846 Conductive Carbon Grease

MG Chemicals UK Limited

Chemwatch Hazard Alert Code: **2**

Chemwatch: **9-38418**
Version No: **1.1**
Safety Data Sheet (Conforms to Regulations (EC) No 453/2010)

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Initial Date: **Not Available**
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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

1.1.Product Identifier

Product name	846 Conductive Carbon Grease
Chemical Name	Not Applicable
Synonyms	SDS Code: 846; Part Numbers: 846-80G, 846-1P
Proper shipping name	Not Applicable
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	Not Applicable
EC number	Not Applicable
Index number	Not Applicable
REACH registration number	Not Applicable

1.2.Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	For lubricating switches and improving electrical connections
Uses advised against	Not Applicable

1.3.Details of the manufacturer/importer

Registered company name	MG Chemicals UK Limited	MG Chemicals (Head Office)
Address	DMC, Ensor House, Ensor Way, SK22 4NQ New Mills, High Peak United Kingdom	9347-193 Street, Surrey V4N 4E7 British Columbia Canada
Telephone	+44 1663 362888	+1-604-888-3084
Fax	Not Available	+1-604-888-7754
Website	Not Available	www.mgchemicals.com
Email	sales@mgchemicals.com	info@mgchemicals.com

1.4. Emergency telephone number

Association / Organisation	CHEMTREC	Not Available
Emergency telephone numbers	+(44)-870-8200418	Not Available
Other emergency telephone numbers	+(1) 703-527-3887	Not Available

SECTION 2 HAZARDS IDENTIFICATION

2.1.Classification of the substance or mixture

Considered a dangerous mixture according to directive 1999/45/EC, Reg. (EC) No 1272/2008 (if applicable) and their amendments. Not classified as Dangerous Goods for transport purposes.

DSD classification	In case of mixtures, classification has been prepared by following DPD (Directive 1999/45/EC) and CLP Regulation (EC) No 1272/2008 regulations
DPD classification [1]	R40(3) Limited evidence of a carcinogenic effect. R66 Repeated exposure may cause skin dryness and cracking. R7 May cause fire.
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*
Classification according to regulation (EC) No 1272/2008 [CLP] [1]	Carcinogen Category 2
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

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2.2. Label elements

CLP label elements	
SIGNAL WORD	**WARNING**

Hazard statement(s)

H351	Suspected of causing cancer

Supplementary statement(s)

EUH066	Repeated exposure may cause skin dryness or cracking

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised chemical landfill or if organic to high temperature incineration

DSD / DPD label elements



Relevant risk statements are found in section 2.1

Indication(s) of danger	O, Xn

SAFETY ADVICE

S13	Keep away from food, drink and animal feeding stuffs.
S36	Wear suitable protective clothing.
S37	Wear suitable gloves.
S40	To clean the floor and all objects contaminated by this material, use water and detergent.
S46	If swallowed, seek medical advice immediately and show this container or label.
S53	Avoid exposure - obtain special instructions before use.
S56	Dispose of this material and its container at hazardous or special waste collection point.

2.3. Other hazards

	May produce discomfort of the eyes, respiratory tract and skin*.
	Cumulative effects may result following exposure*.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

3.1. Substances

3.2. Mixtures

1. CAS No 2. EC No 3. Index No 4. REACH No	%[weight]	Name	Classification according to directive 67/548/EEC [DSD]	Classification according to regulation (EC) No 1272/2008 [CLP]
1. 63148-62-9 2. 613-156-5 3. Not Available 4. Not Available	78-86	polydimethylsiloxane	Not Applicable	Not Applicable
1. 1333-86-4 2. 603-226-3, 215-609-9, 612-159-9, 619-291-6 3. Not Available 4. 01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX	20-22	carbon black	R40(3), R7[1]	Carcinogen Category 2; H351[1]

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Legend:	*1. Classified by Chemwatch; 2. Classification drawn from EC Directive 67/548/EEC - Annex I; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

SECTION 4 FIRST AID MEASURES

4.1. Description of first aid measures

General	▸ Immediately give a glass of water. ▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary. 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. 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.
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, aerosols or combustion products are inhaled remove from contaminated area. ▸ 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.

4.2 Most important symptoms and effects, both acute and delayed

	See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

	Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

5.1. Extinguishing media

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

5.2. 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

5.3. 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	▸ High temperature decomposition products include silicon dioxide, small amounts of formaldehyde, formic acid, acetic acid and traces of silicon polymers. ▸ These gases may ignite and, depending on circumstances, may cause the resin/polymer to ignite. ▸ An outer skin of silica may also form. Extinguishing of fire, beneath the skin, may be difficult. Combustible. Will burn if ignited. Combustion products include: , carbon monoxide (CO) , carbon dioxide (CO2) , silicon dioxide (SiO2) , other pyrolysis products typical of burning organic material May emit poisonous fumes. May emit corrosive fumes. **CARE**: Water in contact with hot liquid may cause foaming and a steam explosion with wide scattering of hot oil and possible severe burns. Foaming may cause overflow of containers and may result in possible fire. A fire in bulk finely divided carbon may not be obviously visible unless the material is disturbed and sparks appear. A straw broom may be useful to produce the disturbance. Explosion and Ignition Behaviour of Carbon Black with Air

Continued...

