



JTC IMPORT/EXPORT P/L

Personal Care - Nasal Spray – Saline

JTC Import Export Pty Ltd

Chemwatch: 7956-82

Version No: 2.1

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

Chemwatch Hazard Alert Code: 0

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

Product Identifier

Product name	Personal Care - Nasal Spray – Saline
Chemical Name	Not Applicable
Synonyms	Product Code: 82365 Personal Care® - Nasal Spray – Saline
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	Therapeutic or pharmacologically-active agent. An NF-κB specific inhibitor or activator. NF-κB (Nuclear factor kappa-light-chain-enhancer of activated B cells) is a protein complex that controls transcription of DNA. NF-κB is found in almost all animal cell types and is involved in cellular responses to stimuli such as stress, cytokines, free radicals, ultraviolet irradiation, oxidized LDL, and bacterial or viral antigens. NF-κB plays a key role in regulating the immune response to infection. Incorrect regulation of NF-κB has been linked to cancer, inflammatory, and autoimmune diseases, septic shock, viral infection, and improper immune development. NF-κB has also been implicated in processes of synaptic plasticity and memory. There are five proteins in the mammalian NF-κB family: NF-κB1, NF-κB2, RelA, RelB, c-Rel. Nuclear factor kappa B (NF-κB) transcription factors regulate the expression of a great number of genes which control several important physiological processes, including inflammation and immune responses, cell growth, apoptosis, cancer development and progression, diabetes, stroke, muscle wasting, a variety of auto-immune diseases and the expression of certain viral genes. Therefore, the NF-κB signaling pathway has also provided a focus for pharmacological intervention, primarily in situations of chronic inflammation or in cancer, where the pathway is often constitutively active and plays a key role in the disease. Nuclear factor kappa B (NF-κB) signaling is implicated in all major human chronic diseases, with its role in transcription of hundreds of genes well established in the literature. This has propelled research into targeting the NF-κB pathways for modulating expression of those genes and the diseases mediated by them. In spite of the critical, but often promiscuous role played by this pathway and the inhibition causing adverse drug reaction, currently many biologics, macromolecules, and small molecules that modulate this pathway are in the market or in clinical trials. Furthermore, many marketed drugs that were later found to also have NF-κB targeting activity were repurposed for new therapeutic interventions. NF-κB is involved in the expression of proinflammatory genes (e.g., cytokines and chemokines), and therefore, is a master regulator of inflammation. NF-κB is activated in response to various stimuli, such as infections and stress signals. Its dysregulation is associated with a variety of diseases, such as atherosclerosis, infections, and cancer progression. NF-κB inhibitors are likely to be developed as treatments for certain cancers and neurodegenerative and inflammatory diseases. Moreover, the therapeutic and preventative effects of many natural products may, at least in part, be due to their ability to inhibit NF-κB. Over 750 inhibitors of the NFκB pathway have been identified, including a variety of natural and synthetic molecules. These compounds include antioxidants, peptides, small RNA/DNA, microbial and viral proteins, small molecules, and engineered dominant-negative or constitutively active polypeptides. Several of these molecules act as general inhibitors of NF-κB induction, whereas others inhibit specific pathways of induction. In addition, some compounds appear to target multiple steps in the NF-κB pathway. NF-κB transcription factors control the expression of genes involved in many critical physiological responses, including immune and acute phase inflammatory responses, cell adhesion, differentiation, oxidative stress responses and apoptosis. NF-κB complexes are known to play key roles in the inhibition of apoptosis, and in cell proliferation, and as a consequence, mis-regulated, usually sustained, NF-κB activity also contributes to human disease states, including most chronic inflammatory diseases and many cancers. The molecular cascade of signaling events provides several steps for specific inhibition of NF-κB activity. Generally speaking, inhibition of NF-κB activation can occur by three mechanisms: (1) blockage of the incoming stimulating signal at an early stage (e.g., binding of ligand to its receptor) resulting in complete abrogation of the signal's effect; (2) interference with a cytoplasmic step in the NF-κB activation pathway by blockage of a specific component of the cascade (e.g., the recruitment of an adaptor to the receptor complex, the activation of the IKK complex, or degradation of IκB); or (3) blockage of NF-κB nuclear activity, that is, inhibiting its translocation to the nucleus, its binding to DNA, a nuclear modification of NF-κB that affects its activity or specificity, or an interaction of NF-κB on DNA with specific or basal transcription machinery. In most cases, each of these three major steps is not susceptible to the same types of inhibitors. Activation of NF-κB is mediated by two major signaling pathways, termed as the canonical and non-canonical NF-κB signaling pathway. The canonical (or classic) pathway regulates the release and nuclear translocation of the canonical NF-κB subunits (such as NF-κB1 p50, RelA, and c-Rel), whereas the non-canonical pathway is selectively responsible for the generation of NF-κB2 p52). The non-canonical signaling pathway is principally induced by a subset of tumor necrosis factor receptor (TNFR) family members [such as CD40, B cell activating factor receptor (BAFFR), and lymphotoxin-beta receptor (LTβR)]. Vertebrate NF-κB transcription complexes can be any of a variety of homo- and heterodimers formed by the subunits p50, p52, c-Rel, RelA (p65) and RelB. These complexes bind to DNA regulatory sites called κB sites, generally to activate specific target gene expression. The target gene specificity of NF-κB dimers is thought to arise from a number of considerations, including the specific NF-κB complexes that are in different cell types, the distinct κB target site binding specificities of different NF-κB complexes, and the different protein-protein interactions and posttranslational modifications that NF-κB complexes undergo in different contexts. In most cell types, NF-κB dimers are located in the cytoplasm in an inactive form through association with any of several IκB inhibitor proteins (IκB-α, -β, -ε, -γ, p105 and p100). In response to a wide array of stimuli, many of which are involved in intercellular communication such as proinflammatory molecules, IκB is rapidly phosphorylated, ubiquitinated and degraded by the proteasome. The freed NF-κB dimer then translocates to the nucleus where it can modulate specific gene expression. NF-κB is a molecular target for anticancer therapies because of the effects of NF-κB target genes on tumor proliferation and microenvironment. NF-κB regulated genes involved in tumor progression, angiogenesis, and metastasis include IL-6, IL-8, uPA, VEGF, and MMP-9. In particular, NF-κB signaling is essential for estrogen receptor-negative (ER-) breast cancer tumorigenesis, progression, and metastasis. Furthermore, the role of NF-κB in expressing proinflammatory cytokines and enzymes contribute to the strong correlation between
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inflammation and breast cancer. Among the proinflammatory enzymes that are transcriptionally controlled by NF- κ B are cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (NOS2). COX-2 and NOS2 expression in ER- breast cancer is highly correlated with poor patient prognosis. The predictive value of NOS2 was tumor-node-metastasis stage independent suggesting that NF- κ B activation of NOS2 may have a role in the initiation and progression of the disease state. These findings, in addition to many other reports, imply that pharmacologic targeting of NF- κ B activity may provide a unique mechanism to limit cancer morbidity and mortality in patients with ER- disease.

