

Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block

DENTSPLY Canada Ltd.

Chemwatch: 7963-75

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 1

Initial Date: 19/06/2025

Revision Date: 19/06/2025

Print Date: 20/06/2025

L.GHS.AUS.EN

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

Product Identifier

| | |
|-------------------------------|---|
| Product name | Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block |
| 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 | For dental use only. Use according to manufacturer's directions. |
|--------------------------|---|

Details of the manufacturer or importer of the safety data sheet

| | |
|-------------------------|---|
| Registered company name | DENTSPLY Canada Ltd. |
| Address | 525 Rowntree Dairy Road, Unit 1 Woodbridge Ontario L4L 7K6 Canada |
| Telephone | (800)-263-1437 (732)-389-8540 |
| Fax | Not Available |
| Website | https://www.dentsplysirona.com/en-ca |
| Email | Canada.CustomerCare@dentsplysirona.com |

Emergency telephone number

| | | |
|-------------------------------------|-------------------------------------|-------------------------------------|
| Association / Organisation | DENTSPLY Canada Ltd. | CHEMWATCH EMERGENCY RESPONSE (24/7) |
| Emergency telephone number(s) | (877) 819-6206 (Mon-Fri 9am to 6pm) | +61 1800 951 288 (ID#: 7963-75) |
| Other emergency telephone number(s) | (732)-389-8540 (Mon-Fri 9am to 6pm) | +61 3 9573 3188 |

SECTION 2 Hazards identification

Classification of the substance or mixture

| | |
|-------------------------------|---|
| Poisons Schedule | Not Applicable |
| Classification ^[1] | Non hazardous |
| 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) | Not Applicable |
| Signal word | Not Applicable |

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

| CAS No | %[weight] | Name |
|--|-----------|--|
| Not Available | 100 | Ingredients determined not to be hazardous |
| 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 | <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none">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.Generally not applicable. |
| Skin Contact | <p>If skin contact occurs:</p> <ul style="list-style-type: none">Immediately remove all contaminated clothing, including footwear.Flush skin and hair with running water (and soap if available).Seek medical attention in event of irritation.Generally not applicable. |
| Inhalation | <ul style="list-style-type: none">If fumes or combustion products are inhaled remove from contaminated area.Lay patient down. Keep warm and rested.Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures.Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.Transport to hospital, or doctor.Generally not applicable. |
| Ingestion | <ul style="list-style-type: none">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.Generally not applicable. |

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.

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- It is unlikely that methylene blue would be effective against the occasional 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]

SECTION 5 Firefighting measures**Extinguishing media**

- There is no restriction on the type of extinguisher which may be used.
- Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

| | |
|------------------------------|--|
| Fire Fighting | <ul style="list-style-type: none"> ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ DO NOT approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use. <p>Slight hazard when exposed to heat, flame and oxidisers.</p> |
| Fire/Explosion Hazard | <p>Articles and manufactured articles may constitute a fire hazard where polymers form their outer layers or where combustible packaging remains in place.</p> <p>Certain substances, found throughout their construction, may degrade or become volatile when heated to high temperatures. This may create a secondary hazard.</p> <p>carbon dioxide (CO₂) nitrogen oxides (NO_x) metal oxides other pyrolysis products typical of burning organic material.</p> <p>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.</p> <p>May emit poisonous fumes. May emit corrosive fumes.</p> |
| 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 | <ul style="list-style-type: none"> ▸ Clean up all spills immediately. ▸ Secure load if safe to do so. ▸ Bundle/collect recoverable product. ▸ Collect remaining material in containers with covers for disposal. |
| Major Spills | <ul style="list-style-type: none"> ▸ 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. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services. ▸ Clean up all spills immediately. ▸ Wear protective clothing, safety glasses, dust mask, gloves. ▸ Secure load if safe to do so. Bundle/collect recoverable product. |

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- ▶ Use dry clean up procedures and avoid generating dust.
 - ▶ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use).
 - ▶ Water may be used to prevent dusting.
 - ▶ Collect remaining material in containers with covers for disposal.
 - ▶ Flush spill area with water.
- Minor hazard.
- ▶ Clear area of personnel.
 - ▶ Alert Fire Brigade and tell them location and nature of hazard.
 - ▶ Control personal contact with the substance, by using protective equipment as required.
 - ▶ Prevent spillage from entering drains or water ways.
 - ▶ Contain spill with sand, earth or vermiculite.
 - ▶ Collect recoverable product into labelled containers for recycling.
 - ▶ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal.
 - ▶ Wash area and prevent runoff into drains or waterways.
 - ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

| | |
|-------------------|--|
| Safe handling | <ul style="list-style-type: none">▶ Avoid all personal contact, including inhalation.▶ Wear protective clothing when risk of exposure occurs.▶ Use in a well-ventilated area.▶ Prevent concentration in hollows and sumps.▶ DO NOT enter confined spaces until atmosphere has been checked.▶ DO NOT allow material to contact humans, exposed food or food utensils.▶ Avoid contact with incompatible materials.▶ When handling, DO NOT eat, drink or smoke.▶ Keep containers securely sealed when not in use.▶ Avoid physical damage to containers.▶ Always wash hands with soap and water after handling.▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.▶ Use good occupational work practice.▶ Observe manufacturer's storage and handling recommendations contained within this SDS.▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| Other information | <ul style="list-style-type: none">▶ Store away from incompatible materials. |