Lower Limit for Explosion:	50 g/m3 (carbon black in air)
Maximum Explosion Pressure:	10 bar
Maximum Rate of Pressure Rise:	30-100 bar/sec
Minimum Ignition Temperature:	315 deg. C.
Ignition Energy:	>1 kJ
Glow Temperature:	500 deg. C. (approx.)

Notes on Test Methods:
Tests 1, 2 and 3 were conducted by Bergwerkeschaftliche Versuchstrecke, Dortmunde-Derne, using a 1 m3 vessel with two chemical igniters having an intensity of 5000 W.S.
Tests 1 and 2 results are confirmed by information in the Handbook of Powder Technology, Vol. 4 (P. Field)
In Test 4, a modified Godbert-Greenwald furnace was used. See U.S. Bureau of Mines, Report 5624, 1960, p.5, "Lab Equipment and Test Procedures".
Test 5 used a 1 m3 vessel with chemical igniters of variable intensity.
Test 6 was conducted in a laboratory oven. Active glowing appeared after 3 minutes exposure.
(European Committee for Biological Effects of Carbon Black) (2/84)

SECTION 6 ACCIDENTAL RELEASE MEASURES

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills	▸ Clean up all spills immediately. ▸ Avoid breathing vapours/ aerosols/ or dusts and avoid 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. ▸ Place in a suitable, labelled container for waste disposal.
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.

6.4. Reference to other sections

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

SECTION 7 HANDLING AND STORAGE

7.1. Precautions for safe handling

Safe handling	**NOTE:** ▸ Wet, activated carbon removes oxygen from the air thus producing a severe hazard to workers inside carbon vessels and in enclosed or confined spaces where activated carbons might accumulate. ▸ Before entry to such areas, sampling and test procedures for low oxygen levels should be undertaken; control conditions should be established to ensure the availability of adequate oxygen supply. ▸ 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 MSDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Fire and explosion protection	See section 5
Other information	Carbon and charcoal may be stabilised for storage and transport, without moistening, by treatment with hot air at 50 deg. C.. Use of oxygen-impermeable bags to limit oxygen and moisture uptake has been proposed. Surface contamination with oxygenated volatiles may generate a heat of reaction (spontaneous heating). Should stored product reach 110 deg. C., stacked bags should be pulled apart with each bag separated by an air space to permit cooling away from other combustible materials.

7.2. 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.

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Storage incompatibility	Traces of benzene, a carcinogen, may form when silicones are heated in air above 230 degrees C. Concentrated acids and bases cause degradation of polymer. Boiling water may soften and weaken material. For carbon powders: ▸ Avoid oxidising agents, reducing agents. ▸ Reaction with finely divided metals, bromates, chlorates, chloramine monoxide, dichlorine oxide, iodates, metal nitrates, oxygen difluoride, peroxyformic acid, peroxyfuroic acid and trioxygen difluoride may result in an exotherm with ignition or explosion. Less active forms of carbon will ignite or explode on suitably intimate contact with oxygen, oxides, peroxides, oxosalts, halogens, interhalogens and other oxidising species. ▸ Explosive reaction with ammonium nitrate, ammonium perchlorate, calcium hypochlorite and iodine pentoxide may occur following heating. Carbon may react violently with nitric acid and may be explosively reactive with nitrogen trifluoride at reduced temperatures. In the presence of nitrogen oxide, incandescence and ignition may occur. Finely divided or highly porous forms of carbon, exhibiting a high surface area to mass (up to 2000 m2/g) may function as unusually active fuels possessing both adsorptive and catalytic properties which accelerate the release of energy in the presence of oxidising substances. Dry metal-impregnated charcoal catalysts may generate sufficient static, during handling, to cause ignition. ▸ Graphite in contact with liquid potassium, rubidium or caesium at 300 deg. C. produces intercalation compounds (C8M) which ignite in air and may react explosively with water. The fusion of powdered diamond and potassium hydroxide may produce explosive decomposition. ▸ Activated carbon, when exposed to air, represents a potential fire hazard due to a high surface area and adsorptive capacity. Freshly prepared material may ignite spontaneously in the presence of air especially at high humidity. Spontaneous combustion in air may occur at 90-100 deg. C. The presence of moisture in air facilitates the ignition. Drying oils and oxidising oils promote spontaneous heating and ignition; contamination with these must be avoided. Unsaturated drying oils (linseed oil etc.) may ignite following adsorption owing to an enormous increase in the surface area of oil exposed to air; the rate of oxidation may also be catalysed by metallic impurities in the carbon. A similar, but slower effect occurs on fibrous materials such as cotton waste. Spontaneous heating of activated carbon is related to the composition and method of preparation of the activated carbon. Free radicals, present in charcoal, are responsible for autoignition. Self-heating and autoignition may also result from adsorption of various vapours and gases (especially oxygen). For example, activated carbon auto- ignites in flowing air at 452-518 deg. C.; when the base, triethylenediamine, is adsorbed on the carbon (5%) the autoignition temperature is reduced to 230-260 deg. C.. An exotherm is produced at 230-260 deg. C., at high flow rates of air, although ignition did not occur until 500 deg. C.. Mixtures of sodium borohydride with activated carbons, in air, promote the oxidation of sodium borohydride, producing a self-heating reaction that may result in the ignition of charcoal and in the production of hydrogen through thermal decomposition of the borohydride.