However, there are multiple pathways for activation of NF- κ B. The two most common pathways are the canonical (or classical) and the non-canonical (or alternative) pathway. In the canonical pathway, a complex such as p50-RelA/I κ Balpa is activated by an IKK complex containing IKK alpha/IKK beta/NEMO, with IKK beta being the primary kinase for I κ Balpa. The IKK (I κ B kinase) is an enzyme complex that is involved in propagating the cellular response to inflammation. The IKK enzyme complex is part of the upstream NF- κ B signal transduction cascade

In the non-canonical pathway, the latent cytoplasmic NF- κ B complex is p100/RelB. Upon activation by certain receptor signals, an IKK alpha homodimer complex becomes activated and phosphorylates p100 at two C-terminal serine residues. This then promotes the ubiquitination of p100 and the proteasomal processing of the complex to p52/RelB.

There are also additional non-IKK-dependent NF- κ B pathways: for example, ultraviolet irradiation-induced NF- κ B activation does not appear to use the IKK complex, and phosphorylation of I κ Balpa at tyrosine residue 42 can lead to activation of NF- κ B

Toll-like receptor inhibitor/ modulator.

Toll-like receptors (TLRs) are innate immune receptors. They recognize pathogen-associated molecular patterns (PAMPs), which are molecular structures conserved among different pathogens. They also recognize host-derived molecules named alarmins (danger-associated molecular patterns (DAMPs)), which mediate sterile inflammation after trauma, stress, and injury. Ligand of PAMPs and DAMPs by TLRs in antigen-presenting cells, such as macrophages or dendritic cells, leads to the activation of transcription factors (e.g., nuclear factor kappa B (NF- κ B) or activator protein 1 (AP-1)). These transcription factors promote the production of cytokines and chemokines.

These, in turn, can lead to the activation of the adaptive immune system. It has been suggested that the endogenous ligand-recognition response could be the underlying mechanism of inflammatory processes observed in autoimmune diseases (e.g., systemic lupus erythematosus, psoriasis) and cancer. The use small-molecule inhibitors to block TLR activity prior to the manifestation of chronic inflammation is of interest. Several small molecules and peptides that inhibit the inflammatory response mediated by several TLRs have been identified.

