Troy Nutripet High-energy Vitamin concentrate

Troy Laboratories Pty Ltd

Chemwatch Hazard Alert Code: **2**

Chemwatch: **5398-46**
Version No: **4.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **10/03/2023**
Print Date: **31/03/2025**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	Troy Nutripet High-energy Vitamin concentrate
Chemical Name	Not Applicable
Synonyms	Nutrigel High-energy Vitamin concentrate
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	A palatable high-energy dietary supplement for dogs and cats. To be used as directed on product label.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Troy Laboratories Pty Ltd**
Address	37 Glendenning Road Glendenning NSW 2761 Australia
Telephone	02 8808 3600
Fax	02 9677 9300
Website	www.Troylab.com.au
Email	admin@troylab.com.au

Emergency telephone number

Association / Organisation	**Ixom Emergency Response Service**
Emergency telephone number(s)	1800 033 111 (24 hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Sensitisation (Respiratory) Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

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

Page 1 continued...

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.

Precautionary statement(s) Prevention

P261	Avoid breathing mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
8001-22-7	10-30	soybean oil
9000-01-5	1-10	gum arabic
7695-91-2	<1	DL-alpha-tocopherol acetate
532-32-1	<1	sodium benzoate
67-03-8	<1	thiamine hydrochloride
98-92-0	<1	niacinamide
137-08-6	<1	D-pantothenic acid, calcium salt
58-56-0	<1	pyridoxine hydrochloride
79-81-2	<1	retinol palmitate
6184-17-4	<1	riboflavin 5'-monophosphate sodium salt
67-97-0	<1	cholecalciferol
68-19-9	<1	cyanocobalamin
Not Available	balance	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***

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SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the 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	▸ 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 courses. ▸ Use water delivered as a fine spray to control fire and cool adjacent 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	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include: carbon dioxide (CO_2) acrolein hydrogen iodide metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes.
HAZCHEM	Not Applicable

Continued...

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	▸ Clean up all spills immediately. ▸ Avoid contact with skin and eyes. ▸ Wear impervious gloves and safety goggles. ▸ Trowel up/scrape up. ▸ Place spilled material in clean, dry, sealed container. ▸ Flush spill area with water.
Major Spills	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	▸ 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 the dark. Consider storage under inert gas. ▸ 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	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Continued...

Not Available

Ingredient	Original IDLH	Revised IDLH
soybean oil	Not Available	Not Available
gum arabic	Not Available	Not Available
DL-alpha-tocopherol acetate	Not Available	Not Available
sodium benzoate	Not Available	Not Available
thiamine hydrochloride	Not Available	Not Available
niacinamide	Not Available	Not Available
D-pantothenic acid, calcium salt	Not Available	Not Available
pyridoxine hydrochloride	Not Available	Not Available
retinol palmitate	Not Available	Not Available
riboflavin 5'-monophosphate sodium salt	Not Available	Not Available
cholecalciferol	Not Available	Not Available
cyanocobalamin	Not Available	Not Available

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.
The basic types of engineering controls are:
Process controls which involve changing the way a job activity or process is done to reduce the risk.
Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.
Employers may need to use multiple types of controls to prevent employee overexposure.

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

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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
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:
Troy Nutripet High-energy Vitamin concentrate

Material	CPI
NATURAL RUBBER	A
NATURAL+NEOPRENE	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.

Ansell Glove Selection

Glove — *In order of recommendation*
AlphaTec® Solvex® 37-675
MICROFLEX® 63-864
MICROFLEX® 93-244
MICROFLEX® 93-252
MICROFLEX® 93-260
MICROFLEX® 93-843
MICROFLEX® 93-833
MICROFLEX® Blaze® N48
MICROFLEX® 93-856
MICROFLEX® 93-853

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

Respiratory protection

Type A-P 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 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)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Shiny brown thick homogeneous gel with yeast, caramel odour; does not mix with water.

Physical state	Gel	**Relative density (Water = 1)**	Not Available

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Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Applicable	**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 Available	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Immiscible	**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
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	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
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	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.

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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. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.
Skin Contact	The material produces moderate skin irritation; evidence exists, or practical experience predicts, that the material either ▸ produces moderate inflammation of the skin in a substantial number of individuals following direct contact, and/or ▸ produces significant, but moderate, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing dermatitis condition Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Respiratory sensitisation may result in allergic/asthma like responses; from coughing and minor breathing difficulties to bronchitis with wheezing, gasping.

Troy Nutripet High-energy Vitamin concentrate	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
soybean oil	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
gum arabic	**TOXICITY**	**IRRITATION**
	Oral (Rabbit) LD50; 8000 mg/kg[2]	Eye (Rodent - rabbit): 36mg/5H - Severe
DL-alpha-tocopherol acetate	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >3000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
	Oral (Mouse) LD50; >49700 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
sodium benzoate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Inhalation (Rat) LC50: >12.2 mg/L4h[1]	Skin (Human): 0.5%/20M
	Oral (Rat) LD50: 4070 mg/kg[2]	Skin (Human): 10%/1H
		Skin: no adverse effect observed (not irritating)[1]

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thiamine hydrochloride	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 3710 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
niacinamide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[2]	Eye (Rodent - rabbit): 100mg
	Inhalation (Rat) LC50: >3.8 mg/l4h[1]	Eye (Rodent - rabbit): 100mg - Severe
	Oral (Rat) LD50: >2500 mg/kg[1]	Eye (Rodent - rabbit): 280mg/7D - Moderate
		Eye: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
D-pantothenic acid, calcium salt	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10000 mg/kg[2]	Not Available
pyridoxine hydrochloride	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 4000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
retinol palmitate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >2000 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Human - woman): 0.55%/1W (intermittent)
		Skin: adverse effect observed (irritating)[1]
riboflavin 5'-monophosphate sodium salt	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
cholecalciferol	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 42 mg/kg[2]	Not Available
cyanocobalamin	**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***

SOYBEAN OIL

Refined grades are edible. Non irritant.
For omega 6 fatty acids and derivatives:
Some medical research suggests that excessive levels of certain omega-6 fatty acids relative to certain omega-3 fatty acids may increase the probability of a number of diseases.
Modern Western diets typically have ratios of omega-6 to omega-3 in excess of 10 to 1, some as high as 30 to 1; the average ratio of omega-6 to omega-3 in the Western diet is 15:1–16.7:1. Humans are thought to have evolved with a diet of a 1-to-1 ratio of omega-6 to omega-3 and the optimal ratio is thought to be 4 to 1 or lower although some sources suggest ratios as low as 1:1). A ratio of 2–3:1 omega 6 to omega 3 helped reduce inflammation in patients with rheumatoid arthritis. A ratio of 5:1 had a beneficial effect on patients with asthma but a 10:1 ratio had a negative effect. A ratio of 2.5:1 reduced rectal cell proliferation in patients with colorectal cancer, whereas a ratio of 4:1 had no effect.
Excess omega-6 fatty acids from vegetable oils interfere with the health benefits of omega-3 fats, in part because they compete for the same rate-limiting enzymes. A high proportion of omega-6 to omega-3 fat in the diet shifts the physiological state in the tissues toward the pathogenesis of many diseases: prothrombotic, proinflammatory and proconstrictive.
Chronic excessive production of omega-6 eicosanoids is correlated with arthritis, inflammation, and cancer. Many of the medications used to treat and manage these conditions work by blocking the effects of the COX-2 enzyme. Many steps in formation and action of omega-6 prostaglandins from omega-6 arachidonic acid proceed more vigorously than the corresponding competitive steps in formation and action of omega-3 hormones from omega-3 eicosapentaenoic acid The COX-1 and COX-2 inhibitor medications, used to treat inflammation and pain, work by preventing the COX enzymes from turning arachidonic acid into inflammatory compounds.The LOX inhibitor medications often used to treat asthma work by preventing the LOX enzyme from converting arachidonic acid into the leukotrienes. Many of the anti-mania medications used to treat bipolar disorder work by targeting the arachidonic acid cascade in the brain.
A high consumption of oxidised polyunsaturated fatty acids (PUFAs), which are found in most types of vegetable oil, may increase the likelihood that postmenopausal women will develop breast cancer. Similar effect was observed on prostate cancer, but the study was performed on mice Another "analysis suggested an inverse association between total polyunsaturated fatty acids and breast cancer risk, but individual polyunsaturated fatty acids behaved differently [from each other]. [...] a 20:2 derivative of linoleic acid [...] was inversely associated with the risk of breast cancer"
PUFAs are prone to spontaneous oxidation/ peroxidation. The feeding of lipid oxidation products and oxidised fats has been reported to cause adverse biological effects on laboratory animals, including growth retardation, teratogenicity, tissue damage

