	4

pentaerythritol tetranitrate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	24h	Crustacea	>49mg/l	4
EC50	48h	Crustacea	>49mg/l	4
LC50	96h	Fish	~926mg/l	2

tetrazene

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

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

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
nitroglycerin	LOW (Half-life = 14 days)	LOW (Half-life = 0.73 days)
dibutyl phthalate	LOW (Half-life = 23 days)	LOW (Half-life = 3.08 days)
2,4-dinitrotoluene	HIGH (Half-life = 360 days)	MEDIUM (Half-life = 118.33 days)
diphenylamine	LOW (Half-life = 56 days)	Not Available
tetrazene	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
dibutyl phthalate	LOW (BCF = 176)
2,4-dinitrotoluene	HIGH (BCF = 2507)
diphenylamine	LOW (BCF = 253)
tetrazene	LOW (LogKOW = -2.0204)

Mobility in soil

Ingredient	Mobility

Continued...

Ingredient	Mobility
dibutyl phthalate	LOW (KOC = 1460)
2,4-dinitrotoluene	LOW (KOC = 363.8)
diphenylamine	LOW (KOC = 1887)
tetrazene	LOW (KOC = 238.4)

SECTION 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: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and SDS and observe all notices pertaining to the product. ▸ Explosives which are surplus, deteriorated or considered unsafe for transport, storage or use shall be destroyed and the statutory authorities shall be notified. ▸ Explosives must not be thrown away, buried, discarded or placed with garbage. ▸ This material may be disposed of by burning or detonation but the operation must be performed under the control of a person competent in the destruction of explosives. Disposal by detonation: ▸ The explosives to be destroyed must be placed in direct contact with fresh priming charge in a hole which is at least 0.6 metre deep and then adequately stemmed. ▸ No detonators shall be inserted into defective explosives. ▸ Personnel must be evacuated to a safe distance prior to initiation/firing of the charge. Disposal by burning: ▸ Make a sawdust bed or trail adequate for the quantity of explosives to be burned, approximately 400 mm wide and 40 mm deep, upon which the explosive will be laid. ▸ If sawdust is not available, newspaper may be used. ▸ Normal precautions shall be taken to avoid the spread of fire. ▸ Individual trails should not be closer together than 600 mm and should contain not more than 12 kg of explosive. ▸ Trails should be side by side, NOT in-line, and not more than four should be set up at one time. ▸ Remove any explosive that is not to be burnt to a distance of at least 300 metre. ▸ Sufficient diesel oil (never petrol or other highly flammable liquid) should be used to thoroughly wet the sawdust (or paper) at least 4 litre per trail is recommended. ▸ Light the trail from a long, rolled paper wick which should be placed downwind and in contact with the end 1m of trail that is not covered with explosive. The wind should blow so that the flame from the wick (and later from the burning explosive) will blow away from the unburned explosive as detonation is more likely to occur if the explosive is preheated by the flame. ▸ If plastic igniter cord (slow) is available, its use for lighting is recommended instead of paper. One end should be coiled into the sawdust or under the paper and the other end lit from a minimum distance of 7m from the trail. ▸ Retire at least 300m or to a safe place. ▸ **DO NOT** return to the site for at least 30 minutes after the burning has apparently finished. ▸ If the fire goes out do not approach for at least 15 minutes after all trace of fire has gone. ▸ **DO NOT** add more diesel oil unless certain that the flame is completely extinguished. [DYNO] ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	1YE

Land transport (ADG)

UN number	0012
UN proper shipping name	CARTRIDGES FOR WEAPONS, INERT PROJECTILE or CARTRIDGES, SMALL ARMS
Transport hazard class(es)	Class: 1.4S Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Environmentally hazardous

Continued...