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

7.3. Specific end use(s)

See section 1.2

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

8.1. Control parameters

DERIVED NO EFFECT LEVEL (DNEL)

Not Available

PREDICTED NO EFFECT LEVEL (PNEC)

Not Available

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	carbon black	Carbon black	3.5 mg/m3	7 mg/m3	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	TEEL-0	TEEL-1	TEEL-2	TEEL-3
polydimethylsiloxane		1.5 mg/m3	16 mg/m3	990 mg/m3
carbon black		9 mg/m3	99 mg/m3	590 mg/m3

Ingredient	Original IDLH	Revised IDLH
polydimethylsiloxane	Not Available	Not Available
carbon black	N.E. mg/m3 / N.E. ppm	1,750 mg/m3

MATERIAL DATA

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m3 and considers the material to be an occupational carcinogen.
The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs".
This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

8.2. Exposure controls

8.2.1. Appropriate engineering controls	Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air. Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde] 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.

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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.

8.2.2. Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- 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.

Thermal hazards

Not Available

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:
846 Conductive Carbon Grease Not Available

Material	CPI

* 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.

Respiratory protection

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.
Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face
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)

8.2.3. Environmental exposure controls

See section 12

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Continued...

9.1. Information on basic physical and chemical properties

Appearance	Not Available

Physical state	Free-flowing Paste	**Relative density (Water = 1)**	Not Available
Odour	Odourless	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	200	**Molecular weight (g/mol)**	Not Available
Flash point (°C)	>300	**Taste**	Not Available
Evaporation rate	<1	**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)	<0.13	**Gas group**	Not Available
Solubility in water (g/L)	Immiscible	**pH as a solution(1%)**	Not Available
Vapour density (Air = 1)	>1	**VOC g/L**	Not Available

9.2. Other information

	Not Available

SECTION 10 STABILITY AND REACTIVITY

10.1. Reactivity	See section 7.2
10.2. Chemical stability	▸ Silicone fluids are stable under normal storage conditions. ▸ Hazardous polymerisation will not occur. ▸ At temperatures > 150 C, silicones can slowly react with the oxygen in air. ▸ When heated > 300 C, silicones can slowly depolymerise to volatile siloxanes whether or not air is present. Product is considered stable and hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 TOXICOLOGICAL INFORMATION

11.1. Information on toxicological effects

Inhaled	Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. Not normally a hazard due to non-volatile nature of product Although carbon itself has no toxic action, associated impurities may be toxic. Iodine is often found as an impurity and air-borne carbon dusts, as a result, may produce irritation of the mucous membranes, the eyes, and skin. Symptoms of exposure may include coughing, irritation of the nose and throat and burning of the eyes.
Ingestion	High molecular weight material; on single acute exposure would be expected to pass through gastrointestinal tract with little change / absorption. Occasionally accumulation of the solid material within the alimentary tract may result in formation of a bezoar (concretion), producing discomfort. 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. Ingestion of finely divided carbon may produce gagging and constipation. Aspiration does not appear to be a concern as the material is generally regarded as inert and is often used as a food additive. Ingestion may produce a black stool.
Skin Contact	Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions. Repeated exposure may cause skin cracking, flaking or drying following normal handling and use. Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.