Conditions for safe storage, including any incompatibilities

| | |
|-------------------------|--|
| Suitable container | Generally packaging as originally supplied with the article or manufactured item is sufficient to protect against physical hazards. If repackaging is required ensure the article is intact and does not show signs of wear. As far as is practicably possible, reuse the original packaging or something providing a similar level of protection to both the article and the handler. |
| Storage incompatibility | <ul style="list-style-type: none">▶ Avoid reaction with oxidising agents |

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

| Ingredient | Original IDLH | Revised IDLH |
|---|---------------|---------------|
| Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block | Not Available | Not Available |

MATERIAL DATA

Exposure controls

| | |
|----------------------------------|--|
| Appropriate engineering controls | Articles or manufactured items, in their original condition, generally don't require engineering controls during handling or in normal use. Exceptions may arise following extensive use and subsequent wear, during recycling or disposal operations where substances, found in the article, may be released to the environment. |
|----------------------------------|--|

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Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

| Type of Contaminant: | Air Speed: |
|---|------------------------------|
| solvent, vapours, degreasing etc., evaporating from tank (in still air). | 0.25-0.5 m/s (50-100 f/min.) |
| aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) |
| grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). | 2.5-10 m/s (500-2000 f/min.) |

Within each range the appropriate value depends on:

| Lower end of the range | Upper end of the range |
|--|----------------------------------|
| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents |
| 2: Contaminants of low toxicity or of nuisance value only. | 2: Contaminants of high toxicity |
| 3: Intermittent, low production. | 3: High production, heavy use |
| 4: Large hood or large air mass in motion | 4: Small hood-local control only |

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Individual protection measures, such as personal protective equipment



Eye and face protection

- ▶ Safety glasses with side shields.
 - ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
 - ▶ 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].
- No special equipment required due to the physical form of the product.

Skin protection

See Hand protection below

Hands/feet protection

- ▶ Wear chemical protective gloves, e.g. PVC.
 - ▶ Wear safety footwear or safety gumboots, e.g. Rubber
- No special equipment required due to the physical form of the product.

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:

Respiratory protection

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

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"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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| Material | CPI |
|----------|-----|
| BUTYL | A |
| NITRILE | A |

* 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.

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 |
|------------------------------------|--|----------------------|----------------------|
| up to 10 | 1000 | A-AUS / Class1 P2 | - |
| up to 50 | 1000 | - | A-AUS / Class 1 P2 |
| up to 50 | 5000 | Airline * | - |
| up to 100 | 5000 | - | A-2 P2 |
| up to 100 | 10000 | - | A-3 P2 |
| 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)

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

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 | Cylinder with no odour; 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 (°C) | Not Available |
| Melting point / freezing point (°C) | Not Available | Viscosity (cSt) | Not Applicable |
| Initial boiling point and boiling range (°C) | Not Available | 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 | Not Available | pH as a solution (1%) | Not Applicable |
| Vapour density (Air = 1) | Not Available | VOC g/L | Not Available |
| Heat of Combustion (kJ/g) | Not Available | Ignition Distance (cm) | Not Available |

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

SECTION 11 Toxicological information

Information on toxicological effects

| | |
|---|---|
| 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 | Based on available data, the classification criteria are not met. |
| 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 | 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. 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. |
| Ingestion | Accidental ingestion of the material may be damaging to the health of the individual. |
| Skin Contact | Limited 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. |
| Eye | Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to 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, 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. Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. |

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Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.

After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans.

At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively. Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramidal-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations.

Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA). Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia*, August 1995]

The main target organs of aluminium are the central nervous system and bone. Aluminium binds with dietary phosphorus and impairs gastrointestinal absorption of phosphorus. The decreased phosphate body burden results in osteomalacia (softening of the bones due to defective bone mineralization) and rickets. Aluminium's neurotoxicity is believed to involve several mechanisms. Changes in cytoskeletal protein functions as a result of altered phosphorylation, proteolysis, transport, and synthesis are believed to be one cause. Aluminium may induce neurobehavioral effects by affecting permeability of the blood-brain barrier, cholinergic activity, signal transduction pathways, lipid peroxidation, and impair neuronal glutamate nitric oxide-cyclic GMP pathway, as well as interfere with metabolism of essential trace elements because of similar coordination chemistries and consequent competitive interactions. It has been suggested that aluminium's interaction with estrogen receptors, but studies have not been able to establish a clear link between aluminium and increased risk of breast cancer. Certain aluminium salts induce immune responses by activating inflammasomes.

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.

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/m³, 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.

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 excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.

Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.

High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.

Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.

[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

Chronic exposure to tin dusts and fume results in "stannosis" a mild form of pneumoconiosis. Chest symptoms develop several years after breathing difficulties (dyspnoea) occur. No case of massive fibrosis from over-exposure to tin has been reported.