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and increased liver and kidney weights, as well as cellular damage to the testes and epididymes, increased peroxidation of membrane and tissue lipids and induction of cytochrome P450 activities in the colon and liver.
The propensity for PUFAs to oxidise leads to the generation of free radicals and eventually to rancidity.
Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of PUFAs. Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease.
Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during “on-site” frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein.
The end products of lipid peroxidation are reactive aldehydes, such as malondialdehyde (MDA) and 4-hydroxynonenal (HNE), the second one being known also as "second messenger of free radicals" and major bioactive marker of lipid peroxidation, due to its numerous biological activities resembling activities of reactive oxygen species. end-products of lipid peroxidation may be mutagenic and carcinogenic malondialdehyde reacts with deoxyadenosine and deoxyguanosine in DNA, forming DNA adducts. Malondialdehyde produces mutagenic effects in several bioassays.
Side products of lipid peroxidation can also exert toxic effects, even at sites distant from the primary oxidation site. Such products (typically malondialdehyde and a large group of hydroxyalkenals - alpha-beta-unsaturated aldehydes) may interact with protein thiols (producing intermolecular cross-links) and, as a result produce functional impairment to enzyme systems, receptors and structural proteins. Aldehydes may also inhibit protein biosynthesis and increase osmotic fragility of lysosymes (releasing hydrolytic enzymes) and other subcellular organelles. They may also react with nucleic acids.
The toxicity of lipid hydroperoxides to animals is best illustrated by the lethal phenotype of glutathione peroxidase 4 (GPX4) knockout mice. These animals do not survive past embryonic day 8, indicating that the removal of lipid hydroperoxides is essential for mammalian life.
Peroxidised linoleic acid applied to the shaved skin of guinea pigs, in a patch test experiment, produced necrosis and bleeding. When the abdominal skin of guinea pig was patched for 8 days with a cream containing 25 nmol (in terms of malondialdehyde) of lipid peroxides per gram, a thickening of the epidermis was found
Lipid peroxidation in cellular membranes may produce several morphological alterations resulting, for example, in membrane aggregation, deformation or breakage. This may result in the release of hydrolytic enzymes which in turn may degrade functional macromolecules and cause secondary damage. In addition membrane-bound enzyme systems may be disrupted.
Epoxidation of double bonds is a common bioactivation pathway for alkenes. The allylic epoxides, so formed, were found to possess sensitising capacity in vivo and in vitro and to chemically reactive towards a common hexapeptide containing the most common nucleophilic amino acids. Further-more, a SAR study of potentially prohaptenic alkenes demonstrated that conjugated dienes in or in conjunction with a six-membered ring are prohaptens, whereas related alkenes containing isolated double bonds or an acyclic conjugated diene were weak or nonsensitizing compounds.This difference in sensitizing capacity of conjugated dienes as compared to alkenes with isolated double bonds was found to be due to the high reactivity and sensitizing capacity of the allylic epoxides metabolically formed from conjugated dienes.
Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers.
Ann-Therese Karlberg et al: Chem. Res. Toxicol. 2008, 21, pp 53–69
https://ftp.cdc.gov/pub/Documents/OEL/06.%20Dotson/References/Karlberg_2008.pdf
For Group E aliphatic esters (polyol esters):
According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group E substances are esters of monoacids, mainly common fatty acids, and trihydroxy or polyhydroxyalcohols or polyols, such as pentaerythritol (PE), 2-ethyl-2-(hydroxymethyl)- 1,3-propanediol or trimethylolpropane (TMP), and dipentaerythritol (diPE). The Group E substances often are referred to as "polyol esters" The polyol esters are unique in their chemical characteristics since they lack beta-tertiary hydrogen atoms, thus leading to stability against oxidation and elimination. The fatty acids often range from C5-C10 to as high as C18 (e.g., oleic, stearic, isostearic, tall oil fatty acids) in carbon number and generally are derived from naturally occurring sources. Group E esters may have multiple ester linkages and may include mixed esters derived from different carbon-length fatty acid mixtures. The lack of beta-tertiary hydrogen atoms in the structure of the polyol esters makes them characteristically and chemically stable against oxidation and elimination in comparison to other ester classes or groups. For these reasons, trimethylolpropane (TMP) and pentaerythritol (PE) esters with fatty acids of C5 to C10 carbon-chain length have applications as synthetic lubricants for passenger car motor oil and military and civilian jet engines. TMP and PE esters of C18 acids (e.g., isostearic and oleic acids) also have found use in synthetic lubricant applications, including refrigeration lubricants and hydraulic fluids. Because of their higher thermal stability characteristics, they also find use in a variety of high temperature applications such as industrial oven chain oils, high temperature greases, fire resistant transformer coolants and turbine engines
Polyol esters that are extensively esterified also have greater polarity, less volatility and enhanced lubricity characteristics. **Acute toxicity:** Depending on the degree of esterification, the polyol esters can be resistant or slow towards chemical or enzymatic hydrolysis (i.e., esterase or lipases) as a result of steric hindrance. PE and diPE esters that are capable of being enzymatically hydrolyzed will generate pentaerythritol or dipentaerythritol, and the corresponding fatty acids which, for most of the Group E esters, are comprised mainly of oleic, linoleic and stearic acids as well as the fatty acids in the C5-10 carbon-length. Similarly, TMP esters can undergo metabolism to yield trimethylolpropane (2-ethyl-2-hydroxymethyl-1,3-propanediol) and fatty acid constituents. Pentaerythritol and trimethylolpropane have been reported to have a low order of toxicity The acute oral LD50 for these substances was greater than 2000 mg/kg indicating a relatively low order of toxicity. The similarity in the low order of toxicity for these substances is consistent with their similar chemical structure and physicochemical properties.
Metabolic studies of polyglyceryl esters indicated that these esters are hydrolyzed in the gastrointestinal (GI) tract, and utilization and digestibility studies supported the assumption that the fatty acid moiety is metabolized in the normal manner. Analytical studies have produced no evidence of accumulation of the polyglycerol moiety in body tissues.
In an acute dermal toxicity study in rats, the LD50 of 1,2,3-propanetriol, homopolymer, diisooctadecanoate was>5000 mg/kg Low toxicity was reported in acute oral studies. In rats, the LD50 >2000 mg/kg for polyglyceryl-3 caprate, polyglyceryl-3 caprylate, polyglyceryl-4 caprate, diisostearoyl polyglyceryl-3 dimer dilinoleate, and the LD50 was >5000 mg/kg for polyglyceryl-3 iso-stearate, polyglyceryl-3-oleate, polyglyceryl-2 diisostearate and polyglyceryl-3 diisostearate.
The ability to enhance skin penetration was examined for several of the polyglyceryl fatty acid esters.
Repeat dose toxicity: Polyol esters are generally well tolerated by rats in 28-day oral toxicity studies. NOAEL for these substances was 1000 mg/kg/day in Sprague-Dawley rats. The TMP ester of heptanoic and octanoic acid did not produce signs of overt systemic toxicity at any dose levels tested (i.e., 100, 300, and 1000 mg/kg/day). There were no treatment-related clinical in-life, functional observation battery, or gross postmortem findings. There were no treatment related mortality, and no adverse effects on body weight, food consumption, clinical laboratory parameters, or organ weights. However, there were increased