The transcription factor NF- κ B is involved in the expression of proinflammatory genes (e.g., cytokines and chemokines), and therefore, is a master regulator of inflammation. NF- κ B is activated in response to various stimuli, such as infections and stress signals. Its dysregulation is associated with a variety of diseases, such as atherosclerosis, infections, and cancer progression. Activating signals for TLR receptors lead to phosphorylation of the I κ B (inhibitor of NF- κ B) kinases IKKalpha and IKKbeta. In turn, the active IKK complex phosphorylates I κ Balpa which leads to its degradation by ubiquitin-activating enzymes and to the liberation of NF- κ B. The cytoplasmic NF- κ B then translocates into the nucleus to initiate the expression of over 500 genes involved in inflammation, carcinogenesis, and apoptosis. A substantial number of small molecule inhibitors of IKKalpha and IKKbeta have been reported. These can be classified as adenosine triphosphate (ATP) analogues, allosteric effectors, and compounds that interact with the activation loop of IKKbeta. However, only a few of these inhibitors are selective for either of these two kinases. The design of selective small molecule inhibitors of IKKalpha and IKKbeta has proven to be challenging because the active sites of both enzymes share high structural homology. Attenuating TLR signalling is an attractive consideration in the mode of regulation of inflammation.

Toll-like receptors (TLRs) are a class of proteins that play a key role in the innate immune system. They are single, membrane-spanning, non-catalytic receptors usually expressed in sentinel cells such as macrophages and dendritic cells, that recognize structurally conserved molecules derived from microbes. Once these microbes have breached physical barriers such as the skin or intestinal tract mucosa, they are recognized by TLRs, which activate immune cell responses. The TLRs include TLR1, TLR2, TLR3, TLR4, TLR5, TLR6, TLR7, TLR8, TLR9, TLR10, TLR11, TLR12, and TLR13. Toll-Like Receptors (TLRs) play a critical role in the early innate immune response to invading pathogens and are involved in sensing endogenous danger signals. TLRs are evolutionarily conserved receptors are homologues of the Drosophila Toll protein, discovered to be important for defense against microbial infection. TLRs recognize highly conserved structural motifs known as pathogen-associated microbial patterns (PAMPs), which are exclusively expressed by microbial pathogens

Toll-like receptor 4 (TLR4) modulator

Toll-like receptor 4 (TLR4), also designated as CD284 (cluster of differentiation 284), is a key activator of the innate immune response and plays a central role in the fight against bacterial infections. TLR4 is a transmembrane protein of approximately 95 kDa that is encoded by the TLR4 gene

Toll-like receptor 4 (TLR4) induced proinflammatory signaling has been directly implicated in severe sepsis and represents an attractive therapeutic target

Most known synthetic toll-like receptor 4 (TLR4) agonists are carbohydrate-based lipid-A mimetics containing several fatty acyl chains, including a labile 3-O-acyl chain linked to the C-3 position of the non-reducing sugar known to undergo cleavage impacting stability and resulting in loss of activity

Toll-like receptor 4 (TLR4) controls the major responding signaling system that detects the presence of Gram-negative infectious pathogens, by responding to endotoxin from their outer membrane. Normally, TLR-bearing cells signal the immune system to mount a pro-inflammatory, antibacterial response and resolve infection. However, TLR4 can also respond to a variety of 'endogenous' ligands, such as fibronectin and heat shock proteins. Overstimulation or continued stimulation of TLR4 by any ligand can result in a systemic inflammatory response, progressing to hypotension, shock, organ failure and even death.

Sepsis occurs when microbes activate toll-like receptors (TLRs) stimulating widespread inflammation and activating coagulation cascades. TLR4 signal transduction has been recognized as a key pathway for lipopolysaccharide (LPS)-induced activation of various cells and an attractive target for treatment of sepsis. Inhibition of TLR4 signal transduction may suppress coagulation as well as inflammation during sepsis and may be clinically beneficial in sepsis treatment.

A large body of evidence indicates TLR4 functions to recognize and respond to signals from stressed or injured tissues. Conceptually, these danger signals may be passively released from necrotic cells, actively secreted from stressed cells, or released from the extracellular matrix as it breaks down. When recognized by TLR4 in the setting of injury or ischemia, the available evidence would suggest that these damage-associated molecular patterns (DAMPs) are thought to signal through pattern recognition receptors (PRRs) such as TLR4 in a manner similar to PAMPs. Evidence has evolved for a family of putative danger signals including hyaluronic acid (HA), heparan sulfate (HS), fibrinogen

HMGB1

TLR4 belongs to the toll-like receptor family which is representative of the pattern recognition receptors (PRR), so named for their ability to recognize evolutionarily conserved components of microorganisms (bacteria, viruses, fungi and parasites) called pathogen-associated molecular patterns (PAMPs). The recognition of a PAMP by a PRR triggers rapid activation of the innate immunity essential to fight infectious disease

TLR4 is expressed in immune cells mainly of myeloid origin, including monocytes, macrophages and dendritic cells (DC). It is also expressed at a lower level on some non-immune cells, including epithelium, endothelium, placental cells and beta cells in Langerhans islets. Most myeloid cells express also high amounts of plasma membrane-anchored CD14, which facilitates the activation of TLR4 by LPS and controls the subsequent internalization of the LPS-activated TLR4 important for receptor signaling and degradation.