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 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.

Oral administration of zirconium sulfate (5 ppm) to rats fed from weanlings to natural death failed to influence longevity, tumour incidence or organ to body weight ratios. Zirconium tended to accumulate in the spleen. One-year inhalation studies using zirconium oxide zirconium tetrachloride (3.5 mg Zr/m³) failed to produce adverse effects in rats.

Zirconium is routinely found with low levels of hafnium since separation of the two metals is difficult.

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 Al₂O₃ 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 alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.

Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature.

Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC).

Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).

Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.

Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block

Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin). Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.

Yttrium is a member of the so-called heavy-group (the yttriums) of the rare earths (or lanthanoids). No occupational diseases or cases of poisoning in workers producing rare earth elements have been described.

Lanthanoids entering the human body due to exposure to various industrial processes can affect metabolic processes. Trivalent lanthanoid ions, especially lanthanum 3+ and gadolinium 3+, can interfere with calcium channels in human and animal cells.

Lanthanoids can also alter or even inhibit the action of various enzymes. Lanthanoid ions found in neurons can regulate synaptic transmission, as well as block some receptors (for example, glutamate receptors). Lanthanoids target the liver causing fatty liver degeneration and a decrease in liver glycogen and blood glucose levels.

Lanthanoids because of their high density can produce significant abnormalities in a chest X-ray. Lanthanoids are generally not fibrogenic and lesions typically have little or no clinical importance. Occasional cases of suspected pneumoconiosis have however been reported. The toxicity of all elements in the yttrium group has been investigated in workers and animals alike. Effects on peripheral blood including a decrease haemoglobin and erythrocyte content and changes in the leucocyte formula have been recorded. Studies show that yttrium is deposited in the skeleton within only a few hours of intake. Animal lungs show productive inflammation and a tendency to develop nodular or diffuse sclerosis following administration by intratracheal injection. The main risks to workers involved in production of rare earths are due to dust inhalation.

Based on the available toxicity data, the rare earth chlorides appear to have moderate acute and chronic toxicity. However these substances cause severe eye irritation and severe irritation in abraded skin. They are poorly absorbed by the gastrointestinal tract and by unbroken skin but slight liver damage has been demonstrated in subchronic oral toxicity studies at high doses. The literature indicates that chronic inhalation exposure to the rare earth chlorides may cause pneumoconiosis in humans. There are no indications of carcinogenicity in the rare earth chlorides. Mutagenicity studies on these substances have mixed results, but are predominantly negative.

* IUPAC currently recommends the name lanthanoid rather than lanthanide, as the suffix "-ide" generally indicates negative ions whereas the suffix "-oid" indicates similarity to one of the members of the containing family of elements. In the older literature, the name "lanthanon" was often used.

| Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block | 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

| | | | |
|-----------------------------------|---|--------------------------|---|
| 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

| Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block | 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 | | | | |

DO NOT discharge into sewer or waterways.

Persistence and degradability

Continued...

Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|------------|---------------------------------------|---------------------------------------|
| | No Data available for all ingredients | No Data available for all ingredients |

Bioaccumulative potential

| Ingredient | Bioaccumulation |
|------------|---------------------------------------|
| | No Data available for all ingredients |

Mobility in soil

| Ingredient | Mobility |
|------------|---------------------------------------|
| | No Data available for all ingredients |

SECTION 13 Disposal considerations

Waste treatment methods

| | |
|------------------------------|--|
| Product / Packaging disposal | <ul style="list-style-type: none"> ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ 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 |
|--------------|-------|
|--------------|-------|

14.7.3. Transport in bulk in accordance with the IGC Code

| Product name | Ship Type |
|--------------|-----------|
|--------------|-----------|

SECTION 15 Regulatory information

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

Additional Regulatory Information

Not Applicable

National Inventory Status

| National Inventory | Status |
|---|---------------|
| Australia - AIIC / Australia Non-Industrial Use | Not Available |

Continued...

Celtra Duo, Celtra Press, Cerec Tessera, Celtra CAD, CEREC Tessera Abutment Block

| National Inventory | Status |
|-------------------------------|--|
| Canada - DSL | Not Available |
| Canada - NDSL | Not Available |
| China - IECSC | Not Available |
| Europe - EINEC / ELINCS / NLP | Not Available |
| Japan - ENCS | Not Available |
| Korea - KECI | Not Available |
| New Zealand - NZIoC | Not Available |
| Philippines - PICCS | Not Available |
| USA - TSCA | Not Available |
| Taiwan - TCSI | Not Available |
| Mexico - INSQ | Not Available |
| Vietnam - NCI | Not Available |
| Russia - FBEPH | Not Available |
| Legend: | <p><i>Yes = All CAS declared ingredients are on the inventory</i></p> <p><i>No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.</i></p> |

SECTION 16 Other information

| | |
|----------------------|------------|
| Revision Date | 19/06/2025 |
| Initial Date | 19/06/2025 |

SDS Version Summary

| Version | Date of Update | Sections Updated |
|---------|----------------|--|
| 2.1 | 19/06/2025 | Composition / information on ingredients - Ingredients |

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

Continued...

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- 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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