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numbers of hyaline droplets in the proximal cortical tubular epithelium of the 300 and 1000 mg/kg/day in male rats. Based on these findings (hyaline droplets), the NOAEL for this polyol ester
was established at 100 mg/kg/day for male rats. Hyaline droplet formation observed in the male kidneys is believed to be a sex/species condition specific to only male rats, which has little relevance to humans.
The results from these repeated dose dermal toxicity studies suggest that polyol esters exhibit a low order of toxicity following repeated application. This may be attributable to similarities in their chemical structures, physicochemical properties, and common metabolic pathways (i.e., esters can be enzymatically hydrolyzed to the corresponding polyalcohol and the corresponding fatty acids) The polyol, hexanedioic acid, mixed esters with decanoic acid, heptanoic acid, octanoic acid and PE, was applied to the skin of groups of 10 (male and female) rats for five days a week for four (4) weeks at dose levels of 0, 125, 500 and 2000 mg/kg/day. Treated animals exhibited no signs indicative of systemic toxicity. No visible signs of irritation were observed a treatment sites. Microscopically, treated skin (viz., greater than or equal to 500 mg/kg/day) exhibited a dose-related increased incidence and severity of hyperplasia and hyperkeratosis of the epidermis and sebaceous gland hyperplasia. These effects were reversible. None of the minor changes in haematology and serum chemistry parameters were considered biologically significant. High dose females (2000 mg/kg/day) exhibited a significant increase in relative adrenal and brain weights when compared to the controls. These differences were attributed to the lower final body weight of the female animals. The NOAEL in this study for systemic toxicity was established as 500 mg /kg/day and 125 mg/kg/day for skin irritation.
Two 28-day study conducted with fatty acids, C5-10, esters with pentaerythritol (CAS RN: 68424-31-7) and dipentaerythritol ester of n-C5/iso-C9 acids (CAS RN: 647028-25-9) showed no signs of overt toxicity. The 90-day study pentaerythritol ester of pentanoic acids and isononanoic acid (CAS RN: 146289-36-3) did not show any signs of overt toxicity. However, increased kidney and liver weights in the male animals was observed. In conclusion, since the effects observed are not considered to be systemic and relevant for humans, the NOAEL was found to exceed 1000 mg/kg bw for all substances based on the result from the 28 and 90-day studies.
Reproductive and developmental toxicity: Since metabolism of the polyol esters can occur, leading to the generation of the corresponding fatty acids and the polyol alcohol (such as pentaerthyritol, trimethylolpropane, and dipentaerythritol), the issue of whether these metabolites may pose any potential reproductive/developmental toxicity concerns is important.. However, the polyol alcohols such as pentaerthyritol, trimethylolpropane, and dipentaerythritol, would be expected to undergo further metabolism, conjugation and excretion in the urine. Available evidence indicates that these ester hydrolysates (i.e., hydrolysis products), primarily fatty acids (e.g., heptanoic, octanoic, and decanoic acids) and secondarily the polyol alcohols should exhibit a low order of reproductive toxicity. it can be concluded that this group of high molecular weight polyol esters should not produce profound reproductive effects in rodents.
Genotoxicity: Polyols tested for genetic activity in the Salmonella assay, have been found to be inactive. Several polyol esters have been adequately tested for chromosomal mutation in the in vitro mammalian chromosome aberration assay, and all were inactive. Two TMP esters were also tested for in vivo chromosomal aberration in rats, and both demonstrated no activity. Thus, it is unlikely that these substances are chromosomal mutagens.
Carcinogenicity: In a 2-yr study, 28 male and 28 female rats were fed 5% polyglyceryl ester in the diet. No adverse effects on body weight, feed consumption, haematology values, or survival rate were noted. Liver function tests and renal function tests performed at 59 and 104 wks of the study were comparable between the test group and a control group fed 5% ground nut oil. The carcass fat contained no polyglycerol, and the levels of free fatty acid, unsaponifiable residue and fatty acid composition of carcass fat were not different from the controls. Organ weights, tumour incidence and tumour distribution were similar in control and test groups. A complete histological examination of major organs showed nothing remarkable
For polyunsaturated fatty acids and oils (triglycerides)
Studies on animals have shown a link between polyunsaturated fat and the incidence of tumours. In some of these studies the incidence of tumours increased with increasing intake of polyunsaturated fat, up to about 5% of total energy, near to the middle of the current dietary intake in humans.
The propensity for polyunsaturated fats to oxidise is another possible risk factor. This leads to the generation of free radicals and eventually to rancidity
Research evidence suggests that consuming high amounts of polyunsaturated fat may increase the risk of cancer spreading. Researchers found that linoleic acid in polyunsaturated fats produced increasing membrane phase separation, and thereby increased adherence of circulating tumour cells to blood vessel walls and remote organs.
At least one study in mice has shown that consuming high amounts of polyunsaturated fat (but not monounsaturated fat) may increase the risk of metastasis in cancer.
Lipid peroxides with complex components can damage macromolecules, such as DNA, proteins, and membrane lipids. Some components of lipid peroxides, for example, 4,5(E)-epoxy-2(E)-heptenal (EH) can react with L-lysine and damage proteins . 4,5-epoxy-2-alkenals can react with phenylalanine and cause strecker-type degradation of amino acids. Autoxidized methyl linoleate can decrease DNA synthesis in thymocytes Animals consuming oxidized lipids suffered a wide array of biological consequences, such as decreased feed utilization and performance, oxidative stress and tissue lipid oxidation and, most strikingly, adverse effects on redox indices and shelf life of meat. This manifested in malondialdehyde (MDA) content reduced activities of antioxidant enzymes and elevated transcript levels of oxidative stress-responsive genes
The intestinal mucosa is directly exposed to oxidized fatty acids of dietary origin and this tissue readily experiences redox imbalances and oxidative stress after the ingestion of large amounts of oxidized fat . As the first line of defense, the intestines with abundant gut-associated lymphoid tissues (GALTs) and lymphocytes play an important role in immune defense. The immune response in the intestinal tract is complex and is impaired by any damage to the mucosal barrier. When oxidative stress of the intestines caused by oxidized fat occurs, its immune competence and responsiveness may be compromised by the peroxides they contain
When body insulin levels are low, fatty acids flow from the fat cells into the bloodstream and are taken up by various cells and metabolised in a process called beta-oxidation. The end result of beta-oxidation is a molecule called acetyl-coA, and as more fatty acids are released and metabolised, acetyl-coA levels in the cells rise. Liver cells shunt excess acetyl-coA into "ketogenesis", or the making of ketone bodies. When the rate of synthesis of ketone bodies exceeds the rate of utilisation, their concentration in blood increases; this is known as ketonaemia. This is followed by ketonuria – excretion of ketone bodies in urine. The overall picture of ketonaemia and ketonuria is commonly referred as ketosis. Smell of acetone in breath is a common feature in ketosis
For polyunsaturated fatty acids and oils (triglycerides), products of heating and recycling.*
Culinary oils, when heated, undergo important chemical reaction involving self-sustaining, free radical-mediated oxidative deterioration of polyunsaturated fatty acids (PUFAs). Such by-products may be cytotoxic, mutagenic, reproductive toxins and may produce chronic disease.
Saturated fatty acid (SFA)-rich fats also undergo such reactions but to a substantially lower degree.

