API Super Ick Cure

Mars Fishcare North America, Inc.

Chemwatch Hazard Alert Code: **0**

Chemwatch: **4658-22**
Version No: **11.1**
Safety Data Sheet according to OSHA HazCom Standard (2024) requirements

Initial Date: **12/19/2005**
Revision Date: **09/04/2026**
Print Date: **09/04/2026**
L.GHS.USA.EN

SECTION 1 Identification

Product Identifier

Product name	API Super Ick Cure
Chemical Name	Not Applicable
Synonyms	Solution ID# 3305; API White Spot Cure (UK); API Fish White Spot Cure
Chemical formula	Not Applicable
Other means of identification	Not Available

Recommended use of the chemical and restrictions on use

Relevant identified uses	For product 12. Use according to manufacturer's directions.

Name, address, and telephone number of the chemical manufacturer, importer, or other responsible party

Registered company name	**Mars Fishcare North America, Inc.**
Address	50 E. Hamilton Street, Chalfont PA 18914 United States
Telephone	215 822 8181
Fax	215 997 1290
Website	Not Available
Email	Not Available

Emergency phone number

Association / Organisation	**ChemTel**	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone number(s)	1-800-255-3924	+1 855-237-5573 (ID#: 4658-22)
Other emergency telephone number(s)	ChemTel: 1-813-248-0585	+61 3 9573 3188

SECTION 2 Hazard(s) identification

Classification of the substance or mixture

NFPA 704 diamond



Note: The hazard category numbers found in GHS classification in section 2 of this SDSs are NOT to be used to fill in the NFPA 704 diamond. Blue = Health, Red = Fire, Yellow = Reactivity and White = Special (Oxidizer or water reactive substances)

Classification	Acute Toxicity (Oral) Category 4, Acute Toxicity (Dermal) Category 3

Label elements

Hazard pictogram(s)	

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Signal word	**Danger**

Hazard statement(s)

H302	Harmful if swallowed.
H311	Toxic in contact with skin.

Hazard(s) not otherwise classified

Not Applicable

Precautionary statement(s) General

P101	If medical advice is needed, have product container or label at hand.
P102	Keep out of reach of children.
P103	Read carefully and follow all instructions.

Precautionary statement(s) Prevention

P280	Wear protective gloves and protective clothing.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.

Precautionary statement(s) Response

P302+P352	IF ON SKIN: Wash with plenty of water.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P361+P364	Take off immediately all contaminated clothing and wash it before reuse.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

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

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available		malachite green oxalate, as
2437-29-8	<0.1	MALACHITE GREEN OXYLATE

The specific chemical identity and/or exact percentage (concentration) of composition has been withheld as a trade secret.

SECTION 4 First-aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with eyes: ‣ Wash out immediately with water. ‣ If irritation continues, seek medical attention. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ‣ Flush skin and hair with running water (and soap if available). ‣ Seek medical attention in event of irritation.
Inhalation	‣ If fumes, aerosols or combustion products are inhaled remove from contaminated area.

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	‣ Other measures are usually unnecessary.
Ingestion	‣ Immediately give a glass of water. ‣ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

Most important symptoms and effects, both acute and delayed

See Section 11

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Fire-fighting measures

Extinguishing media

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

Special hazards arising from the substance or mixture

Fire Incompatibility	None known.

Special protective equipment and precautions for fire-fighters

Fire Fighting	‣ 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	‣ Non combustible. ‣ Not considered a significant fire risk, however containers may burn.

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 breathing vapours and contact with skin and eyes. ‣ Control personal contact with the substance, by using protective equipment. ‣ Contain and absorb spill with sand, earth, inert material or vermiculite. ‣ Wipe up. ‣ Place in a suitable, labelled container for waste disposal.
Major Spills	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Control personal contact with the substance, by using protective equipment. ‣ Prevent spillage from entering drains, sewers or water courses. ‣ Recover product wherever possible. ‣ Put residues in labelled containers for disposal. ‣ 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	‣ Limit all unnecessary personal contact. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ 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.