The main ligands for TLR4 are lipopolysaccharides (LPS), the major components of the outer membrane of Gram-negative bacteria and some Gram-positive bacteria. TLR4 can also be activated by endogenous compounds called damage-associated molecular patterns (DAMPs), including high mobility group box protein 1 (HMGB1), S100 proteins, or histones. These compounds are released during tissue injury and by dying or necrotic cells.

The first function described for TLR4 was the recognition of exogenous molecules from pathogens (PAMPs), in particular LPS molecules from gram-negative bacteria. As pattern recognition receptor, TLR4 plays a fundamental role in pathogen recognition and activation of innate immunity which is the first line of defense against invading micro-organisms. During infection, TLR4 responds to the LPS present in tissues and the bloodstream and triggers pro-inflammatory reactions facilitating eradication of the invading bacteria.

TLR4 activation by LPS enables a rapid stimulation of a wide range of innate immune cells such as macrophages and DCs. This leads to the secretion of pro-inflammatory and type I interferons cytokines, chemokines. Production levels of these cytokines/chemokines vary according to the degree of activation of the MyD88 and TRIF signaling pathways by TLR4 agonist molecules. TLR4 activation also induces the stimulation of antigen presentation and upregulation of costimulatory molecules (such as CD40, CD80 and CD86) on innate immune cells which are required for antigen presentation for T lymphocytes. This explains why TLR4 activation by LPS is also known to stimulate the generation of effective adaptive immune responses and to induce their recruitment, polarization and maintenance via the panel of cytokines and chemokines produced

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TLR4 is also involved in the recognition of endogenous DAMP molecules leading to different signaling outcomes than PAMPs, both quantitatively and qualitatively. DAMPs can activate TLR4 in non-infectious conditions to induce tissue repair and the activation of mainly proinflammatory responses. Generally, inflammation has a protective role. It is a complex and coordinated process followed by the induction of resolution pathways that restore tissue integrity and function. However, in some cases, an excessive and/or poorly regulated inflammatory response to DAMPs can be detrimental to the organism, accelerating the development or progression of pathologies such as a number of cancers and neurodegenerative diseases.

TLR4 binds LPS with the help of LPS-binding protein (LBP) and CD14, and an indispensable contribution of the MD-2 protein stably associated with the extracellular fragment of the receptor.[15] TLR4 signaling responds to signals by forming a complex using an extracellular leucine-rich repeat domain (LRR) and an intracellular toll/interleukin-1 receptor (TIR) domain. LPS stimulation induces a series of interactions with several accessory proteins which form the TLR4 complex on the cell surface. LPS recognition is initiated by an LPS binding to an LBP protein. This LPS-LBP complex transfers the LPS to CD14 which is a glycosylphosphatidylinositol-anchored membrane protein that binds the LPS-LBP complex and facilitates the transfer of LPS to MD-2 protein, which is associated with the extracellular domain of TLR4. LPS binding promotes the dimerization of TLR4/MD-2 complex. The conformational changes of the TLR4 induce the recruitment of intracellular adaptor proteins containing the TIR domain which is necessary to activate the downstream signaling pathway.

The binding of an LPS molecule to the TLR4/MD-2 complex involves acyl chains and phosphate groups of lipid A, the conserved part of LPS and the main inducer of pro-inflammatory responses to LPS.

TLR4 activation and response to LPS is greatly influenced by the polysaccharide domain and the molecular structure of Lipid A moiety of the LPS molecules. Hexa-acylated and diphosphorylated LPS, like Escherichia coli LPS (O111:B4), is one of the most potent agonists of TLR4 whereas under-acylated LPS and dephosphorylated LPS species have a weaker pro-inflammatory activity especially in human cells.[18]

Structural determinants of this phenomenon are found in the TLR4/MD-2 complex and also in CD14 protein. The polysaccharide portion covalently bound to lipid A plays also an indispensable role in TLR4 activation through CD14/TLR4/MD-2.[20] However, in addition to the lipid A domain, the polysaccharide moiety plays an important role in the binding and activation of the LPS molecules as the lipid A moiety alone was demonstrated to be significantly less active than the full LPS molecule.

Unlike all the other TLRs, TLR4 stimulation triggers two signaling pathways called the MyD88-dependent and the TRIF-dependent one after the adaptor proteins involved in their induction. The MyD88-dependent signaling is triggered by TLR4 localized to the plasma membrane, while the TRIF-dependent one by the TLR4 internalized in endosomes.

These signaling pathways lead to the production of two sets of cytokines. The MyD88-dependent pathway induces the production of pro-inflammatory cytokines while TRIF-dependent pathway induces the production of type I interferons and chemokines.] The molecular structure of TLR4 ligands (in particular LPS), as well as their complexation with proteins or lipids, greatly influence the action of these TLR4-related signaling pathways, leading to different cytokine balances.

Use according to manufacturer's directions.

SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.