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	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. Low molecular weight silicone fluids may exhibit solvent action and may produce skin irritation. Excessive use or prolonged contact may lead to defatting, drying and irritation of sensitive skin
Eye	Limited evidence or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals. 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. Symptoms of exposure by the eye to carbon particulates include irritation and a burning sensation. Following an industrial explosion, fine particles become embedded in the cornea and conjunctiva resulting in an inflammation which persisted for 2-3 weeks. Some particles remained permanently producing a punctate purplish-black discolouration. When the eyes of human subjects where exposed to silicone fluids, there was evidence of transitory conjunctival irritation within a few hours; this resolved within 24 hours. When applied to the eyes of rabbits, silicone fluids produced transitory irritation which lasted no longer than 48 hours. Injection into the various structures of the eye of animals produced corneal scarring, degenerative changes in the retina, foreign body reaction and cataracts.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Chronic inhalation exposure of production workers has caused decreased pulmonary function ad myocardial dystrophy. There is suggestive but inconclusive evidence that carbon black containing polyaromatic hydrocarbons (PAHs) has been responsible for induction of skin cancers in exposed workers. Long term inhalation of carbon black can cause cough, phlegm, tiredness,chest pain and headache. Dermal, mucosal, or inhalation exposure can cause irritation. Inhalation of carbon black by mice,rats and monkeys caused thickened alveolar walls, increased pulmonary collagen, right atrial and ventricular strain, hypertrophy of the right atrial and ventricular septum and increased heart weights. Although carbon black itself did not cause cancer in treated animals, carbon black containing polyaromatic hydrocarbons (PAHs) did cause cancer following chronic administration by all routes tested. Epidemiological studies of workers in the carbon black producing industries of North America and Western Europe show no significant health effect due to occupational exposure to carbon black. Several other studies provide conflicting evidence. Early studies in the former USSR and Eastern Europe report respiratory diseases amongst workers exposed to carbon black, including bronchitis, pneumonia, emphysema and rhinitis. These studies are of questionable validity due to inadequate study design and methodology, lack of appropriate controls for cigarette smoking and other confounding factors such as concurrent exposure to carbon dioxide, coal oil and petroleum vapours. Moreover, review of these studies indicates that the concentrations of carbon black were greater than current occupational standards. Carbon black may cause adverse pulmonary changes following prolonged or repeated inhalation of the dust; these include oral mucosal lesions, bronchitis and pneumoconiosis which may lead to lung tumours. The body of evidence of carcinogenicity in animal studies comes from two chronic inhalation studies and two intratracheal instillation studies in rats, which showed significantly elevated rates of lung cancer in exposed animals. An inhalation study was tested on mice, but did not show significantly elevated rates of lung cancer in exposed animals. Epidemiologic data comes from three different cohort studies of carbon black production workers. Two studies, from the United Kingdom and Germany, with over 1,000 workers in each study group, showed elevated mortality from lung cancer in the carbon black workers. Another study of over 5,000 workers in the United States did not show elevated mortality from lung cancer in the carbon black workers. Newer findings of increased lung cancer mortality in an update from the UK study may suggest that carbon black could be a late-stage carcinogen. However, a more recent and larger study from Germany did not confirm this hypothesis that carbon black acts as a late-stage carcinogen. In studies employing channel and furnace black, hamsters, mice, guinea pigs, rabbits and monkeys exposed to dusts for 7 hours/day, 5 days/week, at concentrations of 87.4 mg/m3 for channel black and 56.5 mg/m3 for furnace black, no malignancies were observed in any of the animals. Channel black had little if any absorbed polyaromatic hydrocarbons (PAHs) (as benzene extractables) whilst furnace black had 0.28%. Several findings have strengthened the association between inflammation and cancer and between the particle surface area dose of carbon black and other poorly soluble low toxicity (PSLT) particles and the pulmonary inflammation response in mice and the proinflammatory effects in lung cells in vitro. Other evidence suggests that in addition to a cancer mechanism involving indirect genotoxicity through inflammation and oxidative stress, nanoparticles may act as direct carcinogens . Carbon black appears to act like PSLT particles, which can elicit lung tumours in rats following prolonged exposure to sufficiently high concentrations of particles. Particle surface area dose was found to be most predictive of pulmonary inflammation and tumour response in rats when comparing the dose-response relationships for various types and sizes of PSLT including carbon black. Compared to fine PSLT, much lower concentrations of ultrafine PSLT (e.g. 2.5, 6.5 or 11.5 mg/m3 carbon black and ~10 mg/m3 ultrafine titanium dioxide) were associated with impaired clearance, persistent inflammation, and malignant lung tumours in chronic inhalation studies in rats. Most evidence suggests that carbon black and other PSLT-elicited lung tumours occurs through a secondary genotoxic mechanism, involving chronic inflammation and oxidative stress. Experimental studies have shown that when the particle lung dose reaches a sufficiently high concentration (e.g.,mass dose of ~0.5 mg fine-sized PSLT/g lung in rats), the alveolar macrophage-medicated clearance process begins to be impaired (complete impairment occurs at ~10 mg/g lung. Overloading of lung clearance is accompanied by pulmonary inflammation, leading to increased production of reactive oxygen and nitrogen species, depletion of antioxidants and/or impairment of other defense mechanisms, cell injury, cell proliferation, fibrosis, and as seen in rats, induction of mutations and eventually cancer. Rats appear to be more sensitive to carbon black and other PSLT than other rodent species. Although studies in humans have not shown a direct link between inhaled PSLT and lung cancer, many of the steps in the mechanism observed in rats have also been observed in humans who work in dusty jobs, including increased particle lung retention and pulmonary inflammation in workers exposed to coal dust or crystalline silica and elevated lung cancer has been observed in some studies of workers exposed to carbon black, crystalline silica, and diesel exhaust particles Monkeys exposed to channel black for 1000-1500 hours showed evidence of electrocardiac changes indicative of right atrial and right ventricular strain. These changes increased progressively until after 10,000 hours of exposure, when the changes were marked. The authors of this study concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of non-toxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours' exposure and marked atrial and right ventricular strain after 10,000 hours' exposure. The authors concluded that there was no significant effect due to prolonged exposure other than those expected from the accumulation of nontoxic dusts in the pulmonary system. Exposure to furnace black produced a similar picture although electrocardiographic change was first observed in monkeys after 2500 hours exposure and marked atrial and right ventricular strain after 10,000 hours exposure. Chromatographic fractions of oily material extracted from carbon black have been shown to be carcinogenic whilst the unfractionated extracts are not. The activity of some carcinogens appear to be inhibited by carbon black itself. Long term exposure to dust is considered an aggravating factor for chronic simple bronchitis resulting from factors such as infectious diseases or smoking. Chronic inhalation of high levels of dust may produce a mild lung fibrosis. Although the effects of green or calcined coke on man have not been studied, there have been several epidemiology studies conducted at manufacturing plants where petroleum coke was in use. The common feature of these studies was the examination of the effects of dusts and PAHs on the workforce, but in none of them was it possible to identify the contribution of coke to the effects observed. One study was conducted to evaluate respiratory function and reported respiratory disease among workers exposed to petroleum coke dust. In this study, 90 employees (55% of the workforce) participated in a medical investigation which included a respiratory questionnaire, pulmonary function tests (PFT) and chest X-ray. The medical evaluation revealed abnormal PFT results among 9 (10%) current employees. The PFT abnormalities were significantly related to dust exposure as measured by length of employment, age and a history of working for 5 years or longer in the mobile equipment department. Chest X-rays showed no evidence of pneumoconiosis. Although no pneumoconiosis was detected, the medical study did find evidence of occupationally-related pulmonary function abnormalities. In an occupational study involving dock workers, decreased lung function and shortness of breath were found in those workers who were involved with unloading bulk shipments. Coal and cokes were the main products handled. Potroom workers in aluminium smelting industries showed an increased rate of lung cancer mortality. One report from the former Soviet Union associated such an increase with concentrations of tarry substances between 27 and 2130 mg/m3 (B[a]P levels 0.6 to 56 ug/m3). High respiratory mortality has been reported among coke oven workers in Great Britain whilst kidney and lung cancers were prevalent among American coke-oven workers predominantly exposed for more than 5 years. Dense dust generated by the handling and/or processing of this material may be irritating to the eyes, skin, nose, and throat. Ash from furnaces where coke has