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	‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Polyethylene or polypropylene container. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	Avoid contamination of water, foodstuffs, feed or seed. None known



+	x	+	o	+	+	o

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
\+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
API Super Ick Cure	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
MALACHITE GREEN OXYLATE	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.

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air)	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers,	0.5-1 m/s (100-

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	welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	200 f/min.)
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min)
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

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

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

Individual protection measures, such as personal protective equipment	
Eye and face protection	‣ Safety glasses with side shields ‣ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
Skin protection	See Hand protection below
Hands/feet protection	‣ Wear general protective gloves, eg. light weight rubber gloves.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. **OTHERWISE:** ‣ Overalls. ‣ Barrier cream. ‣ Eyewash unit.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Dark bluish green slightly acidic liquid with no odour; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	0.998
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	3.4-5.5	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available

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Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available
Nanoform Solubility	Not Available	**Nanoform Particle Characteristics**	Not Available
Particle Size	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	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	The material is not classified as causing respiratory irritation under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be implemented in an occupational setting.
Ingestion	The material is not expected to produce harmful effects following ingestion based on the available information. However, the material may still cause adverse health effects following ingestion, particularly in individuals with pre-existing organ damage (e.g. liver or kidney impairment). Gastrointestinal discomfort may occur and may result in nausea and vomiting. In an occupational setting, ingestion of incidental quantities should be avoided, and good hygiene practices should be followed to prevent ingestion.
Skin Contact	The material is not classified as a skin irritant under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting.
Eye	Although the liquid is not classified as an eye irritant under Regulation (EC) No 1272/2008 (CLP), as amended, direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	The product is not classified as causing specific target organ toxicity following repeated exposure under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, exposure by all routes should be minimised as a matter of course.

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API Super Ick Cure	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
MALACHITE GREEN OXYLATE	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 50 mg/kg[2]	Eye (Rodent - rabbit): 76mg - Severe

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***

MALACHITE GREEN OXYLATE

Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans.
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.
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.
As cationic polymers possess unique physical structures and surface properties, various kinds of cationic polymers have been developed over the past few decades for a wide spectrum of nanomedical applications in the central nervous system (CNS). Although cationic polymers could be successfully used for gene transfer, drug delivery, and diagnostic imaging, after entering into the CNS, they may cause neurotoxicity and induce CNS damage, which seriously limits their applications. The neurotoxic effects of cationic polymers on CNS are mostly studied in mice, and have not been examined in detail.
While evaluating the neurotoxicity of cationic polymers, the surface charge, surface area, coating, size, shape, and the basic materials that cationic polymers are made up of are expected to show important roles, and should be carefully considered. Apoptosis, necrosis, autophagy, oxidative stress, inflammation, and inflammasome; which are expected to be the most important problems in the evaluation of cationic polymers-induced neurotoxicity.
Adverse effects of cytokines have been linked to many disease states and conditions ranging from schizophrenia, major depressio] and Alzheimer's disease to cancer. regulatory cells (Tregs) and related-cytokines are effectively engaged in the process of tumor immune escape and functionally inhibit immune response against the tumor. Forkhead box protein 3 (Foxp3) as a transcription factor is an essential molecular marker of Treg cells. Foxp3 polymorphism (rs3761548) might be involved in cancer progression like gastric cancer through influencing Tregs function and the secretion of immunomodulatory cytokines such as IL-10, IL-35, and TGF-ß.[34] Normal tissue integrity is preserved by feedback interactions between diverse cell types

