Jasart Tie Dye Sets

Jasco Pty Limited

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5469-09**
Version No: **3.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **19/05/2021**
Revision Date: **02/02/2026**
Print Date: **29/10/2026**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Jasart Tie Dye Sets
Chemical Name	Not Applicable
Synonyms	Not Available
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	Consumer use. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**Jasco Pty Limited**
Address	1-5 Commercial Road Kingsgrove NSW 2208 Australia
Telephone	+61 2 9807 1555
Fax	Not Available
Website	www.jasco.com.au
Email	sales@jasco.com.au

Emergency telephone number

Association / Organisation	**Australian Poisons Centre**	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone number(s)	**13 11 26 (24/7)**	**+61 1800 951 288 (ID#: 5469-09)**
Other emergency telephone number(s)	**Not Available**	**+61 3 9573 3188**

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Serious Eye Damage/Eye Irritation Category 2A, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 3
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	**Warning**

Hazard statement(s)

H319	Causes serious eye irritation.
H412	Harmful to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P273	Avoid release to the environment.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

Page 1 continued...

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
144-55-8	<70	sodium bicarbonate
23354-52-1	<40	C.I. Reactive Red 194
73049-92-0	<40	copper phthalocyanine, sulfonyl derivatives
6539-67-9	<40	C.I. Reactive Yellow 3
12238-00-5	<30	C.I. Reactive Red 24
12225-26-2	<30	C.I. Reactive Black 8
12677-16-6	<30	C.I. Reactive Blue 74
2580-78-1	<25	C.I. Reactive Blue 19
7647-14-5	<25	sodium chloride
497-19-8	<20	sodium carbonate
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: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
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.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

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]

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Periodic medical surveillance should be carried out on persons in occupations exposed to the manufacture or bulk handling of the product and this should include hepatic function tests and urinalysis examination. [ILO Encyclopaedia]

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substance or mixture

Fire Incompatibility	‣ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use water delivered as a fine spray to control fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ Combustible. ‣ Slight fire hazard when exposed to heat or flame. ‣ Heating may cause expansion or decomposition leading to violent rupture of containers. ‣ On combustion, may emit toxic fumes of carbon monoxide (CO). ‣ May emit acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: ‣ carbon dioxide (CO_2) hydrogen chloride phosgene nitrogen oxides (NOx) sulfur oxides (SOx) metal oxides ‣ other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.
HAZCHEM	Not Applicable

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	‣ Remove all ignition sources. ‣ Clean up all spills immediately. ‣ Avoid breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	Moderate hazard. ‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ No smoking, naked lights or ignition sources. ‣ Increase ventilation. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Absorb remaining product with sand, earth or vermiculite. ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ **DO NOT** allow clothing wet with material to stay in contact with skin ‣ Avoid skin contact, including inhalation.

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	‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ Avoid smoking, naked lights or ignition sources. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ No smoking, naked lights or ignition sources. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Glass container is suitable for laboratory quantities ‣ Polyethylene or polypropylene container. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	‣ Avoid reaction with oxidising agents, bases and strong reducing agents. ‣ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Individual protection measures, such as personal protective equipment	
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. OTHERWISE: ‣ Safety glasses with side 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	No special equipment needed when handling small quantities. **OTHERWISE**: Wear general protective gloves, e.g. light weight rubber gloves.
Body protection	See Other protection below
Other protection	‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

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:
Jasart Tie Dye Sets

Material	CPI
NATURAL RUBBER	A
NITRILE	A

Respiratory protection

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

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator

Continued...

NATURAL+NEOPRENE	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.

up to 10	1000	A-AUS / Class1	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

· 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.
Class P2 particulate filters are used for protection against mechanically and thermally generated particulates or both.
P2 is a respiratory filter rating under various international standards, Filters at least 94% 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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Various coloured liquid.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	6	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Available
Initial boiling point and boiling range (°C)	100	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available