Continued...

been burned can contain high concentrations of metals such as nickel and vanadium which, if inhaled in sufficient quantities, can be harmful. The ash may also cause skin irritation and allergic skin reactions following extended skin contact. Inhalation of nickel compounds are linked to lung and nasal cancer in humans.
When rats and monkeys were exposed to petroleum coke dusts in a 2-year study (10.2 or 30.7 mg/m3) no significant toxic effects were observed in monkeys, whilst rats exhibited inflammatory responses at the lower level and metaplastic changes after 18 months exposure to the higher level.
There was no observation of a carcinogenic effect at any dose following a lifetime exposure. There is no evidence of pneumoconiosis or carcinogenicity in human health studies.
24 months of exposure in monkeys and rats to either 10.2 or 30.7 mg/m3 of coke dust resulted in lung accumulation of dust. There was no associated tissue abnormality in monkeys. More significant inflammatory changes occurred in the rat lung at 30.7 mg/m3 (similar to those produced by non-specific respiratory irritants). Overall, it was concluded that neither calcined nor green coke caused a fibrogenic effect in the lungs when compared to silicon dioxide and titanium dioxide. Although some pulmonary inflammation occurred, it was less severe than that caused by silicon dioxide, and more severe than that caused by titanium dioxide. Inflammation was slightly greater in the green coke
There was no evidence of carcinogenicity in either species. Coke dust was found to be non-carcinogenic in mice in chronic skin painting studies. Petroleum coke was found not to be carcinogenic in monkeys and rats in a two-year inhalation study at concentrations up to 30 mg/m3. Petroleum coke (delayed process and fluid process) was found not to be mutagenic in a rat in vivo bone marrow cytogenetic test, a mouse lymphoma assay and an Ames mutagenicity assay.
No significant chromosomal aberrations were observed in rats exposed to 10.2 and 30.7 mg/m3 for 2 years (6 hr/day, 5 day/wk). One study reported no mutagenic activity for petroleum coke while a second study reported a dose related doubling of the number of revertant colonies in the Salmonella/microsome test.
In an in vivo bone marrow cytogenetics study, rats exposed by inhalation to 10 mg/m3, 6 hours/day, 5 days/week for 20 exposures had no bone marrow alterations, but an increase in the frequency of point chromosomal abnormalities were seen in rats exposed to 40 mg/m3, 6 hours/ day for 5 consecutive exposures. To further evaluate this finding, rats were exposed by inhalation to 30.7 mg/m3 of the coke, 6 hours per day, 5 days per week for 5 days, 12 months or 22 months. No increase in the frequency of chromosomal abnormalities was detected in the rats exposed to the micronised delayed process coke. Therefore, micronised delayed process coke is not considered to be mutagenic.

846 Conductive Carbon Grease

TOXICITY	IRRITATION
Not Available	Not Available

polydimethylsiloxane

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3000 mg/kg*	Eye (rabbit): 100 mg/1h - mild
Inhalation (rat) LC50: >1100 mg/m3*	
Oral (rat) LD50: >35000 mg/kg*	
Not Available	Not Available

carbon black

TOXICITY	IRRITATION
Dermal (rabbit) LD50: >3000 mg/kg	
Inhalation (rat) TCLo: 7 mg/m3	
Not Available	Not Available