Details of the manufacturer or importer of the safety data sheet

Registered company name	JTC Import Export Pty Ltd
Address	98-126 South Park Drive Dandenong South VIC 3175 Australia
Telephone	+61 3 9532 5100
Fax	+61 3 9532 6102
Website	https://www.jtcimportexport.com.au
Email	sales@jtcimportexport.com.au

Emergency telephone number

Association / Organisation	JTC Import Export Pty Ltd	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	+61 3 9532 5100 (Mon-Thurs 8.30am to 5.30pm; Friday 8.30am to 3pm)	+61 1800 951 288 (ID#: 7956-82)
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]	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
7647-14-5	0.6-0.73	<u>sodium chloride</u>
100-51-6	<0.5	<u>benzyl alcohol</u>
7558-79-4	<0.5	<u>sodium phosphate, dibasic</u>
7558-80-7	<0.5	<u>sodium phosphate, monobasic, anhydrous</u>
139-07-1	<0.1	<u>dodecyl dimethylbenzylammonium chloride</u>
7732-18-5	>60	<u>water</u>
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 eyes: ▶ Wash out immediately with water. ▶ If irritation continues, 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, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
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.

SECTION 5 Firefighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

- ▶ foam.
- ▶ dry chemical powder.
- ▶ carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	▶ 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.
Fire/Explosion Hazard	▶ The material is not readily combustible under normal conditions. ▶ However, it will break down under fire conditions and the organic component may burn. ▶ Not considered to be a significant fire risk. ▶ Heat may cause expansion or decomposition with violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material. May emit corrosive fumes.
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

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See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	▶ 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. ▶ 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.

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 all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	▶ Store in original containers. ▶ Keep containers securely sealed. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Avoid contamination of water, foodstuffs, feed or seed.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Ingredient	Original IDLH	Revised IDLH
sodium chloride	Not Available	Not Available
benzyl alcohol	Not Available	Not Available
sodium phosphate, dibasic	Not Available	Not Available
sodium phosphate, monobasic, anhydrous	Not Available	Not Available
dodecyl dimethylbenzylammonium chloride	Not Available	Not Available
water	Not Available	Not Available

Exposure controls

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

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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.

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. 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].

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

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.

· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

· Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Continued...

Personal Care - Nasal Spray – Saline

	Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
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:
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Material	CPI
BUTYL	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
PVA	C
VITON	C

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

Ansell Glove Selection

Glove — In order of recommendation
TouchNTuff® 92-600
TouchNTuff® 92-500
TouchNTuff® 92-605
TouchNTuff® 93-250
TouchNTuff® 93-700
AlphaTec® Solvex® 37-185
AlphaTec® 38-612
AlphaTec® 58-008
AlphaTec® 58-530B
AlphaTec® 58-530W

The suggested gloves for use should be confirmed with the glove supplier.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Colourless liquid with no odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	6-8	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available

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
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

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 Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	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	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	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Not normally a hazard due to non-volatile nature of product
Ingestion	The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence.
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population.

Personal Care - Nasal Spray – Saline	TOXICITY	IRRITATION
	Not Available	Not Available
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]
benzyl alcohol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2000 mg/kg ^[2]	Eye (Rodent - rat): 0.1mL
	Inhalation (Rat) LC50: >4.178 mg/L4h ^[2]	Eye: adverse effect observed (irritating) ^[1]
	Oral (Rat) LD50: 1230 mg/kg ^[2]	Skin (Human - man): 16mg/48H - Mild

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		Skin (Human): 1%/2D
		Skin (Mammal - pig): 100% - Moderate
		Skin (Rodent - rabbit): 100mg/24H - Moderate
		Skin: no adverse effect observed (not irritating) ^[1]
sodium phosphate, dibasic	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >300 mg/kg ^[1]	Eye (Rodent - rabbit): 500mg/24H - Mild
	Inhalation (Rat) LC50: >0.83 mg/l4h ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Oral (Rat) LD50: >500 mg/kg ^[1]	Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating) ^[1]
sodium phosphate, monobasic, anhydrous	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >300 mg/kg ^[1]	Eye (Human): 50mg - Mild
	Inhalation (Rat) LC50: >0.83 mg/l4h ^[1]	Eye (Rodent - rabbit): 150mg - Mild
	Oral (Rat) LD50: >500 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
dodecyl dimethylbenzylammonium chloride	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2730 mg/kg ^[1]	Eye (Rodent - rabbit): 1mg
	Oral (Rat) LD50: 400 mg/kg ^[2]	Skin (Rodent - rabbit): 1mg/24H
		Skin: adverse effect observed (corrosive) ^[1]
water	TOXICITY	IRRITATION
	Oral (Rat) LD50: >90000 mg/kg ^[2]	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	