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Solubility in water	Not Available	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial 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 vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Side effects of the inhalation of cobalt and its compounds may include flushing of the face and ringing in the ears (tinnitus). Cobalt inhalation can be lethal in animals if exposure is sufficiently high or prolonged. The acute LC50 for a 30-minute inhalation exposure in rats was 165 mg cobalt/m3 as cobalt hydrocarbonyl. Exposure to 9 mg cobalt/m3 as cobalt hydrocarbonyl for 6 hours/day, 5 days/week for 3 months resulted in 16 deaths out of 75 rats. Death was reported in rats and mice exposed to 19 mg cobalt/m3 (but not 1.9 mg cobalt/m3) as cobalt sulfate over 16 days, but exposure to 11.4 mg cobalt/m3 over 13 weeks was lethal only to mice and not to rats. Exposure to 1.14 mg cobalt/m3 as cobalt sulfate for 104 weeks resulted in no increase in mortality in rats and mice of either sex. Inhalation of stable cobalt by humans and/or animals resulted in respiratory, cardiovascular, hematological, hepatic, renal, endocrine, ocular, and body weight effects. As with exposures in humans, exposures of animals to cobalt-containing aerosols have resulted in pronounced respiratory effects. Animals exposed to aerosols of cobalt oxides and cobalt sulfate developed respiratory effects that varied in severity with exposure level and duration. A single 30-minute exposure of rats to relatively high levels (26-236 mg cobalt/m3 as cobalt hydrocarbonyl) resulted in congestion, edema, and hemorrhage of the lung. Prolonged exposure (3-4 months) of rats and rabbits to mixed cobalt oxides (0.4-9 mg cobalt/m3) resulted in lesions in the alveolar region of the respiratory tract characterised histologically by nodular accumulation of Type II epithelial cells, accumulations of enlarged highly vacuolated macrophages, interstitial inflammation, and fibrosis In at least one instance, the lesions appeared to regress when exposure was terminated . Guinea pigs sensitized to cobalt by repeated dermal application and then exposed to 2.4 mg cobalt/m3 as cobalt chloride showed pulmonary inflammatory changes (altered BAL fluid recovery, increased neutrophils and eosinophils in the recovered BAL fluid) that were different than those in exposed animals not sensitised to cobalt. Decreased lung compliance was found in pigs exposed to 0.1 mg cobalt/m3 as cobalt dust for 3 months. Lifetime exposure of hamsters to 7.9 mg cobalt/m3 as cobalt oxide resulted in emphysema. Necrosis and inflammation of the respiratory tract epithelium (nasal turbinates, larynx, trachea, bronchioles) were reported in rats exposed to 19 mg cobalt/m3 and mice exposed to 1.9 mg cobalt/m3 or greater as cobalt sulfate over 16 days. Exposure of rats and mice to cobalt as cobalt sulfate for 13 weeks resulted in adverse effects on all parts of the respiratory tract, with the larynx being the most sensitive part Symptoms of sodium carbonate inhalation may include coughing, sore throat, and laboured breathing. Severe or continued inhalation exposure may cause pulmonary oedema (lung damage). 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.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.

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	Studies have shown that soluble cobalt compounds are generally more acutely toxic than insoluble cobalt compounds. When expressed in terms of the cobalt ion for the sake of comparison, however, the differences in lethality values from the available studies are within an order of magnitude Animal test indicate an increase in red blood cells (polycythaemia) following the absorption of cobalt salts. [ICI] In toxic doses soluble cobalt salts act locally on the gastro-intestinal tract to produce pain and vomiting. Systemic effects in man include a peculiar vasodilation (flushing) of the face and ears, mild hypotension, rash, tinnitus (ringing in the ears) and nerve deafness. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products] Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products.] Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain is availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper.
Skin Contact	The material produces mild skin irritation; evidence exists, or practical experience predicts, that the material either ‣ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ‣ produces significant, but mild, 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. 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. Contact with concentrated solutions of sodium carbonate may cause tissue damage "soda ulcers" Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature.
Eye	510sodacarb This material causes serious eye irritation. Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Occupational exposure to reactive dyes may result in respiratory sensitisation. Several cases of allergy of the respiratory tract have been attributed to the sensitising effects of certain reactive dyes inhaled in the dust form. The handling of other reactive dyes may also involve a risk of this type if care and cleanliness are compromised. At present there is no recognised test to detect this risk [Sandoz Products, Switzerland] Because of similarities in structure to thalidomide, concerns have been raised about the potential teratogenicity of all phthalimides (the basic building block of phthalocyanine). Animal studies, in part, appear to support this proposition. The phthalimide fungicides are typically teratogens. Captan, for example, is teratogenic, foetotoxic, and/or embryotoxic in a variety of rodent species and has caused stillbirths in dogs. Folpet was teratogenic in rabbits, chicks, and hamsters. Captafol has been embryo-or foetotoxic in experimental animals.Captan, folpet, and captafol have all induced testicular degeneration in laboratory animals. In general, available cohort studies in humans have not reported a significant increase in total mortality as a result of cobalt exposure. Several studies have noted increased mortality rates resulting from lung cancer following occupational exposure to cobalt, either as a mixture of cobalt compounds or as hard metal, a metal alloy with a tungsten carbide and cobalt matrix. Fatal cases of hard metal disease and cardiomyopathy believed to have resulted from occupational cobalt exposure have also been reported. However, in the majority of these and other reported occupational studies, co-exposure to other substances was common, and was unable to be corrected for in the analysis. The effects of chronic occupational exposure to cobalt and cobalt compounds on the respiratory system in humans are well-documented. These effects include respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis and occurred at exposure levels ranging from 0.007 to 0.893 mg cobalt/m3 (exposure from 2 to 17 years). These effects have been observed in workers employed in cobalt refineries, as well as hard metal workers, diamond polishers, and ceramic dish painters (painting with cobalt blue dye). Occupational asthma attributed to the inhalation of cobalt powder has been confirmed following bronchial challenge tests. Chest tightness and chronic bronchitis have been recorded in hard-metal workers exposed to cobalt. Cobalt is known to function as a hapten, resulting in the generation of antibodies against cobalt-protein complexes. Although the minimum exposure level associated with cobalt sensitisation has not been determined, sensitisation has been demonstrated in hard metal workers with work-related asthma who have experienced prolonged occupational exposure (>3 years) to levels ranging from 0.007 to 0.893 mg cobalt/m3. The sensitisation phenomenon includes the production of IgE and IgA antibodies to cobalt. Exposure to inhaled cobalt chloride aerosols can precipitate an asthmatic attack in sensitised individuals believed to be the result of an allergic reaction within the lungs.