Special precautions for user	Special provisions: 364 Limited quantity: 5 kg

Air transport (ICAO-IATA / DGR)

UN number	0012
UN proper shipping name	Cartridges, small arms
Transport hazard class(es)	ICAO/IATA Class: 1.4S ICAO / IATA Subrisk: Not Applicable ERG Code: 3L
Packing group	Not Applicable
Environmental hazard	Environmentally hazardous
Special precautions for user	Special provisions: A802 Cargo Only Packing Instructions: 130 Cargo Only Maximum Qty / Pack: 100 kg Passenger and Cargo Packing Instructions: 130 Passenger and Cargo Maximum Qty / Pack: 25 kg Passenger and Cargo Limited Quantity Packing Instructions: Forbidden Passenger and Cargo Limited Maximum Qty / Pack: Forbidden

Sea transport (IMDG-Code / GGVSee)

UN number	0012
UN proper shipping name	CARTRIDGES FOR WEAPONS, INERT PROJECTILE or CARTRIDGES, SMALL ARMS
Transport hazard class(es)	IMDG Class: 1.4S IMDG Subrisk: Not Applicable
Packing group	Not Applicable
Environmental hazard	Marine Pollutant
Special precautions for user	EMS Number: F-B, S-X Special provisions: 364 Limited Quantities: 5 kg

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
copper	Not Available
lead	Not Available
nitrocellulose	Not Available
zinc	Not Available
aluminium	Not Available
antimony	Not Available
nitroglycerin	Not Available
dibutyl phthalate	Not Available
antimony trisulfide	Not Available
barium nitrate	Not Available
2,4-dinitrotoluene	Not Available
diphenylamine	Not Available
lead styphnate	Not Available
nickel	Not Available
pentaerythritol tetranitrate	Not Available
tetrazene	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
copper	Not Available
lead	Not Available
nitrocellulose	Not Available

Continued...

Product name	Ship Type
zinc	Not Available
aluminium	Not Available
antimony	Not Available
nitroglycerin	Not Available
dibutyl phthalate	Not Available
antimony trisulfide	Not Available
barium nitrate	Not Available
2,4-dinitrotoluene	Not Available
diphenylamine	Not Available
lead styphnate	Not Available
nickel	Not Available
pentaerythritol tetranitrate	Not Available
tetrazene	Not Available

SECTION 15 Regulatory information

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

copper is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

lead is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

nitrocellulose is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

zinc is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

aluminium is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

antimony is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

nitroglycerin is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
FEI Equine Prohibited Substances List - Controlled Medication
FEI Equine Prohibited Substances List (EPSL)

dibutyl phthalate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

antimony trisulfide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

barium nitrate is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2A: Probably carcinogenic to humans

2,4-dinitrotoluene is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

diphenylamine is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

lead styphnate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

nickel is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

pentaerythritol tetranitrate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
FEI Equine Prohibited Substances List - Banned Substances
FEI Equine Prohibited Substances List (EPSL)

tetrazene is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (tetrazene)
Canada - NDSL	No (copper; lead; nitrocellulose; zinc; aluminium; antimony; nitroglycerin; dibutyl phthalate; antimony trisulfide; barium nitrate; 2,4-dinitrotoluene; diphenylamine; lead styphnate; nickel; pentaerythritol tetranitrate)
China - IECSC	No (lead styphnate; tetrazene)
Europe - EINEC / ELINCS / NLP	No (nitrocellulose)
Japan - ENCS	No (copper; lead; zinc; aluminium; antimony; nickel; tetrazene)
Korea - KECI	No (tetrazene)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (lead styphnate; pentaerythritol tetranitrate; tetrazene)
USA - TSCA	Yes
Taiwan - TCSI	No (tetrazene)
Mexico - INSQ	No (tetrazene)
Vietnam - NCI	No (lead styphnate; tetrazene)
Russia - FBEPH	No (lead styphnate; tetrazene)
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	14/03/2022
Initial Date	14/03/2022

SDS Version Summary

Version	Date of Update	Sections Updated

Continued...

Version	Date of Update	Sections Updated
2.1	14/03/2022	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Chronic Health, Classification, Fire Fighter (fire/explosion hazard)

Other information

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

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

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on 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