SODIUM CHLORIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
BENZYL ALCOHOL	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. Adverse reactions to fragrances in perfumes and fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, sensitivity to light, immediate contact reactions, and pigmented contact dermatitis. Airborne and connubial contact dermatitis occurs. Contact allergy is a lifelong condition, so symptoms may occur on re-exposure. Allergic contact dermatitis can be severe and widespread, with significant impairment of quality of life and potential consequences for fitness for work. If the perfume contains a sensitizing component, intolerance to perfumes by inhalation may occur. Symptoms may include general unwellness, coughing, phlegm, wheezing, chest tightness, headache, shortness of breath with exertion, acute respiratory illness, hayfever, asthma and other respiratory diseases. Perfumes can induce excess reactivity of the airway without producing allergy or airway obstruction. Breathing through a carbon filter mask had no protective effect. Occupational asthma caused by perfume substances, such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms, even though the exposure is below occupational exposure limits. Prevention of contact sensitization to fragrances is an important objective of public health risk management. Hands: Contact sensitization may be the primary cause of hand eczema or a complication of irritant or atopic hand eczema. However hand eczema is a disease involving many factors, and the clinical significance of fragrance contact allergy in severe, chronic hand eczema may not be clear. Underarm: Skin inflammation of the armpits may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a skin specialist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy. Face: An important manifestation of fragrance allergy from the use of cosmetic products is eczema of the face. In men, after-shave products can cause eczema around the beard area and the adjacent part of the neck. Men using wet shaving as opposed to dry have been shown to have an increased risk of allergic to fragrances. Irritant reactions: Some individual fragrance ingredients, such as citral, are known to be irritant. Fragrances may cause a dose-related contact urticaria (hives) which is not allergic; cinnamal, cinnamic alcohol and Myroxylon pereirae are known to cause hives, but others, including menthol, vanillin and benzaldehyde have also been reported. Pigmentary anomalies: Type IV allergy is responsible for "pigmented cosmetic dermatitis", referring to increased pigmentation on the face and neck. Testing showed a number of fragrance ingredients were associated, including jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol and geranium oil. Light reactions: Musk ambrette produced a number of allergic reactions mediated by light and was later banned from use in Europe. Furocoumarins (psoralens) in some plant-derived fragrances have caused phototoxic reactions, with redness. There are now limits for the amount of furocoumarins in fragrances. Phototoxic reactions still occur, but are rare. General/respiratory: Fragrances are volatile, and therefore, in addition to skin exposure, a perfume also exposes the eyes and the nose / airway. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. A significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients and hand eczema. Fragrance allergens act as haptens, low molecular weight chemicals that cause an immune response only when attached to a carrier protein. However, not all sensitizing fragrance chemicals are directly reactive, but require previous activation. A prehapten is a chemical that itself causes little or no sensitization, but is transformed into a hapten in the skin (bioactivation), usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prehapten or a prohapten , or both. Prohaptens: Compounds that are bioactivated in the skin and thereby form haptens are referred to prohaptens. The possibility of a prohapten being activated cannot be avoided by outside measures. Activation processes increase the risk for cross-reactivity between

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fragrance substances. Various enzymes play roles in both activating and deactivating prohaptens. Skin-sensitizing prohaptens can be recognized and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or studies of sensitization.

QSAR prediction: Prediction of sensitization activity of these substances is complex, especially for those substances that can act both as pre- and prohaptens.

CYP1A2 is a member of the cytochrome P450 super family, is one of the best characterized. It is responsible for the metabolism of commonly drugs belonging to classes such as antidepressants, antipsychotics, mood stabilizers, beta blockers and sedative/hypnotics. CYP1A2 also metabolises a number of procarcinogens (such as those in cigarettes). Cigarette smoking may lead to three fold increase in 1A2 activity, which explains why smokers require higher doses of beta blockers than non-smokers.

Drugs that inhibit CYP1A2 will predictably increase the plasma concentrations of the medications or decrease in clearance of substrates. Drugs such as ciprofloxacin, fluvoxamine, verapamil, cimetidine, caffeine and isoniazid are inhibitors of CYP1A2 enzyme. Vegetables such as grape fruit juice, cumic and turmeric are inhibitors of the CYP1A2 enzyme which may lead to increase plasma concentration of psychotropics.

Inhibition of NF- κ B in vivo can be detrimental. NF- κ B controls multiple functions in homeostasis including a functional immune response, cell cycle, and cell death. Genetic studies in mice and analysis of naturally occurring mutations in humans point to specific developmental and immune consequences due to altering NF- κ B activity.

The same functions that make NF- κ B attractive for developing inhibitors for treating disease also play a role in homeostasis, and disruption of the NF- κ B pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.

NF- κ B plays a role in multiple homeostatic cellular processes including response to stimuli, cell proliferation, and death, regulating communication between cells, but is also tightly linked with other signaling pathways within the cell, such as p38 and JNK. In addition to mediating proinflammatory responses, NF- κ B may regulate apoptotic and cell cycle changes induced by cellular stress, DNA damage or oncogenes by communication with the tumor suppressor p53. Disruption of normal cellular responses by inhibiting NF- κ B can have adverse consequences such as immune suppression and tissue damage.