Continued...

Allergic dermatitis of an erythematous papular type may also occur following occupational exposure. Dermatitis is a common result of dermal exposure to cobalt in humans that has been verified in a large number of studies. Using patch tests and intradermal injections, it has been demonstrated that the dermatitis is probably caused by an allergic reaction to cobalt. Contact allergy was reported in 22 of 223 (9.9%) nurses who were tested with a patch test of 1.0% cobalt chloride as well as 16 of 79 (20.3%) of examined dentists. Persons with body piercings showed an increased prevalence of allergy to cobalt, with the incidence of contact allergy being proportional to number of piercings
The prevalence of sensitivity to cobalt following exposure to cobalt as a component of metal implants is low, with only 3.8% of patients developing a new sensitivity to cobalt following insertion of the implant
Exposure levels associated with the development of dermatitis have not been identified. It appears that the allergic properties of cobalt result mainly from exposure to the metal itself, rather than a salt, as it has been demonstrated that daily repeated exposure to aqueous cobalt salts did not result in hand eczema in patients known to have cobalt allergy.
Occupational exposure to cobalt in humans has been reported to cause several effects on the nervous system, including memory loss , nerve deafness, and a decreased visual acuity. It should be noted though, that both of the studies reporting on these findings, had small numbers of subjects, and exposure characterization was not reported.
Chronic exposure to cobalt produces polycythaemia (increase in blood haemoglobin), increased production of cells of the bone marrow and thyroid gland, pericardial effusion and damage to the alpha cells of the pancreas. Chronic exposure to cobalt compounds may result in pericardial effusion, polycardial effusion, cardiac failure, vomiting, convulsions and thyroid enlargement.
Chronic administration of cobaltous chloride has produced goiter, reduced thyroid activity and lowered synthesis rates and levels of cytochrome P-450, an enzymatic system responsible for chemical detoxification, in the liver. A toxic nephritis (kidney disease) may also develop.
Epidemic cardiomyopathy (heart disease) among heavy beer drinkers in the 1960's in Canada, the USA and Belgium has been attributed to the addition of up to 1.5 ppm of cobalt as a foam restorative and stabiliser. Other factors are probably implicated as therapeutic doses of cobalt, up to 50 mg/day (in the treatment of refractory anaemias) do not produce this effect. Inadequate protein or vitamin intake amongst heavy drinkers, or the effects of alcohol in rendering the heart more susceptible to disease may be important.
Single and repeated subcutaneous or intramuscular injection of cobalt powder and salts to rats may cause sarcoma at the injection site but evidence for carcinogenicity by any other route of exposure does not exist. A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopedic implants containing cobalt.
Animals, exposed to cobalt compounds also exhibit an increase in respiration, as well as tremor and convulsion. Exposure of rats and mice to aerosols of cobalt (as cobalt sulfate) at concentrations from 0.11 to 1.14 mg cobalt/m3 for 2 years resulted in a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract of male and female rats and mice. Squamous metaplasia of the larynx occurred in rats and mice at exposure concentrations of .0.11 mg cobalt/m3, with severity of the lesion increasing with increased cobalt concentration. Hyperplastic lesions of the nasal epithelium occurred in rats at concentrations of .0.11 mg cobalt/3, and in mice at concentrations of .0.38 mg cobalt/m3. Both sexes of rats had greatly increased incidences (>90% incidence) of alveolar lesions at all exposure levels, including inflammatory changes, fibrosis, and metaplasia. Similar changes were seen in mice at all exposure levels, though the changes in mice were less severe.