846 Conductive Carbon Grease

For siloxanes:
Effects which based on the reviewed literature do not seem to be problematic are acute toxicity, irritant effects, sensitization and genotoxicity.
Some studies indicate that some of the siloxanes may have endocrine disrupting properties, and reproductive effects have caused concern about the possible effects of the siloxanes on humans and the environment.
Only few siloxanes are described in the literature with regard to health effects, and it is therefore not possible to make broad conclusions and comparisons of the toxicity related to short-chained linear and cyclic siloxanes based on the present evaluation. Data are primarily found on the cyclic siloxanes D4 (octamethylcyclotetrasiloxane)
and D5 (decamethylcyclopentasiloxane) and the short-linear HMDS (hexamethyldisiloxane).
These three siloxanes have a relatively low order of acute toxicity by oral, dermal and inhalatory routes and do not require classification for this effect.
They are not found to be irritating to skin or eyes and are also not found sensitizing by skin contact. Data on respiratory sensitization have not been identified.
Subacute and subchronic toxicity studies show that the liver is the main target organ for D4 which also induces liver cell enzymes. This enzyme induction contributes to the elimination of the substance from the tissues. Primary target organ for D5 exposure by inhalation is the lung. D5 has an enzyme induction profile similar to that of D4. Subacute and subchronic inhalation of HMDS affect in particular the lungs and kidneys in rats.
None of the investigated siloxanes show any signs of genotoxic effects *in vitro* or *in vivo*. Preliminary results indicate that D5 has a potential carcinogenic effect.
D4 is considered to impair fertility in rats by inhalation and is classified as a substance toxic to reproduction in category 3 with the risk phrase R62 ('Possible risk of impaired fertility').
The results of a study to screen for oestrogen activity indicate that D4 has very weak oestrogenic and antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen). It is not uncommon for compounds that are weakly
oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic potency of D4 relative to ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent than ethinyloestradiol in the rat stain Sprague- Dawley and 3.7 million times less potent than ethinyloestradiol in the Fisher-344 rat strain. Because of the lack of effects on other endpoints designated to assess oestrogenicity, the oestrogenicity as mode of action for the D4 reproductive effects has been questioned. An indirect mode of action causing a delay of the LH (luteinising hormone) surge necessary for optimal timing of ovulation has been suggested as the mechanism.
Based on the reviewed information, the critical effects of the siloxanes are impaired fertility (D4) and potential carcinogenic effects (uterine tumours in females). Furthermore there seem to be some effects on various organs following
repeated exposures, the liver (D4), kidney (HMDS) and lung (D5 and HMDS) being the target organs.
A possible oestrogenic effect contributing to the reproductive toxicity of D4 is debated. There seems however to be some indication that this toxicity may be caused by another mechanism than oestrogen activity
No significant acute toxicological data identified in literature search.
The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

POLYDIMETHYLSILOXANE

For siloxanes:
Effects which based on the reviewed literature do not seem to be problematic are acute toxicity, irritant effects, sensitization and genotoxicity.
Some studies indicate that some of the siloxanes may have endocrine disrupting properties, and reproductive effects have caused concern about the possible effects of the siloxanes on humans and the environment.
Only few siloxanes are described in the literature with regard to health effects, and it is therefore not possible to make broad conclusions and comparisons of the toxicity related to short-chained linear and cyclic siloxanes based on the present evaluation. Data are primarily found on the cyclic siloxanes D4

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	(octamethylcyclotetrasiloxane) and D5 (decamethylcyclopentasiloxane) and the short-linear HMDS (hexamethyldisiloxane). These three siloxanes have a relatively low order of acute toxicity by oral, dermal and inhalatory routes and do not require classification for this effect. They are not found to be irritating to skin or eyes and are also not found sensitizing by skin contact. Data on respiratory sensitization have not been identified. Subacute and subchronic toxicity studies show that the liver is the main target organ for D4 which also induces liver cell enzymes. This enzyme induction contributes to the elimination of the substance from the tissues. Primary target organ for D5 exposure by inhalation is the lung. D5 has an enzyme induction profile similar to that of D4. Subacute and subchronic inhalation of HMDS affect in particular the lungs and kidneys in rats. None of the investigated siloxanes show any signs of genotoxic effects *in vitro* or *in vivo*. Preliminary results indicate that D5 has a potential carcinogenic effect. D4 is considered to impair fertility in rats by inhalation and is classified as a substance toxic to reproduction in category 3 with the risk phrase R62 ('Possible risk of impaired fertility'). The results of a study to screen for oestrogen activity indicate that D4 has very weak oestrogenic and antioestrogenic activity and is a partial agonist (enhances the effect of the estrogen). It is not uncommon for compounds that are weakly oestrogenic to also have antioestrogenic properties. Comparison of the oestrogenic potency of D4 relative to ethinyloestradiol (steroid hormone) indicates that D4 is 585,000 times less potent than ethinyloestradiol in the rat stain Sprague- Dawley and 3.7 million times less potent than ethinyloestradiol in the Fisher-344 rat strain. Because of the lack of effects on other endpoints designated to assess oestrogenicity, the oestrogenicity as mode of action for the D4 reproductive effects has been questioned. An indirect mode of action causing a delay of the LH (luteinising hormone) surge necessary for optimal timing of ovulation has been suggested as the mechanism. Based on the reviewed information, the critical effects of the siloxanes are impaired fertility (D4) and potential carcinogenic effects (uterine tumours in females). Furthermore there seem to be some effects on various organs following repeated exposures, the liver (D4), kidney (HMDS) and lung (D5 and HMDS) being the target organs. A possible oestrogenic effect contributing to the reproductive toxicity of D4 is debated. There seems however to be some indication that this toxicity may be caused by another mechanism than oestrogen activity The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. No toxic response noted during 90 day subchronic inhalation toxicity studies The no observable effect level is 450 mg/m3. Non-irritating and non-sensitising in human patch test. [Xerox]*
CARBON BLACK	No significant acute toxicological data identified in literature search. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. Inhalation (rat) TCLo: 50 mg/m3/6h/90D-I Nil reported