Understanding the consequences of lack of NF- κ B activity in adult humans comes from observation of naturally occurring genetic deficiencies in this pathway. Mutations have been discovered in humans in signaling molecules upstream of NF- κ B resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF- κ B activation including IKK gamma (NEMO), a subunit of the IKK complex, and I κ Balpha. The IKK gamma mutations result in a defective IKK complex and the I κ Balpha mutation results in an I κ Balpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF- κ B activation and ectodermal dysplasia with immunodeficiency. In general patients with these genetic defects have multiple immunological defects including impaired innate immunity, impaired antibody production, and ultimately severe bacterial infections.

Understanding the immune defects and susceptibilities in patients with genetic defects in the NF- κ B pathway will help prepare for potential adverse effects of pharmacologic NF- κ B inhibitors.

The requirement for NF- κ B in the development and maintenance of the immune system is well documented. NF- κ B is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF- κ B or the IKKbeta gene results in death during fetal development primarily due to massive liver apoptosis.

Fetal liver stem cells from p65 or IKKbeta deficient mice have been transplanted into irradiated hosts revealing a specific requirement of NF- κ B for T-cells, B-cells, and common lymphoid progenitor development but not for myeloid cells or stem cells. The failure to produce lymphocytes is mediated through hypersensitivity to TNF due to lack of NF- κ B activity. Lymphocyte depletion with chemical or genetic inhibition of NF- κ B have implications for therapeutic potential use in humans. The double-sided nature of NF- κ B inhibition is clear in this instance where chemical inhibition in vivo mimics genetic experiments inducing rapid TNF-dependent apoptosis. Rapid induction of apoptosis may be an advantage for treating some forms of cancer, but at the same time cause depletion of some lymphocyte populations. In addition to controlling lymphocyte development, NF- κ B plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF- κ B which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF- κ B through phosphorylation of CARMA1 by PKC theta and PKC beta respectively, resulting in recruitment and activation of IKK and ultimately expression of genes that control cellular activation, proliferation, and survival. In addition, NF- κ B plays a role in T-cell response to costimulatory signals. Cells respond to pathogenic microorganisms in part through recognition by Toll-like receptors (TLRs). TLR-family members recognize different molecular structures present in microbes and respond by activating signaling pathways including NF- κ B leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF- κ B during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF- κ B is clearly required for normal mature B-cell and T-cell maintenance and function, including regulatory, memory, and natural killer-like T cells. Inhibition of NF- κ B activation in lymphocytes results in defects in growth, survival, and cytokine production and blocks multiple steps in germinal center formation. Given the diverse roles NF- κ B plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF- κ B pathway are susceptible to parasitic and bacterial infection.

The role of NF- κ B in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF- κ B deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF- κ B targeted to the liver have normal liver function, but have severe liver damage after challenge with concanavalin A, a pan-T cell activator. Liver damage occurs due to sustained activation of JNK due to accumulation of reactive oxygen species (ROS) in the absence of normal NF- κ B activation.

The aryl alkyl alcohol (AAA) fragrance ingredients have diverse chemical structures, with similar metabolic and toxicity profiles. The AAA fragrances demonstrate low acute and subchronic toxicity by skin contact and swallowing. At concentrations likely to be encountered by consumers, AAA fragrance ingredients are non-irritating to the skin. The potential for eye irritation is minimal. With the exception of benzyl alcohol, phenethyl and 2-phenoxyethyl AAA alcohols, testing in humans indicate that AAA fragrance ingredients generally have no or low sensitization potential. Available data indicate that the potential for photosensitization is low.

Testing suggests that at current human exposure levels, this group of chemicals does not cause maternal or developmental toxicity. Animal testing shows no cancer-causing evidence, with little or no genetic toxicity. It has been concluded that these materials would not present a safety concern at current levels of use, as fragrance ingredients.

This is a member or analogue of a group of benzyl derivatives generally regarded as safe (GRAS), based partly on their self-limiting properties as flavouring substances in food. In humans and other animals, they are rapidly absorbed, broken down and excreted, with a wide safety margin. They also lack significant potential to cause genetic toxicity and mutations. The intake of benzyl derivatives as natural components of traditional foods is actually higher than the intake as intentionally added flavouring substances.

Unlike benzylic alcohols, the beta-hydroxyl group of the members of benzyl alkyl alcohols contributes to break down reactions but do not undergo phase II metabolic activation. Though structurally similar to cancer causing ethyl benzene, phenethyl alcohol is only of negligible concern due to limited similarity in their pattern of activity.

For benzoates:

Benzyl alcohol, benzoic acid and its sodium and potassium salt have a common metabolic and excretion pathway. All but benzyl alcohol are considered to be unharmed and of low acute toxicity. They may cause slight irritation by oral, dermal or inhalation exposure except sodium benzoate which doesn't irritate the skin. Studies showed increased mortality, reduced weight gain, liver and kidney effects at higher doses, also, lesions of the brains, thymus and skeletal muscles may occur with benzyl alcohol. However, they do not cause cancer, genetic or reproductive toxicity. Developmental toxicity may occur but only at maternal toxic level.