Cobalt metal dust inhalations by miniature swine resulted in early marked decrease in lung compliance and increases in septal collagen. After a one-week "sensitising period", followed by a 10-day lapse period, further exposures resulted in wheezing produced by hypersensitivity reactions.
Some anthraquinone (also known as anthracenedione) dyes are carcinogenic while others are positive allergens which cause hypersensitivity responses in unsensitised humans or cause or cause immunotoxic responses. Some of these dyes cause dermatitis whilst others produce slight teratogenic effects when administered intraperitoneally to pregnant mice. Information on the neurotoxic effects and metabolism on most members of this class of dyes is missing.
Anthraquinones are classified with a large number of other quinone molecules that can be derived from aromatic molecules such as benzene, naphthalene, and anthracene. Reactive oxygen species generated by metabolism of a variety of quinones may be associated with DNA damage or activation of signaling pathways involved in initiation, promotion, and progression of carcinogenesis. A high percentage (36/80) of phenolic anthraquinones have been reported to be mutagenic in ***Salmonella.***
Quinone molecules can be reduced to a relatively stable hydroquinone, which usually is not associated with oxidative stress, or they may be reduced in a one-electron reduction to semiquinone free radicals that give rise to superoxide anions, hydrogen peroxide, and other reactive oxygen species Quinones may be produced from benzene, polycyclic aromatic hydrocarbons, estrogens, and catecholamines and give rise to reactive oxygen species that can damage DNA and other cellular macromolecules and activate signaling pathways. These molecular events may be associated with the initiation, promotion, and progression of carcinogenesis.
Many azo dyes have been found to be carcinogenic in laboratory animals, affecting the liver, urinary bladder and intestines. Specific toxicity effects in humans have not been established but some dyes are known to be mutagenic.
The simplest azo dyes, which raise concern, have an exocyclic amino-group that is the key to any carcinogenicity for it is this group which undergoes biochemical N-oxidation and further reaction to reactive electrophiles. The DNA adducts formed by covalent binding through activated nitrogen have been identified. However not all azo compounds possess this activity and delicate alterations to structure vary the potential of carcinogenicity / acid, reduces or eliminates the effect. Complex azo dyes consisting of more than one azo (N=N) linkage may be metabolised to produce complexed carcinogenic aromatic amines such as benzidine
Benzidine and its metabolic derivatives have been detected in the urine of workers exposed to Direct azo dyes. An epidemiological study of silk dyers and painters with multiple exposures to benzidine based and other dyes indicate a strong association with bladder cancer.
Most organic azo dyes are potential skin sensitisers, the most important of which are para-phenylenediamine and its analogs. Water soluble azo dyes are more likely to cause clinical sensitisation than insoluble dyes. In addition to allergic eczematous contact dermatitis, color developing solutions have caused lichen planus like eruptions
NTP studies of nitro- and amino-anthraquinones, have demonstrated that each compound tested has some activity as a mutagen. Most compounds of this class that have been the subjects of two-year studies have also been found to be carcinogenic in one or more species. Sites of tumor development include the urinary bladder in rats and the liver of both rats and mice.
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.
Chronic severe inhalation exposure to sodium carbonate may result in perforation of the nasal septum and serious pulmonary oedema (lung damage).

Jasart Tie Dye Sets

TOXICITY	IRRITATION
Not Available	Not Available

sodium bicarbonate

TOXICITY	IRRITATION
Oral (Mouse) LD50; 3360 mg/kg[2]	Eye (Rodent - rabbit): 100mg/30S - Mild
	Eye: no adverse effect observed (not irritating)[1]
	Skin (Human): 30mg/3D - Mild
	Skin: no adverse effect observed (not irritating)[1]

Continued...