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Samples of repeatedly used oils collected from fast-food retail outlets and restaurants have confirmed the production of aldehydic lipid oxidation products (LOPs, active aldehydes) at levels exceeding 10 exp-2 moles per kilogram (mol/kg) during “on-site” frying episodes. Volatile emissions from heated culinary oils used in Chinese-style cooking are mutagenic; exposure to such indoor air pollution may render humans more susceptible to contracting lung or further cancers, together with rhinitis and diminished lung function. The high temperatures used in standard (especially Chinese) frying result in fumes that are rich in volatile LOPs, including acrolein.
Teratogenic actions. In principle, if aldehydic LOPs induce DNA and chromosomal damage during embryo development, foetal malformations may arise. A study was conducted to investigate the ability of the chain-breaking antioxidant a-tocopherol (a-TOH, vitamin E) to prevent the teratogenic effects of uncontrolled diabetes mellitus in rats (a study based on the hypothesis that diabetic animals have an elevated level of oxidative stress and therefore in vivo lipid peroxidation when expressed relative to that of healthy controls). It found that a PUFA-rich culinary oil (which served as a vehicle for oral administration of a-TOH) increased the rate of malformations and reabsorptions in both normal and diabetic pregnancies. Further investigations revealed that safflower oil subjected to thermal stressing episodes (according to standard frying practices for a period of 20 minutes) markedly enhanced its teratogenic effects. That is, the evidence indicates that the LOPs therein are primarily responsible for these actions.
Further adverse health effects of dietary LOPs. Further documented health effects of LOPs include their pro-inflammatory and gastropathic properties (for the latter, oral administration of the LOP, 4-hydroxy-trans-2-nonenal -HNE- to rats at a dose level of only 0.26 umol·dm-3, a level similar to that of healthy human blood plasma, induced peptic ulcers), and also a significant elevation in systolic blood pressure and an impaired vasorelaxation observed in rats fed pre-heated soy oil
Oxidative degradation process involving culinary oils, can generate extremely toxic conjugated lipid hydroperoxydienes (CHPDs). These are unstable at standard frying temperatures (ca. 180 degrees C) and are degraded to a broad range of secondary products, particularly saturated and unsaturated aldehydes, together with di- and epoxyaldehydes. Such aldehydic fragments also have toxicological properties in humans owing to their high reactivity with critical biomolecules in vivo (proteins such as low-density lipoprotein, amino acids, thiols such as glutathione, DNA, etc.). Despite their reactivities, high levels of CHPDs can remain in PUFA-rich oils which have been subjected to routine frying practices.
Thermally stressed PUFA-containing culinary oils contain high levels of alpha,beta-unsaturated aldehydes (including trans-2-alkenals, and cis,trans- and trans,trans-alka-2,4-dienals, the latter including the mutagen trans,trans-2,4-decadienal), and n-alkanals, together with their CHPD and hydroxydiene precursors .
Toxicological and pathogenic properties of dietary LOPS
Potential influence of dietary LOPS on metabolic pathways. As a consequence of their absorption from the gut into the systemic circulation, LOPs may penetrate cellular membranes, allowing their entry into particular intracellular sites/organelles where many critical metabolic processes occur. Literature evidence indicates that feeding thermally stressed or repeatedly used culinary oils to experimental animals induces significant modifications to key liver microsomal pathways and to the mitochondrial respiratory chain, for example. These effects are likely to occur via reactions of LOPs with key enzymes (and more especially their active sites), for example, the oxidation of active methioninyl and cysteinyl residues by CHPDs, or alteration of critical side-chain amino acid amine or thiol groups with aldehydes via Schiff base or Michael addition reactions.
Atherosclerosis. Investigations have revealed that dietary derived LOPs can accelerate all three stages of the development of atherosclerosis (i.e., endothelial injury, accumulation of plaque, and thrombosis). Animal studies have shown that diets containing thermally stressed, PUFA-laden (and hence LOP-rich) oils exhibit a greater atherogenicity than those containing unheated ones .
Because cytotoxic aldehydes can be absorbed, they have the capacity to attack and structurally alter the apolipoprotein B component of low density lipoproteins (LDLs). This mechanism can engender uptake of lipid-loaded LDLs by macrophages, which, in turn, transforms them to foam cells, the accumulation of which is responsible for the development of aortic fatty streaks, a hallmark of the aetiology of atherosclerosis and its pathological sequelae. More recently, our co-investigators found that aldehydic LOPs elevated the expression of the CD36 scavenger receptor of macrophages, a phenomenon that also promotes this process .
Mutagenic and carcinogenic properties. Since they are powerful electrophilic alkylating agents, alpha,beta-unsaturated aldehydes can covalently modify DNA base units via a mechanistically complex process that may involve their prior epoxidation in vivo.Such chemically altered bases may therefore be of mutagenic potential. Additionally, these LOPs can inactivate DNA replicating systems, a process that can, at least in principle, elevate the extent of DNA damage. Hence, following cellular uptake, such aldehydes have the potential to cause both DNA and chromosomal damage.
Malondialdehyde (MDA) is also generated by thermally stressing culinary oils, although at concentrations much lower than those of the more reactive alpha,beta-unsaturated aldehydes. MDA and other aldehydes arising from lipid peroxidation (especially acrolein) present a serious carcinogenic hazard. Indeed, adenomas and carcinomas of the thyroid gland, together with adenomas of the pancreatic islet cells, were induced in rats by MDA in a prolonged gavage study; nasal and laryngeal cancers arose in rats and hamsters, respectively, during long-term acetaldehyde inhalation experiments. Hence, both these aldehydes satisfied the NIOSH criteria for classification as carcinogens, and therefore it has set exacting limits for their occupational exposure.
The most obvious solution to the generation of LOPs in culinary oils during frying is to avoid consuming foods fried in PUFA-rich oils as much as possible. Indeed, consumers, together with those involved in the fast-food sector, could employ culinary oils of only a low PUFA content, or mono-unsaturated fatty acids (MUFA) such as canola (a variety of rape seed oil), olive oil, (both oils are rich in oleic acid) selected palm oils (rich in palmitic acid), or coconut oils (an SFA alternative rich in lauric and myristic acids) - for frying MUFAs such as oleoylglycerol adducts are much more resistant to peroxidative degradation than are PUFAs , and hence markedly lower levels of only selected classes of aldehydes are generated during frying.
Previous studies that investigated the prospective health effects or benefits of dietary PUFAs (i.e., those involving feeding trials with humans or animals or, alternatively, related epidemiological ones) should be scrutinized. With hindsight, it seems to us that many of these experimental investigations were flawed since, in addition to some major design faults, they failed to take into account or even consider the nature and concentrations of any cytotoxic LOPs present in the oils or diets involved. Similarly, corresponding epidemiological (or meta-analysis-based) investigations incorporated only the (estimated) total dietary intake of selected PUFAs and further fatty acids, and ignored any LOPs derived or derivable from frying/cooking. Even if PUFA containing culinary oils are unheated, it is virtually impossible to rule out the presence of traces of LOPs within them (analysis of apparently pure PUFAs or their corresponding triglycerides obtained from reputable commercial sources has revealed that these materials contain traces of CHPDs and/or aldehydes
As expected, the levels of total aldehydes generated increase proportionately with oil PUFA content, and over half are the more highly cytotoxic alpha,beta-unsaturated classes, which include acrolein and 4-hydroxy-trans-2-nonenal (HNE), as well as 4-hydroperoxy-, 4-hydroxy-, and 4,5-epoxy-trans-2-alkenals. Total alpha,beta-unsaturated aldehyde concentrations in culinary oils (heated at 180 deg C for 30-90 minutes or longer) are often higher than 20 mmol/kg and can sometimes approach 50 mmol/kg. Furthermore, relatively low concentrations of detectable aldehydes and their CHPD precursors are even found in newly purchased unheated culinary oils.