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mediated by adhesion molecules and secreted cytokines; disruption of normal feedback mechanisms in cancer threaten tissue integrity
Over-secretion of cytokines can trigger a dangerous cytokine storm syndrome. Cytokine storms may have been the cause of severe adverse events during a clinical trial of TGN1412. Cytokine storms are also suspected to be the main cause of death in the 1918 "Spanish Flu" pandemic. Deaths were weighted more heavily towards people with healthy immune systems, because of their ability to produce stronger immune responses, with dramatic increases in cytokine levels. Another example of cytokine storm is seen in acute pancreatitis. Cytokines are integral and implicated in all angles of the cascade, resulting in the systemic inflammatory response syndrome and multi-organ failure associated with this intra-abdominal catastrophe.[36] In the COVID-19 pandemic, some deaths from COVID-19 have been attributable to cytokine release storms. Current data suggest cytokine storms may be the source of extensive lung tissue damage and dysfunctional coagulation in COVID-19 infections
One of the major limiting factors in the efficacy of type I interferon therapy are the high rates of side effects. Between 15% - 40% of people undergoing type 1 IFN treatment develop major depressive disorders.[Less commonly, interferon treatment has also been associated with anxiety, lethargy, psychosis and parkinsonism.[] Mood disorders associated with IFN therapy can be reversed by discontinuation of treatment, and IFN therapy related depression is effectively treated with the selective serotonin reuptake inhibitor class of antidepressant
interferonopathies are a class of hereditary auto-inflammatory and autoimmune diseases characterised by upregulated type 1 interferon and downstream interferon stimulated genes. The symptoms of these diseases fall in a wide clinical spectrum, and often resemble those of viral infections acquired while the child is in utero, although lacking any infectious origin. The aetiology is largely still unknown, but the most common genetic mutations are associated with nucleic
with increasing numbers of clinical trials, physicians realized that IFN therapy may be associated with significant side effects. Although originally assumed to be due to impurities of natural IFN preparations, these side effects have also been
described in patients treated with recombinant IFN. Adverse effects of IFN can be classified into acute, subacute and chronic reactions. Whereas acute adverse reactions are usually self-limiting and resolve after days or weeks of treatment,
chronic adverse reactions, particularly neurological or psychiatric disorders, can cause significant morbidity and sometimes require dose attenuation or even discontinuation of IFN therapy. Some of these toxic reactions are very rare and have
only occurred after prolonged administration of IFN. Because of the variety of clinical manifestations, their diagnosis may be difficult, and sometimes it is even more difficult to ascertain a causal relationship with the IFN therapy. Mechanisms and predisposing factors for the occurrence of chronic toxic reactions remain be fully investigated.

Acute Toxicity of Interferons

The major side effect of all three IFN is a "flu-like" syndrome which occurs in virtually all patients within 3 h after injection and consists of fever, chills, headache, myalgia, arthralgia, gastrointestinal symptoms (anorexia), and fatigue. Temperatures return to normal spontaneously within 12 h after a single dose of IFN.
These acute side effects are not strictly dose-related and usually disappear after about 2 weeks of IFN therapy tachyphylaxis). They can be avoided or palliated by pretreatment with paracetamol or nonsteroidal anti-inflammatory drugs. Similar symptoms have been observed in patients undergoing treatment with interleukin -1, interleukin -2, interleukin -6, or other cytokines. It has therefore been suggested that the flu-like syndrome of IFN is induced via secondary fever-promoting mediators such as interleukin-l, prostaglandin E2, and tumor necrosis factor