C.I. Reactive Red 194	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
copper phthalocyanine, sulfonyl derivatives	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
C.I. Reactive Yellow 3	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
C.I. Reactive Red 24	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: >1.5 mg/L4h[1]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[2]	
C.I. Reactive Black 8	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 9120 mg/kg[2]	Eye (Rodent - rabbit): 500mg/24H - Mild
		Skin (Rodent - rabbit): 500mg/24H - Mild
C.I. Reactive Blue 74	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
C.I. Reactive Blue 19	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
sodium chloride	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >10000 mg/kg[1]	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Inhalation (Rat) LC50: >10.5 mg/l4h[1]	Eye (Rodent - rabbit): 10mg - Moderate
	Oral (Rat) LD50: 3000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
sodium carbonate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Oral (Rat) LD50: 2800 mg/kg[2]	Eye (Rodent - rabbit): 100mg/30S - Mild
		Eye (Rodent - rabbit): 50mg - Severe
		Eye: adverse effect observed (irritating)[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]

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

SODIUM BICARBONATE	Oral (human-infant) TDLo: 1260 mg/kg Skin (human): 30 mg/3d-I-mild
C.I. REACTIVE BLACK 8	For chrome(III) and other valence states (except hexavalent): For inhalation exposure, all trivalent and other chromium compounds are treated as particulates, not gases. The mechanisms of chromium toxicity are very complex, and although many studies on chromium are available, there is a great deal of uncertainty about how chromium exerts its toxic influence. Much more is known about the mechanisms of hexavalent chromium toxicity than trivalent chromium toxicity. There is an abundance of information available on the carcinogenic potential of chromium compounds and on the genotoxicity and mutagenicity of chromium compounds in experimental systems. The consensus from various reviews and agencies is that evidence of carcinogenicity of elemental, divalent, or trivalent chromium compounds is lacking. Epidemiological studies of workers in a number of industries (chromate production, chromate pigment production and use, and chrome plating) conclude that while occupational exposure to hexavalent chromium compounds is associated with an increased risk of respiratory system cancers (primarily bronchogenic and nasal), results from occupational exposure studies to mixtures that were mainly elemental and trivalent (ferrochromium alloy worker) were inconclusive. Studies in leather tanners, who were exposed to trivalent chromium were consistently negative. In addition to the lack of direct evidence of carcinogenicity of trivalent or elemental chromium and its compounds, the genotoxic evidence is overwhelmingly negative. The lesser potency of trivalent chromium relative to hexavalent chromium is likely related to the higher redox potential of hexavalent chromium and its greater ability to enter cells. enter cells The general inability of trivalent chromium to traverse membranes and thus be absorbed or reach peripheral tissue in significant amounts is generally accepted as a probable explanation for the overall absence of systemic trivalent chromium toxicity. Elemental and divalent forms of chromium are not able to traverse membranes readily either. This is not to say that elemental, divalent, or trivalent chromium compounds cannot traverse membranes and reach peripheral tissue, the mechanism of absorption is simply less efficient in comparison to absorption of hexavalent chromium compounds. Hexavalent chromium compounds exist as tetrahedral chromate anions, resembling the forms of other natural anions like sulfate and phosphate which are permeable across nonselective membranes. Trivalent chromium forms octahedral complexes which cannot easily enter though these channels, instead being absorbed via passive diffusion and phagocytosis. Although trivalent chromium is less well absorbed than hexavalent chromium, workers exposed to trivalent compounds have had detectable levels of chromium in the urine at the end of a workday. Absorbed chromium is widely distributed throughout the body via the bloodstream, and can

Continued...