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	Acrylamide (which can exert toxic effects on the nervous system and fertility, and may also be carcinogenic) can also arise from an acrolein source when asparagine-rich foods are deep-fried in PUFA-rich oils. The levels of acrylamide generated in foods during high-temperature cooking/frying processes are substantially lower than those recorded for aldehydes formed in PUFA-rich culinary oils during frying episodes (to date, the very highest reported levels are only ca. 4 ppm, equivalent to 56 umol/kg). Acrolein is just one of the alpha,beta-unsaturated aldehydes generated in thermally stressed PUFA-rich oils: Many others generated in this manner have comparable toxicological properties The foregoing considerations exclude possible toxicological properties of their isomeric CHPD precursors (also present in the high millimolar range in thermally stressed oils) in a typical fried food meal. Indeed, in one early investigation, a single intravenous dose of methyl linoleate hydroperoxide (20 mg/kg) administered to rats gave rise to a high mortality within 24 hours (animals dying from lung damage), although a higher dose given orally was without effect. This observation may reflect the limited in vivo absorption of these particular aldehyde precursors, in contrast to the known absorption of aldehydes. Furthermore, with regard to the risk of inhalation of aldehydes volatilised during frying practices by humans, the maximum US Occupational Safety and Health (OSHA) permissible exposure limit (PEL) for acrolein, which is an (atmospheric) level of 0.1 ppm (equivalent to only 1.8 umol/kg in the fried food model) for a time-weighted long-term (8 hour) exposure, and 0.3 ppm (5.4 umol/kg)for a short-term (15 minute) one. This 15-minute exposure time can be considered to be less than the time taken to consume a typical fried meal The concentrations of aldehydes generated in culinary oils during episodes of heating at 180 deg C represent only what remains in the oil: Owing to their low boiling points, many of the aldehydes generated are volatilized at standard frying temperatures. These represent inhalation health hazards, in view of their inhalation by humans, especially workers in inadequately ventilated fast-food retail outlets. The composition and content of hazardous LOPs available in fried foods depend on the identity of the frying/cooking oil and its PUFA content, the frying conditions employed, the length of the frying process, exposure of the frying medium to atmospheric oxygen, the reactivities of these agents with a range of other biomolecules (e.g., amino acids and proteins), and, to a limited extent, the antioxidant content of the frying matrix. Experiments have shown that shallow frying gives rise to much higher levels of LOPs than deep frying under the same conditions (reflecting the influence of the surface area of the frying medium, its exposure to atmospheric oxygen, and the subsequent dilution of LOPs generated into the bulk medium). In vivo absorption of dietary LOPs Except for direct damage to the gastrointestinal epithelium, the toxicological actions exerted by LOPs depend on their rate and extent of absorption from the gut into the systemic circulation where they may cause damage to essential organs, tissues, and cells. Experiments in rats have demonstrated that trans-2-alkenals, which are generated in PUFA-containing culinary oils during thermal stressing episodes, are absorbed . Following absorption, these cytotoxic agents are metabolized by a process involving the primary addition (Michael addition reaction) of glutathione across their electrophilic carbon-carbon double bonds and finally excreted in the urine as C-3 mercapturate derivatives. * Martin Grootveld, Victor Ruiz Rodado, and Christopher J.L. Silwood Detection, monitoring, and deleterious health effects of lipid oxidation products generated in culinary oils during thermal stressing episodes American Oil Chemists Society, 25 (10), pp. 614-624. November/December 2014
GUM ARABIC	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. Gum arabic is a technical name for Acacia Senegal Gum. Gum arabic is comprised of various sugars and glucuronic acid residues in a long chain of galactosyl units with branched oligosaccharides. Gum arabic is generally recognized as safe as a direct food additives. Toxicity data on gum arabic indicates little or no acute, short-term, or subchronic toxicity. Gum arabic is negative in several genotoxicity assays, is not a reproductive or developmental toxin, and is not carcinogenic when given intraperitoneally or orally. Clinical testing indicated some evidence of skin sensitization with gum arabic. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
DL-ALPHA-TOCOPHEROL ACETATE	May cause skin and eye irritation * Reproductive and mutagenic effects have been observed in tests with laboratory animals * * Alfa Aeser MSDS Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans. alpha-Tocopherol was non-mutagenic and non-carcinogenic, and the results of reproduction/ teratology studies did not indicate that alpha-tocopherol had adverse effects on reproductive function. However, in a long-term study in rats, a no-effect level could not be established with respect to effects on blood clotting and liver histology, and there was evidence from human studies that excessive intakes of alpha-tocopherol could cause haemorrhage. Other adverse effects noted in clinical studies at doses of > 720 mg alpha-tocopherol/day included weakness, fatigue, creatinuria and effects on steroid hormone metabolism. Clinical studies indicate that, generally, intakes of below about 720 mg/day are without adverse effects in man, but one investigation in elderly patients showed an increase in serum cholesterol at doses of 300 mg alpha-tocopherol daily. Incidences of allergic reactions seem to be very rare. alpha-Tocopherol may be an essential nutrient. The U.S. National Academy of Sciences/National Research Council has recommended a dietary allowance of 0.15 mg/kg b.w./day. However, excessive intakes of alpha-tocopherol produce adverse clinical and biochemical effects, and self-medication with large doses of vitamin E preparations could present a hazard. The previously-allocated ADI was amended to include a lower value, which reflects the fact that alpha-tocopherol may be an essential nutrient. The upper value, which represents the maximum value for the AID, is based on clinical experience in man. IPCS Inchem: https://www.inchem.org/documents/jecfa/jecmono/v21je05.htm **NOTE:** Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