Neurological and Psychiatric Disorders

Central nervous system (CNS) toxicity is a relatively frequent side effect of IFN therapy. It may seriously impair quality of life and lead to premature termination of IFN treatment. Estimates of its frequency vary between 13% and 60% . Data
from published studies are difficult to compare because of differences in dose, route, and schedule of IFN administration, differences in medical diagnoses and clinical characteristics of patients included, and variant methods of neurological and neuropsychiatric assessment. Subtle behavioral changes and other subclinical alterations may be found in the majority of patients receiving high doses of IFN]. A wide spectrum of neurological and psychiatric symptoms has been
described in patients undergoing IFN therapy. In many studies, fatigue and asthenia were the most frequently encountered neurotoxic effects, occurring in up to 90% of patients . Other complications of long-term therapy with IFN are
vertigo, ataxia, apraxia, extrapyramidal disorders, cognitive disturbances
(visuospatial disorientation, confusion, somnolence, coma, hallucinations, dysgeusia, hypogeusia), as well as behavioral and emotional changes (psychomotor retardation, lack of initiative, thought blocking, speech stoppage, anorexia, decreased libido, depression, suicidal behavior, irritability, aggressiveness). In addition, there are case reports describing infrequent neurological complications of IFN therapy such as seizures , leukoencephalopathy , oculomotor nerve paralysis , optic tract neuropathy , and trigeminal sensory neuropathy .
Neurological symptoms generally persist for the duration of therapy and resolve within several days to 3 weeks after withdrawal of IFN. Besides CNS disorders,peripheral nervous system defects have been noted in some trials with IFN.
However the overall incidence of peripheral neuropathies appears to be low in most studies. Out of 1019 IFN recipients evaluated only 7% developed mild
Adverse Effects of Interferon Treatment
c. Aul, N. Gattermann, U. Germing, and A. Heyll
https://link.springer.com/chapter/10.1007/978-3-642-60411-9_15
May produce drug-induced autoimmune hepatitis (DIAIH) is a rare hepatoxicity. It is characterized by the presence of autoantibodies (antinuclear, anti smooth muscle, or anti-liver-kidney-microsomal antibodies), raised immunoglobulins, and a hepatocellular pattern of serum enzyme elevations. Liver biopsy has features of autoimmune hepatitis (AIH), including interface hepatitis with a lymphocytic or lymphoplasmacytic infiltrate and hepatic rosette formation. Symptoms can be insidious and may include fatigue, nausea, rash, arthralgia, abdominal discomfort, jaundice, and pruritis. DIAIH responds to corticosteroids and immune suppressors. Hepatitis resolves with the withdrawal of the inciting drug. Associated drugs include antimicrobials (nitrofurantoin and minocycline), interferon, infliximab, and statins.
steroids may play a role in management if no improvement occurs despite discontinuation of medication.
A rare case involving Apetamin (cyproheptadine, lysine, and vitamin syrup) causing DIAIH. a, is composed of active ingredient cyproheptadine 2 g and L-lysine 150 mg, and B vitamins panthenol 4.5 g, nicotinamide 15 mg, thiamine 2 mg, and pyridoxine 1 mg, per 5 mL of syrup. The drug is unregulated in the United States and marketed for selective weight gain.
panthenol 4.5 g, nicotinamide 15 mg, thiamine 2 mg, and pyridoxine 1 mg, per 5 mL of syrup. The drug is unregulated in the United States and marketed for selective weight gain. laboratory work on presentation was significant for aspartate aminotransferase (AST) 88 U/L, alanine transaminase (ALT) 997 U/L, and alkaline phosphate 90 U/L. Smooth muscle antibody was 5 times the upper limit of normal and IgG 2 times the upper limit of normal (3,162 mg/dL), concerning for AIH. Viral hepatitis serology was negative for hepatitis A IgM, hepatitis B core IgM, hepatitis B surface antigen, and hepatitis C antibody. Human immunodeficiency viruses, Epstein-Barr virus, and Cytomegalovirus, QuantiFERON, and mitochondrial antibody were negative;