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 required to make classification available*
✘ *– Data available but does not fill the criteria for classification*
⊘ *– Data Not Available to make classification*

CMR STATUS

Not Applicable

SECTION 12 ECOLOGICAL INFORMATION

12.1. Toxicity

NOT AVAILABLE

Ingredient	Endpoint	Test Duration	Effect	Value	Species	BCF
polydimethylsiloxane	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available
carbon black	Not Available	Not Available	Not Available	Not Available	Not Available	Not Available

For siloxanes:
Environmental fate:
It is well accepted that polydimethylsiloxane fluids become permanent residents of sediment but should not exert adverse environmental effects.
Silicone fluids are very surface active because the flexible siloxane linkages permit alignment of the hydrophobic methyl substituents towards the non-polar phase, and of the polysiloxane backbone towards the polar phase. The polar medium is generally water, and a polar media to which polydimethylsiloxanes become attached may be textiles, sewage sludge, hair, algae, sediment etc. In aqueous environments, polydimethylsiloxanes are adsorbed onto sedimenting particles. Also, in the presence of nitrate ions, which exist at various concentrations in the environment, short chain siloxanes are photodegraded to the level of silicate within days
The stability of the siloxanes, desirable from a technical point of view, makes the siloxanes very persistent, and once released to the environment the siloxanes remain for many years.
The main source of releases of siloxanes to the air is volatile siloxanes used in cosmetics, wax, polishes, and to a minor extent in several other applications. the volatile siloxanes may account for a significant part of the siloxanes used for cosmetics.
Non-volatile silicone fluids used in cosmetics, wax, polishes, cleaning products and for textile applications (softeners) will to a large extent end up in wastewater and be directed to wastewater treatment plants.
The cyclic siloxanes and small-chain linear siloxanes are bioconcentrated (bioconcentration factors for long-chained siloxanes have not been assessed). The estimated bioconcentration factors (BCF) of the small siloxanes range from 340 for HMDS to 40,000 for a phenylated trisiloxane (phenyl trimethicone). The small phenylated siloxanes seem to have very high BCF, and model estimates indicate that these substances are the most toxic for aquatic organisms.
PBT profiler screening
In order to make a first comparison between the substances as to persistence, bioaccumulation and toxicity, the substances were screened using the PBT profiler developed by U.S. EPA (U.S. EPA 2003). The profiler uses a procedure to predict persistence, bioaccumulation, and toxicity of organic chemicals on the basis of the chemical structure and physical
parameters of the substances combined with experimental parameters for substance with a similar structure, using a QSAR approach.
The results for six members of the siloxane family predict the highest bioconcentration factors for the two phenyl siloxanes, one order of magnitudes higher than the values for the cyclic siloxanes and two orders of magnitudes higher than the values for the small linear methyl siloxanes. The predicted toxicity is as well significantly higher (lowest ChV values) for the phenyl siloxanes. The predicted half-life is nearly the same for all substances.
Using U.S. EPA's criteria, the screening indicates that all substances are of high concern as to environmental toxicity, and that the phenyl siloxanes are considered very bioaccumulative.
Ecotoxicity:

Continued...

The environmental fate and effects of volatile methylsiloxanes (mainly cyclosiloxanes) and polydimethylsiloxane (PDMS) have been reported:
For octamethylcyclosiloxane:
Fish acute LC50 (14 day):: rainbow trout 10 ug/l; sheepshead minnow >6.3 ug/l
Daphnia magna acute EC50 (48 h): >15 ug/l; NOEC 15 ug/l
Mysid shrimp acute LC50 (96 h): >9.1 ug/l; NOEC 9.1 ug/l
For PDMS
Daphnia magna NOEC 572 mg/kg
Physical effects such as surface entrapment have been observed when testing aquatic invertebrates in clean laboratory water, but similar effects are not expected in natural environments where a large variety of other surfaces provide opportunities for deposition
DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
Not Available	No Data available for all ingredients	No Data available for all ingredients

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
Not Available	No Data available for all ingredients

12.4. Mobility in soil

Ingredient	Mobility
Not Available	No Data available for all ingredients

12.5.Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT and vPvB Criteria fulfilled?	Not Available	Not Available	Not Available

12.6. Other adverse effects

No data available

SECTION 13 DISPOSAL CONSIDERATIONS

13.1. Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. ▸ Where possible retain label warnings and MSDS and observe all notices pertaining to the product. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	Class: Not Applicable Subrisk: Not Applicable
14.6. Special precautions for user	Special provisions: Not Applicable Limited quantity: Not Applicable

Continued...