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SODIUM BENZOATE	NOTE: Oral doses of 8-10g may cause nausea and vomiting, though tolerance in human is 50 g/day. Use in food limited to 0.1%. [ICI] For benzoates: **Acute toxicity:** Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol. The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds. Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye. **Sensitisation:** The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers. **Repeat dose toxicity:** For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed. For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur. **Mutagenicity:** All chemicals showed no mutagenic activity in ***in vitro*** Ames tests. Various results were obtained with other ***in vitro*** genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity ***in vivo.*** While some mixed and/or equivocal ***in vitro*** chromosomal/chromatid responses have been observed, no genotoxicity was observed in the ***in vivo*** cytogenetic, micronucleus, or other assays. The weight of the evidence of the ***in vitro*** and ***in vivo*** genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies. In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts. **Developmental toxicity:** In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.
NIACINAMIDE	Mutation in microorganisms The intestinal cytochrome P-450 3A4 system, is responsible for the first-pass metabolism of many medications. Through the inhibition of this enzyme system, inhibitors interact with a variety of medications, leading to elevation of their serum concentrations. Most notable are its effects on cyclosporine, some 1,4-dihydropyridine calcium antagonists, and some 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors. In the case of some drugs, these increased drug concentrations have been associated with an increased frequency of dose-dependent adverse effects. The P-glycoprotein pump, located in the brush border of the intestinal wall, also transports many cytochrome P-450 3A4 substrates, and this transporter also may be affected by CYP3A4 inhibitors.. Most calcium channel blockers (CCBs) are metabolized by CYP3A4 and will be affected by strong inhibitors and inducers of CYP3A4 . Grapefruit juice in sufficient quantities can block intestinal CYP3A4, which can lead to an enhancement of the effects of CCBs. This could affect the blood pressure response for all CCBs Common classes of drugs that are strong inhibitors of CYP3A4 include azole antifungals, macrolide antibiotics (except azithromycin), protease inhibitors used for HIV, amiodarone, diltiazem, and verapamil. Inhibition of several HDACs simultaneously confers greater toxicity and long term side effects. Therefore discovery of isoform-selective HDACs, improve therapeutic potential. The use of histone deacetylase (HDAC) inhibitors (HDIs) as monotherapies for various solid malignancies demonstrate that they are well tolerated with good toxicity profiles compared to current standard cancer therapies. In general, the side effects of HDAC inhibitors are reversible with drug cessation and primarily include fatigue, nausea, dehydration, diarrhea, prolonged QT, thrombocytopenia, lymphopenia, and neutropenia. When used as monotherapies in solid cancers, HDAC inhibitors did not tend to significantly improve results compared to current standard therapies. Most preclinical studies, particularly in solid malignancies, have observed a cytostatic response to HDAC inhibitors when used as monotherapies, but when combined with radiation, chemotherapy, or other targeted agents, these drugs produce a more powerful cytotoxic response. Although this family comprises chemical compounds from unrelated chemical classes that have different HDAC isoform specificities, they surprisingly have very similar toxicity profiles. In contrast, the observed toxicity profile is somewhat different from that of traditional cytotoxic chemotherapeutic agents and from other epigenetic agents. While some of the side effects may be familiar to the oncologist, others are less commonly seen. Some side effects can be routinely managed (e.g., anti-emetics to alleviate associated nausea and vomiting). Due to asymptomatic electrocardiogram (ECG) changes noted in early trials, patients receiving these agents have been closely monitored; however, HDIs do not appear to be associated with a greater incidence of cardiac adverse events than other chemotherapeutic agents. Inhibition of NF-kB in vivo can be detrimental. NF-kB 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-kB activity. The same functions that make NF-kB attractive for developing inhibitors for treating disease also play a role in homeostasis,and disruption of the NF-kB pathway during development or in adults leads to unfavorable and potentially unhealthy consequences.

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NF-kB 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 a p38 and JNK. In addition to mediating proinflammatory responses, NF-kB 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-kB can have adverse consequences such as immune suppression and tissue damage.
Understanding the consequences of lack of NF-kB 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-kB resulting in defects in development or immunity. Genetic defects have also been discovered in genes that immediately affect NF-kB activation including IKK gamma (NEMO), a subunit of the IKK complex, and IkBalpha.The IKK gamma mutations result in a defective IKK complex and the IkBalpha mutation results in an IkBalpha protein that cannot be phosphorylated and degraded. Both genetic defects result in suppressed NF-kB 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-kB pathway will help prepare for potential adverse effects of pharmacologic NF-kB inhibitors
The requirement for NF-kB in the development and maintenance of the immune system is well documented. NF-kB is required for survival during fetal development and for normal lymphocyte generation in adult mice. Removal of the p65 (RelA) subunit of NF-kB 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-kB 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-kB activity. Lymphocyte depletion with chemical or genetic inhibition of NF-kB have implications for therapeutic potential use in humans. The double-sided nature of NF-kB 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-kB plays a major role in both adaptive and innate immunity. Various signaling pathways responding to receptor recognition of immune challenge converge on NF-kB which then regulates genes that control the immune response. Both T-cell receptor and B-cell receptors activate NF-kB 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-kB 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-kB leading to expression of anti-microbial effector molecules, as well as molecules that help in development of the adaptive immune response. Inhibition of NF-kB during TLR stimulation can lead to macrophage apoptosis, a mechanism used by some pathogens to help evade immune response. NF-kB 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-kB 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-kB plays in immune response to pathogens it is not surprising to find mice genetically deficient in components of the NF-kB pathway are susceptible to parasitic and bacterial infection.
The role of NF-kB in inhibition of apoptosis is one of the factors that make it a potential target for cancer therapy. NF-kB deficient mice die during embryogenesis in part due to TNF-mediated liver damage. Adult mice with impaired NF-kB 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-kB activation.
Niacin (nicotinic acid, Vitamin B3, Vitamin PP) and nicotinamide are both converted into the coenzyme NAD. NAD converts to NADP by phosphorylation in the presence of the enzyme NAD+ kinase. NAD and NADP are coenzymes for many dehydrogenases, participating in many hydrogen transfer processes. NAD is important in catabolism of fat, carbohydrate, protein, and alcohol, as well as cell signaling and DNA repair, and NADP mostly in anabolism reactions such as fatty acid and cholesterol synthesis. High energy requirements (brain) or high turnover rate (gut, skin) organs are usually the most susceptible to their deficiency.
Activating HCA2 has effects other than lowering serum cholesterol and triglyceride concentrations: antioxidative, anti-inflammatory, antithrombotic, improved endothelial function and plaque stability, all of which counter development and progression of atherosclerosis
Niacin inhibits cytochrome P450 enzymes CYP2E1, CYP2D6 and CYP3A4. Niacin produces a rise in serum unconjugated bilirubin in normal individuals and in those with Gilbert's Syndrome. However, in the Gilbert's Syndrome, the rise in bilirubin is higher and clearance is delayed longer than in normal people
In animal models and in vitro, niacin produces marked anti-inflammatory effects in a variety of tissues – including the brain, gastrointestinal tract, skin, and vascular tissue – through the activation of hydroxycarboxylic acid receptor 2 (HCA2), also known as niacin receptor 1 (NIACR1) Unlike niacin, nicotinamide does not activate NIACR1; however, both niacin and nicotinamide activate the G protein-coupled estrogen receptor (GPER) in vitro
Niacin reduces synthesis of low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), lipoprotein(a) and triglycerides, and increases high-density lipoprotein cholesterol (HDL-C) The lipid-therapeutic effects of niacin are partly mediated through the activation of G protein-coupled receptors, including hydroxycarboxylic acid receptor 2 (HCA2) and hydroxycarboxylic acid receptor 3 (HCA3), which are highly expressed in body fat HCA2 and HCA3 inhibit cyclic adenosine monophosphate (cAMP) production and thus suppress the release of free fatty acids (FFAs) from body fat, reducing their availability to the liver to synthesize the blood-circulating lipids in question. A decrease in free fatty acids also suppresses liver expression of apolipoprotein C3 and PPARgamma coactivator-1b, thus increasing VLDL-C turnover and reducing its production
Niacin also directly inhibits the action of diacylglycerol O-acyltransferase 2 (DGAT2) a key enzyme for triglyceride synthesis.
The mechanism behind niacin increasing HDL-C is not totally understood, but seems to occur in various ways. Niacin increases apolipoprotein A1 levels by inhibiting the breakdown of this protein, which is a component of HDL-C. It also inhibits HDL-C hepatic uptake by suppressing production of the cholesterol ester transfer protein (CETP) gene. It stimulates the ABCA1 transporter in monocytes and macrophages and upregulates peroxisome proliferator-activated receptor gamma (PPARgamma), resulting in reverse cholesterol transport.
Severe deficiency of niacin in the diet causes the disease pellagra, characterized by diarrhea, sun-sensitive dermatitis involving hyperpigmentation and thickening of the skin, inflammation of the mouth and tongue, delirium, dementia, and if left untreated, death. Common psychiatric symptoms include irritability, poor concentration, anxiety, fatigue, loss of memory, restlessness, apathy, and depression. The biochemical mechanism(s) for the observed deficiency-caused neurodegeneration are not well understood, but may rest on: A) the requirement for nicotinamide adenine dinucleotide (NAD+) to suppress the creation of