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	iron and ceruloplasmin were normal. Right upper quadrant ultrasound showed mild echogenicity of the liver seen with hepatic steatosis, normal portal and hepatic veins, and no biliary dilatation. Percutaneous liver biopsy performed on day 2 of admission showed active hepatitis with increased fibrosis, cholestasis, cholangiolar metaplasia, lymphoplasmacytic inflammation, lobular inflammation, disarray, hepatocyte necrosis, and multinucleated hepatocytes The patient scored a 16 on the AIH scale, with a pretreatment likelihood of definite AIH. On the Roussel Uclaf Causality Assessment Method scale, assessing causality between offending drugs and liver damage, the patient scored 11 indicating highly probable adverse drug reaction.3 Findings indicated DIAIH, and the patient was started on prednisone 40 mg oral daily with rapid improvement in liver function. Cyproheptadine has also been linked to hepatitis. Only a few cases have reported hepatoxicity, developing 1-6 weeks after exposure in patients with no previous liver or biliary disease. A cholestatic or mixed pattern of liver enzyme elevations have been described, and acute liver failure is rarely documented. Patients recovered within weeks of discontinuation. The pathway of injury is unknown but believed to be related to the chemical structure. Malachite green and its major metabolite, leuco-malachite green has been reported to have mutagenic and carcinogenic effects. Rats fed malachite green experience "a dose-related increase in liver DNA adducts" along with lung adenomas. Leuco-malachite green causes an “increase in the number and severity of change". As leuco-malachite green is the primary metabolite of malachite green and is retained in fish muscle much longer, most intake of malachite green would be in the leuco form. During the experiment, rats were fed up to 543 ppm of leuco-malachite green, an extreme amount compared to the average 5 ppb discovered in fish. After a period of two years, an increase in lung adenomas in male rats was discovered but no incidences of liver tumors. This shows that although adducts are formed, they have"little mutagenic or carcinogenic consequence" Therefore it could be concluded that malachite green caused carcinogenic symptoms but a direct link between malachite green and liver tumor could not be proved. The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

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

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

SECTION 12 Ecological information

Toxicity

API Super Ick Cure

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

MALACHITE GREEN OXYLATE

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	48h	Crustacea	0.29mg/L	4
EC50(ECx)	72h	Crustacea	0.22mg/L	4
LC50	96h	Fish	0.14mg/L	4

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

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

Continued...

Other adverse effects

No evidence of ozone depleting properties were found in the current literature.

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	‣ Recycle wherever possible. ‣ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ‣ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ‣ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO

Land transport (DOT): 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
MALACHITE GREEN OXYLATE	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
MALACHITE GREEN OXYLATE	Not Applicable

SECTION 15 Regulatory information

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

MALACHITE GREEN OXYLATE is found on the following regulatory lists

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic
US Toxic Substances Control Act (TSCA) - Chemical Substance Inventory
US TSCA Section 12(b) - List of Chemical Substances Subject to Export Notification Requirements
US TSCA Section 5(a)(2) - Significant New Use Rules (SNURs)

Additional Regulatory Information

Not Applicable

Federal Regulations

Superfund Amendments and Reauthorization Act of 1986 (SARA)

Section 311/312 hazard categories

Flammable (Gases, Aerosols, Liquids, or Solids)	No
Gas under pressure	No

Continued...

Explosive	No
Self-heating	No
Pyrophoric (Liquid or Solid)	No
Pyrophoric Gas	No
Corrosive to metal	No
Oxidizer (Liquid, Solid or Gas)	No
Organic Peroxide	No
Self-reactive	No
In contact with water emits flammable gas	No
Combustible Dust	No
Carcinogenicity	No
Acute toxicity (any route of exposure)	Yes
Reproductive toxicity	No
Skin Corrosion or Irritation	No
Respiratory or Skin Sensitization	No
Serious eye damage or eye irritation	No
Specific target organ toxicity (single or repeated exposure)	No
Aspiration Hazard	No
Germ cell mutagenicity	No
Simple Asphyxiant	No
Hazards Not Otherwise Classified	No

US. EPA CERCLA Hazardous Substances and Reportable Quantities (40 CFR 302.4)

None Reported

US. EPCRA Section 313 Toxic Release Inventory (TRI) (40 CFR 372)

None Reported

Additional Federal Regulatory Information

Not Applicable

State Regulations

US. California Proposition 65

None Reported

Additional State Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (MALACHITE GREEN OXYLATE)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes

Continued...

National Inventory	Status
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory ‘Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (MALACHITE GREEN OXYLATE)
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (MALACHITE GREEN OXYLATE)
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	09/04/2026
Initial Date	12/19/2005

SDS Version Summary

Version	Date of Update	Sections Updated
10.1	05/19/2026	Regulatory Update
11.1	09/04/2026	Identification of the substance / mixture and of the company / undertaking - Supplier Information, Identification of the substance / mixture and of the company / undertaking - Synonyms, Name

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.

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