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

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	ICAO/IATA Class: Not Applicable ICAO / IATA Subrisk: Not Applicable ERG Code: Not Applicable
14.6. Special precautions for user	Special provisions: Not Applicable Cargo Only Packing Instructions: Not Applicable Cargo Only Maximum Qty / Pack: Not Applicable Passenger and Cargo Packing Instructions: Not Applicable Passenger and Cargo Maximum Qty / Pack: Not Applicable Passenger and Cargo Limited Quantity Packing Instructions: Not Applicable Passenger and Cargo Limited Maximum Qty / Pack: Not Applicable

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

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	Not Applicable
14.5. Transport hazard class(es)	IMDG Class: Not Applicable IMDG Subrisk: Not Applicable
14.6. Special precautions for user	EMS Number: Not Applicable Special provisions: Not Applicable Limited Quantities: Not Applicable

Inland waterways transport (ADNR / River Rhine): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Available
14.2. Packing group	Not Available
14.3. UN proper shipping name	Not Available
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	Not Available: Not Applicable
14.6. Special precautions for user	Classification code: Not Available Limited quantity: Not Available Equipment required: Not Available Fire cones number: Not Available

Inland waterways transport (ADN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable
14.2. Packing group	Not Applicable
14.3. UN proper shipping name	Not Applicable
14.4. Environmental hazard	No relevant data
14.5. Transport hazard class(es)	Not Applicable: Not Applicable
14.6. Special precautions for user	Classification code: Not Applicable Limited quantity: Not Applicable Equipment required: Not Applicable Fire cones number: Not Applicable

Continued...

Transport in bulk according to Annex II of MARPOL 73 / 78 and the IBC code

Source	Ingredient	Pollution Category
IMO MARPOL 73/78 (Annex II) - List of Noxious Liquid Substances Carried in Bulk	polydimethylsiloxane	Y

SECTION 15 REGULATORY INFORMATION

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

polydimethylsiloxane(63148-62-9) is found on the following regulatory lists	"European Customs Inventory of Chemical Substances ECICS (English)"
carbon black(1333-86-4) is found on the following regulatory lists	"European Customs Inventory of Chemical Substances ECICS (English)","European Trade Union Confederation (ETUC) Priority List for REACH Authorisation","International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","European Union - European Inventory of Existing Commercial Chemical Substances (EINECS) (English)","European List of Notified Chemical Substances (ELINCS)","UK Workplace Exposure Limits (WELs)"

This safety data sheet is in compliance with the following EU legislation and its adaptations - as far as applicable - : 67/548/EEC, 1999/45/EC, 98/24/EC, 92/85/EC, 94/33/EC, 91/689/EEC, 1999/13/EC, Regulation (EU) No 453/2010, Regulation (EC) No 1907/2006, Regulation (EC) No 1272/2008 and their amendments as well as the following British legislation: - The Control of Substances Hazardous to Health Regulations (COSHH) 2002 - COSHH Essentials - The Management of Health and Safety at Work Regulations 1999

15.2. Chemical safety assessment

For further information please look at the Chemical Safety Assessment and Exposure Scenarios prepared by your Supply Chain if available.

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
polydimethylsiloxane	63148-62-9	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Aquatic Chronic 2, Flam. Liq. 3, Skin Corr. 1A, Eye Dam. 1, Repr. 2, Acute Tox. 1, Acute Tox. 2, Asp. Tox. 1	Wng, GHS09, GHS02, Dgr, GHS05, GHS08, GHS06	H411, H226, H314, H318, H361, H300, H304

Harmonisation Code 1 = The most severe classification. Harmonisation Code = 2 The most prevalent classification

Ingredient	CAS number	Index No	ECHA Dossier
carbon black	1333-86-4	Not Available	01-2119384822-32-XXXX, 01-2119489801-30-XXXX, 01-2119475601-40-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
2	Carc. 2, Eye Irrit. 2, STOT RE 1, Self-heat. 1, Skin Irrit. 2, STOT SE 1, Aquatic Chronic 1, Acute Tox. 4, Flam. Sol. 2	GHS08, Wng, Dgr, GHS06, GHS02, GHS09	H351, H319, H372, H251, H228, H315, H370, H410, H332

Harmonisation Code 1 = The most severe classification. Harmonisation Code = 2 The most prevalent classification

SECTION 16 OTHER INFORMATION

Full text Risk and Hazard codes

H226	Flammable liquid and vapour
H228	Flammable solid
H251	Self-heating; may catch fire
H300	Fatal if swallowed
H304	May be fatal if swallowed and enters airways
H314	Causes severe skin burns and eye damage
H315	Causes skin irritation
H318	Causes serious eye damage
H319	Causes serious eye irritation
H332	Harmful if inhaled
H361	Suspected of damaging fertility or the unborn child
H370	Causes damage to organs
H372	Causes damage to organs through prolonged or repeated exposure
H410	Very toxic to aquatic life with long lasting effects
H411	Toxic to aquatic life with long lasting effects

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.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/references

The (M)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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For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:
EN 166 Personal eye-protection
EN 340 Protective clothing
EN 374 Protective gloves against chemicals and micro-organisms
EN 13832 Footwear protecting against chemicals
EN 133 Respiratory protective devices

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