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	neurotoxic tryptophan metabolites, B) inhibition of mitochondrial ATP generation, resulting in cell damage; C), activation of the poly (ADP-ribose) polymerase (PARP) pathway, as PARP is a nuclear enzyme involved in DNA repair, but in the absence of NAD+ can lead to cell death; D) reduced synthesis of neuro-protective brain-derived neurotrophic factor or its receptor tropomyosin receptor kinase B; or E) changes to genome expression directly due to the niacin deficiency. Hartnup disease is a hereditary nutritional disorder resulting in niacin deficiency. It is caused by a genetic disorder that results in a failure to absorb the essential amino acid tryptophan, tryptophan being a precursor for niacin synthesis. The symptoms are similar to pellagra, including red, scaly rash and sensitivity to sunlight. Oral niacin or niacinamide is given as a treatment for this condition in doses ranging from 50 to 100 mg twice a day, with a good prognosis if identified and treated early. Niacin synthesis is also deficient in carcinoid syndrome, because of metabolic diversion of its precursor tryptophan to form serotonin Cytochrome P450 enzymes are essential for the metabolism of many medications. Although this class has more than 50 enzymes, six of them metabolize 90 percent of drugs, with the two most significant enzymes being CYP3A4 and CYP2D6. Genetic variability (polymorphism) in these enzymes may influence a patient's response to commonly prescribed drug classes, including beta blockers and antidepressants. Cytochrome P450 enzymes can be inhibited or induced by drugs, resulting in clinically significant drug-drug interactions that can cause unanticipated adverse reactions or therapeutic failures. Drugs that inhibit CYP2D6 activity are likely to increase the plasma concentrations of certain medications, and, in some cases, adverse outcomes will occur. Some drugs, such as fluoxetine, paroxetine, and quinidine, are particularly potent inhibitors of CYP2D6; patients on these drugs may have almost no CYP2D6 activity. Clinical results suggest that >30% of patients with a poor or ultrarapid CYP2D6 phenotype may experience an adverse outcome after being prescribed codeine, tramadol, oxycodone, or hydrocodone. These medications are frequently prescribed for pain relief, and ~39% of the US population is expected to carry one of these phenotypes, suggesting that the population-level impact of these gene-drug interactions could be substantial. For drugs that are converted to active metabolites by CYP2D6, the addition of a CYP2D6 inhibitor will tend to inhibit the efficacy of the drug. Genetic variability in CYP2D6 activity also can affect the outcome of CYP2D6 drug interactions. In patients genetically deficient in CYP2D6 and who are taking a CYP2D6 substrate, the addition of a CYP2D6 inhibitor will not result in any change in the plasma concentrations of the substrate. CYP2D6 is highly polymorphic with single-nucleotide polymorphisms, small insertions/deletions and larger structural variants including multiplications, deletions, tandem arrangements, and hybridisations with non-functional CYP2D7 pseudogenes. The frequency of these variants differs across populations, and they significantly influence the drug-metabolising enzymatic function of CYP2D6. Importantly, altered CYP2D6 function has been associated with both adverse drug reactions and reduced drug efficacy, and there is growing recognition of the clinical and economic burdens associated with suboptimal drug utilisation The CYP2D6 genotype is associated with the occurrence of adverse effects and clinical nonresponse in psychiatric patients treated with CYP2D6-dependent antidepressants. The cytochrome P450 isozymes, in particular CYP2D6, is responsible for the biotransformation of many psychopharmacological drugs . Substrates of CYP2D6 include first generation antipsychotics, selective serotonin receptor inhibitors and tricyclic antidepressants1. Based on genetic variation, patients can be divided into poor metabolizers (PM), intermediate metabolizers (IM), extensive metabolizers (EM), and ultrarapid metabolizers (UM). The recommended dosages of psychopharmacological medication that are metabolized by this enzyme are based on the metabolism of the most common genotype, i.e., the EM type (i.e., a normal CYP2D6 function). However, because the plasma level of a drug is related to the genotype, the same dosage will probably lead to a higher plasma level in PMs and IMs, as compared to EMs, and to a lower plasma level in UMs as compared to EMs. The plasma level is often related to the effectiveness of the drug and the risk of dose-related side-effects . Also, when physicians prescribe a drug metabolized by CYP2D6 without taking into account the genotype, the hospital stay is longer (and the costs higher) in patients with a PM and UM profile. hepatitis B vaccine is a vaccine that prevents hepatitis B. The first dose is recommended within 24 hours of birth with either two or three more doses given after that.] This includes those with poor immune function such as from HIV/AIDS and those born premature. It is also recommended that health-care workers be vaccinated.] In healthy people, routine immunisation results in more than 95% of people being protected. Serious side effects from the hepatitis B vaccine are very uncommon. Pain may occur at the site of injection.[13] It is safe for use during pregnancy or while breastfeeding.] It has not been linked to Guillain–Barré syndrome.[Hepatitis B vaccines are produced with recombinant DNA techniques and contain immunologic adjuvant.] They are available both by themselves and in combination with other vaccines several studies have looked for an association between recombinant hepatitis B vaccine and multiple sclerosis (MS) in adults.[] Most studies do not support a causal relationship between hepatitis B vaccination and demyelinating diseases such as MS 2006 study concluded that evidence did not support an association between hepatitis B vaccination and sudden infant death syndrome, chronic fatigue syndrome, or multiple sclerosis. A 2007 study found that the vaccination does not seem to increase the risk of a first episode of MS in childhood.[Hepatitis B vaccination has not been linked to onset of autoimmune diseases in adulthood. Studies have found that that immune memory against HepB is sustained for at least 30 years after vaccination, and protects against clinical disease and chronic HepB infection, even in cases where anti-hepatitis B surface antigen (anti-Hbs) levels decline below detectable levels.[Testing to confirm successful immunization or sustained immunity is not necessary or recommended for most people, but is recommended for infants born to a mother who tests positive for HBsAg or whose HBsAg status is not known; for healthcare and public safety workers; for immunocompromised people such as haemodialysis patients, HIV patients, haematopoietic stem cell transplant [HSCT] recipients, or people receiving chemotherapy; and for sexual partners of HBsAg-positive people The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
D-PANTOTHENIC ACID, CALCIUM SALT	Somnolence, respiratory tract changes recorded.
RETINOL PALMITATE	* REACH Dossier
CHOLECALCIFEROL	Target organ data: Behavioural changes, gastro-intestinal effects, and fetotoxicity.
CYANOCOBALAMIN	Oral (several) species: LD50 >5000 mg/kg* Nil reported Reproductive effector in rats
SOYBEAN OIL & GUM ARABIC & THIAMINE HYDROCHLORIDE & NIACINAMIDE &	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe

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PYRIDOXINE HYDROCHLORIDE	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.
SOYBEAN OIL & GUM ARABIC & RIBOFLAVIN 5'-MONOPHOSPHATE SODIUM SALT	No significant acute toxicological data identified in literature search.
SOYBEAN OIL & RETINOL PALMITATE	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.
GUM ARABIC & SODIUM BENZOATE & NIACINAMIDE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Acute Toxicity	✗	**Carcinogenicity**	✗
Skin Irritation/Corrosion	✓	**Reproductivity**	✗
Serious Eye Damage/Irritation	✓	**STOT - Single Exposure**	✓
Respiratory or Skin sensitisation	✓	**STOT - Repeated Exposure**	✗
Mutagenicity	✗	**Aspiration Hazard**	✗

Legend: ✗ ***– Data either not available or does not fill the criteria for classification***
✓ ***– Data available to make classification***

SECTION 12 Ecological information

Toxicity

Troy Nutripet High-energy Vitamin concentrate

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

soybean oil

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

gum arabic

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

DL-alpha-tocopherol acetate

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>20.6mg/l	2
NOEC(ECx)	96h	Fish	11mg/l	2
EC50	72h	Algae or other aquatic plants	>27.8mg/l	2
LC50	96h	Fish	>11mg/l	2

sodium benzoate

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	<650mg/l	1
EC50	72h	Algae or other aquatic plants	>30.5mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	0.09mg/l	2

thiamine hydrochloride

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	>100mg/l	2
EC50	72h	Algae or other aquatic plants	>100mg/l	2
NOEC(ECx)	48h	Crustacea	58mg/l	2

niacinamide

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	560mg/l	1

Continued...

	LC50	96h	Fish	>1000mg/l	2

D-pantothenic acid, calcium salt					
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
Not Available	Not Available	Not Available	Not Available	Not Available

pyridoxine hydrochloride				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
LC50	96h	Fish	>100mg/l	2
EC50	48h	Crustacea	>100mg/l	2
EC50	72h	Algae or other aquatic plants	72mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	3.3mg/l	2

retinol palmitate				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
EC50	48h	Crustacea	35.34mg/l	2
EC50	72h	Algae or other aquatic plants	154mg/l	Not Available
EC50(ECx)	72h	Algae or other aquatic plants	154mg/l	Not Available
LC50	96h	Fish	10000mg/l	Not Available

riboflavin 5'-monophosphate sodium salt				
Endpoint	**Test Duration (hr)**	**Species**	**Value**	**Source**
Not Available	Not Available	Not Available	Not Available	Not Available

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

cyanocobalamin				
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
DL-alpha-tocopherol acetate	HIGH	HIGH
thiamine hydrochloride	HIGH	HIGH
niacinamide	HIGH	HIGH
D-pantothenic acid, calcium salt	LOW	LOW
pyridoxine hydrochloride	LOW	LOW
retinol palmitate	HIGH	HIGH
cholecalciferol	HIGH	HIGH
cyanocobalamin	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
soybean oil	LOW (LogKOW = 22.65)
DL-alpha-tocopherol acetate	LOW (LogKOW = 12.26)
thiamine hydrochloride	LOW (LogKOW = -4.42)
niacinamide	LOW (LogKOW = -0.37)
D-pantothenic acid, calcium salt	LOW (BCF = 3.162)
pyridoxine hydrochloride	LOW (LogKOW = -0.557)
retinol palmitate	LOW (LogKOW = 15.51)

Continued...

Ingredient	Bioaccumulation
riboflavin 5'-monophosphate sodium salt	LOW (LogKOW = -4.92)
cholecalciferol	LOW (LogKOW = 10.24)
cyanocobalamin	LOW (BCF = 3.162)

Mobility in soil

Ingredient	Mobility
DL-alpha-tocopherol acetate	LOW (Log KOC = 13870000)
thiamine hydrochloride	LOW (Log KOC = 87.51)
niacinamide	LOW (Log KOC = 51.56)
D-pantothenic acid, calcium salt	LOW (Log KOC = 10)
pyridoxine hydrochloride	LOW (Log KOC = 10)
retinol palmitate	LOW (Log KOC = 1053000000)
cholecalciferol	LOW (Log KOC = 1515000)
cyanocobalamin	LOW (Log KOC = 10000000000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ 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
soybean oil	Not Available
gum arabic	Not Available
DL-alpha-tocopherol acetate	Not Available
sodium benzoate	Not Available
thiamine hydrochloride	Not Available
niacinamide	Not Available
D-pantothenic acid, calcium salt	Not Available
pyridoxine hydrochloride	Not Available
retinol palmitate	Not Available
riboflavin 5'-monophosphate sodium salt	Not Available
cholecalciferol	Not Available

Continued...

Product name	Group
cyanocobalamin	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
soybean oil	Not Available
gum arabic	Not Available
DL-alpha-tocopherol acetate	Not Available
sodium benzoate	Not Available
thiamine hydrochloride	Not Available
niacinamide	Not Available
D-pantothenic acid, calcium salt	Not Available
pyridoxine hydrochloride	Not Available
retinol palmitate	Not Available
riboflavin 5'-monophosphate sodium salt	Not Available
cholecalciferol	Not Available
cyanocobalamin	Not Available

SECTION 15 Regulatory information

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

soybean oil is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

gum arabic is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

DL-alpha-tocopherol acetate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium benzoate is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)

thiamine hydrochloride is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

niacinamide is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)

D-pantothenic acid, calcium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

pyridoxine hydrochloride is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)

retinol palmitate is found on the following regulatory lists

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

riboflavin 5'-monophosphate sodium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

cholecalciferol is found on the following regulatory lists

Continued...

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 3
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

cyanocobalamin is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (gum arabic; DL-alpha-tocopherol acetate; sodium benzoate; thiamine hydrochloride; niacinamide; D-pantothenic acid, calcium salt; pyridoxine hydrochloride; retinol palmitate; riboflavin 5'-monophosphate sodium salt; cholecalciferol; cyanocobalamin)
China - IECSC	No (thiamine hydrochloride)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (gum arabic; cyanocobalamin)
Korea - KECI	No (retinol palmitate)
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	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (gum arabic; D-pantothenic acid, calcium salt; retinol palmitate; riboflavin 5'-monophosphate sodium salt; cholecalciferol; cyanocobalamin)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	10/03/2023
Initial Date	06/05/2020

SDS Version Summary

Version	Date of Update	Sections Updated
3.1	20/08/2021	Classification change due to full database hazard calculation/update.
4.1	10/03/2023	Classification change due to full database hazard calculation/update.

Other information

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

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

Definitions and abbreviations

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

Continued...

- 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

end of SDS