	reach the foetus. Although there is ample in vivo evidence that hexavalent chromium is efficiently reduced to trivalent chromium in the gastrointestinal tract and can be reduced to the trivalent form by ascorbate and glutathione in the lungs, there is no evidence that trivalent chromium is converted to hexavalent chromium in biological systems. In general, trivalent chromium compounds are cleared rapidly from the blood and more slowly from the tissues. Although not fully characterized, the biologically active trivalent chromium molecule appears to be chromodulin, also referred to as (GTF). Chromodulin is an oligopeptide complex containing four chromic ions. Chromodulin may facilitate interactions of insulin with its receptor site, influencing protein, glucose, and lipid metabolism. Inorganic trivalent chromium compounds, which do not appear to have insulin-potentiating properties, are capable of being converted into biologically active forms by humans and animals Chromium can be a potent sensitiser in a small minority of humans, both from dermal and inhalation exposures. The most sensitive endpoint identified in animal studies of acute exposure to trivalent chromium appears to involve the respiratory system. Specifically, acute exposure to trivalent chromium is associated with impaired lung function and lung damage. Based on what is known about absorption of chromium in the human body, its potential mechanism of action in cells, and occupational data indicating that valence states other than hexavalent exhibit a relative lack of toxicity the toxicity of elemental and divalent chromium compounds is expected to be similar to or less than common trivalent forms.
C.I. REACTIVE BLUE 19	product:
SODIUM CHLORIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SODIUM CARBONATE	for sodium carbonate: Sodium carbonate has no or a low skin irritation potential but it is considered irritating to the eyes. Due to the alkaline properties an irritation of the respiratory tract is also possible. No valid animal data are available on repeated dose toxicity studies by oral, dermal, inhalation or by other routes for sodium carbonate. A repeated dose inhalation study, which was not reported in sufficient detail, revealed local effects on the lungs which could be expected based on the alkaline nature of the compound. Under normal handling and use conditions neither the concentration of sodium in the blood nor the pH of the blood will be increased and therefore sodium carbonate is not expected to be systemically available in the body. It can be stated that the substance will neither reach the foetus nor reach male and female reproductive organs, which shows that there is no risk for developmental toxicity and no risk for toxicity to reproduction. This was confirmed by a developmental study with rabbits, rats and mice. An ***in vitro*** mutagenicity test with bacteria was negative and based on the structure of sodium carbonate no genotoxic effects are expected.
SODIUM BICARBONATE & C.I. REACTIVE BLACK 8 & C.I. REACTIVE BLUE 19 & SODIUM CHLORIDE & SODIUM CARBONATE	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
C.I. REACTIVE RED 194 & C.I. REACTIVE YELLOW 3 & C.I. REACTIVE BLUE 74	No significant acute toxicological data identified in literature search.
C.I. REACTIVE BLACK 8 & C.I. REACTIVE BLUE 19	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SODIUM CHLORIDE & SODIUM CARBONATE	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.

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

Jasart Tie Dye Sets

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

sodium bicarbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	101mg/l	2
NOEC(ECx)	240h	Algae or other aquatic plants	26.8mg/l	2
EC50	96h	Algae or other aquatic plants	650mg/L	4
LC50	96h	Fish	833.28mg/L	4

C.I. Reactive Red 194

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

copper phthalocyanine, sulfonyl derivatives

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>500mg/l	2
EC50	48h	Crustacea	>100mg/l	2

Continued...

	NOEC(ECx)	168h	Algae or other aquatic plants	10mg/l	2

C.I. Reactive Yellow 3

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

C.I. Reactive Red 24

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2
NOEC(ECx)	168h	Algae or other aquatic plants	100mg/l	2

C.I. Reactive Black 8

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

C.I. Reactive Blue 74

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

C.I. Reactive Blue 19

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	14.4mg/l	2
BCF	672h	Fish	<1.2	7
EC50	48h	Crustacea	>100mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	2.3mg/l	2
LC50	96h	Fish	>100<500mg/l	2

sodium chloride

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	20.76-36.17mg/L	4
EC50	48h	Crustacea	0.004-0.006mg/L	4
NOEC(ECx)	6h	Fish	0.001mg/L	4
EC50	96h	Algae or other aquatic plants	1110.36mg/L	4
LC50	96h	Fish	1000mg/L	4

sodium carbonate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	>800mg/l	2
NOEC(ECx)	48h	Fish	0.011mg/L	4
EC50	48h	Crustacea	156.6-298.9mg/l	4
EC50	96h	Algae or other aquatic plants	242mg/L	4
LC50	96h	Fish	300mg/l	2

Legend: ***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data 8. User defined data***

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
sodium bicarbonate	LOW	LOW
sodium chloride	LOW	LOW
sodium carbonate	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium bicarbonate	LOW (LogKOW = -4.01)
C.I. Reactive Yellow 3	LOW (LogKOW = 1.23)
C.I. Reactive Red 24	LOW (LogKOW = 0.88)
C.I. Reactive Blue 19	LOW (BCF = 13)
sodium chloride	LOW (LogKOW = 0.54)
sodium carbonate	LOW (LogKOW = -0.4605)

Mobility in soil

Ingredient	Mobility
sodium bicarbonate	HIGH (Log KOC = 1)
sodium chloride	LOW (Log KOC = 14.3)
sodium carbonate	HIGH (Log KOC = 1)

Continued...