DODECYL DIMETHYLBENZYLAMMONIUM CHLORIDE	For acid mists, aerosols, vapours Test results suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airway from direct exposure to inhaled acidic mists (which also protects the stomach lining from the hydrochloric acid secreted there). Alkyldimethylbenzylammonium chlorides are in the list of dangerous substances of council directive, classified as "harmful in contact with skin and on ingestion", and "corrosive and very toxic to aquatic organisms". It can cause dose dependent skin and eye irritation with possible deterioration of vision, possible sensitisation in those with pre-existing eczema. It does not cause cancer, genetic defect, foetal or developmental abnormality.
WATER	No significant acute toxicological data identified in literature search.
SODIUM CHLORIDE & SODIUM PHOSPHATE, DIBASIC & SODIUM PHOSPHATE, MONOBASIC, ANHYDROUS &	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

Continued...

DODECYL DIMETHYLBENZYLAMMONIUM CHLORIDE	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.			
SODIUM CHLORIDE & BENZYL ALCOHOL & SODIUM PHOSPHATE, DIBASIC	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.			
SODIUM PHOSPHATE, DIBASIC & SODIUM PHOSPHATE, MONOBASIC, ANHYDROUS	The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.			
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					
Personal Care - Nasal Spray – Saline	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
sodium chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	0.004-0.006mg/L	4
	NOEC(ECx)	6h	Fish	0.001mg/L	4
	EC50	72h	Algae or other aquatic plants	20.76-36.17mg/L	4
	EC50	96h	Algae or other aquatic plants	1110.36mg/L	4
	LC50	96h	Fish	1000mg/L	4
benzyl alcohol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	230mg/l	2
	EC50	72h	Algae or other aquatic plants	500mg/l	2
	NOEC(ECx)	336h	Fish	5.1mg/l	2
	EC50	96h	Algae or other aquatic plants	76.828mg/l	2
	LC50	96h	Fish	10mg/l	2
sodium phosphate, dibasic	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	48h	Crustacea	<59mg/L	4
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
sodium phosphate, monobasic, anhydrous	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	NOEC(ECx)	96h	Fish	100mg/l	2
	EC50	72h	Algae or other aquatic plants	>100mg/l	2
	LC50	96h	Fish	>100mg/l	2
dodecyl dimethylbenzylammonium chloride	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	~0.007mg/l	2
	EC50	72h	Algae or other aquatic plants	0.014mg/L	2
	NOEC(ECx)	72h	Algae or other aquatic plants	<=0.001mg/L	2
	LC50	96h	Fish	0.515mg/l	2
water	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

Ingredient	Persistence: Water/Soil	Persistence: Air
sodium chloride	LOW	LOW
benzyl alcohol	LOW	LOW
dodecyl dimethylbenzylammonium chloride	HIGH	HIGH
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium chloride	LOW (LogKOW = 0.54)
benzyl alcohol	LOW (LogKOW = 1.1)
sodium phosphate, dibasic	LOW (LogKOW = -5.8)
sodium phosphate, monobasic, anhydrous	LOW (LogKOW = -3.96)
dodecyl dimethylbenzylammonium chloride	LOW (LogKOW = 2.93)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
sodium chloride	LOW (Log KOC = 14.3)
benzyl alcohol	LOW (Log KOC = 15.66)
dodecyl dimethylbenzylammonium chloride	LOW (Log KOC = 607500)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging 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. - Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. - Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). - Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
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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 chloride	Not Available
benzyl alcohol	Not Available
sodium phosphate, dibasic	Not Available
sodium phosphate, monobasic, anhydrous	Not Available
dodecyl dimethylbenzylammonium chloride	Not Available

Product name	Group
water	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium chloride	Not Available
benzyl alcohol	Not Available
sodium phosphate, dibasic	Not Available
sodium phosphate, monobasic, anhydrous	Not Available
dodecyl dimethylbenzylammonium chloride	Not Available
water	Not Available

SECTION 15 Regulatory information

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

sodium chloride is found on the following regulatory lists
Australian Inventory of Industrial Chemicals (AIIC)
benzyl alcohol is found on the following regulatory lists
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
sodium phosphate, dibasic is found on the following regulatory lists
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 3
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)
sodium phosphate, monobasic, anhydrous is found on the following regulatory lists
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 3
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)
dodecyl dimethylbenzylammonium chloride 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 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
water is found on the following regulatory lists
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	Yes
Canada - NDSL	No (sodium chloride; benzyl alcohol; sodium phosphate, dibasic; sodium phosphate, monobasic, anhydrous; dodecyl dimethylbenzylammonium chloride; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (dodecyl dimethylbenzylammonium chloride)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

Personal Care - Nasal Spray – Saline

SECTION 16 Other information

Revision Date	23/05/2025
Initial Date	23/05/2025

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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