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
sodium bicarbonate	Not Applicable
C.I. Reactive Red 194	Not Applicable
copper phthalocyanine, sulfonyl derivatives	Not Applicable
C.I. Reactive Yellow 3	Not Applicable
C.I. Reactive Red 24	Not Applicable
C.I. Reactive Black 8	Not Applicable
C.I. Reactive Blue 74	Not Applicable
C.I. Reactive Blue 19	Not Applicable
sodium chloride	Not Applicable
sodium carbonate	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium bicarbonate	Not Applicable
C.I. Reactive Red 194	Not Applicable
copper phthalocyanine, sulfonyl derivatives	Not Applicable
C.I. Reactive Yellow 3	Not Applicable
C.I. Reactive Red 24	Not Applicable
C.I. Reactive Black 8	Not Applicable
C.I. Reactive Blue 74	Not Applicable
C.I. Reactive Blue 19	Not Applicable
sodium chloride	Not Applicable
sodium carbonate	Not Applicable

Continued...

SECTION 15 Regulatory information

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

sodium bicarbonate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Reactive Red 194 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

copper phthalocyanine, sulfonyl derivatives 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)

C.I. Reactive Yellow 3 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Reactive Red 24 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Reactive Black 8 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Reactive Blue 74 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

C.I. Reactive Blue 19 is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium chloride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium carbonate 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)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (C.I. Reactive Red 194; C.I. Reactive Yellow 3; C.I. Reactive Black 8; C.I. Reactive Blue 74)
Canada - NDSL	No (sodium bicarbonate; C.I. Reactive Red 194; copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Red 24; C.I. Reactive Black 8; C.I. Reactive Blue 74; C.I. Reactive Blue 19; sodium chloride; sodium carbonate)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (sodium bicarbonate; C.I. Reactive Red 194; copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Yellow 3; C.I. Reactive Red 24; C.I. Reactive Black 8; C.I. Reactive Blue 74; C.I. Reactive Blue 19; sodium chloride; sodium carbonate)
Europe - EINEC / ELINCS / NLP	No (C.I. Reactive Black 8; C.I. Reactive Blue 74)
Japan - ENCS	No (copper phthalocyanine, sulfonyl derivatives)
Korea - KECI	No (copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Yellow 3)
New Zealand - NZIoC	No (C.I. Reactive Yellow 3; C.I. Reactive Blue 74)
Philippines - PICCS	No (C.I. Reactive Yellow 3; C.I. Reactive Blue 74)
USA - TSCA	TSCA Inventory 'Active' substance(s) (sodium bicarbonate; copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Red 24; C.I. Reactive Blue 19; sodium chloride; sodium carbonate); TSCA Inventory 'Inactive' substance(s) (C.I. Reactive Yellow 3); No (C.I. Reactive Red 194; C.I. Reactive Black 8; C.I. Reactive Blue 74)
Taiwan - TCSI	No (C.I. Reactive Yellow 3; C.I. Reactive Blue 74)
Mexico - INSQ	No (C.I. Reactive Red 194; copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Yellow 3; C.I. Reactive Red 24; C.I. Reactive Black 8; C.I. Reactive Blue 74; C.I. Reactive Blue 19)
Vietnam - NCI	No (C.I. Reactive Yellow 3; C.I. Reactive Blue 74)
Russia - FBEPH	No (C.I. Reactive Red 194; copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Yellow 3; C.I. Reactive Red 24; C.I. Reactive Black 8; C.I. Reactive Blue 74)
UAE - Control List (Banned/Restricted Substances)	No (sodium bicarbonate; C.I. Reactive Red 194; copper phthalocyanine, sulfonyl derivatives; C.I. Reactive Yellow 3; C.I. Reactive Red 24; C.I. Reactive Black 8; C.I. Reactive Blue 74; C.I. Reactive Blue 19; sodium chloride; sodium carbonate)
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.***

Continued...

SECTION 16 Other information

Revision Date	02/02/2026
Initial Date	19/05/2021

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	02/02/2026	Expiration. Review and Update

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

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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