SIDS INITIAL ASSESSMENT PROFILE

CAS No.	**1333-86-4**
Chemical Name	**Carbon Black**
Structural Formula	**Elemental Carbon (C)**

The current assessment focuses exclusively on industrially manufactured carbon black which is a generic term for a high purity elemental form of carbon consisting of near spherical colloidal primary particles (10-500 nm in diameter) fused into aggregates of such particles (80-810 nm in diameter) during a totally enclosed production process. The aggregates are tightly bound, forming the primary, dispersible unit of carbon black, and rapidly form agglomerates in the reactor. Carbon black contains less than 1% organic impurities firmly adsorbed to its surface, including polycyclic aromatic hydrocarbons (PAHs).

Other forms of carbon-containing respirable particles that are released from various industrial and other processes, such as the incomplete combustion of carbonaceous materials (e.g. soot or diesel exhaust particles) and which contain higher proportions of adsorbed organic compounds such as PAHs are not covered by this assessment.

SUMMARY CONCLUSIONS OF THE SIAR

Human Health

Uptake and retention of carbon black particles in lung macrophages has been observed following inhalation. In rats, clearance of carbon black particles from the respiratory tract is delayed at lung burdens equal or greater than 0.5 – 1.0 mg carbon black / g lung ("lung overload"). Little carbon black is found in Peyer`s patches after oral exposure. It is unlikely that the insoluble particles are capable of skin penetration.

The acute oral toxicity of carbon black in animals is very low; no clinical signs of toxicity were noted in rats gavaged with the maximum technically achievable dose (8000-10,000 mg/kg bw). Small inflammatory changes in lung and bronchoalveolar fluid were found in rats after a 7-hour inhalation exposure to a high surface area carbon black (20 nm primary particle size; 1 mg/m^3), whilst low surface area carbon black (200 nm primary particle size; 1 mg/m^3) had no effect.

Carbon black was not irritating to the skin and eyes of rabbits in tests performed similar to current OECD guidelines. As superficial foreign bodies, carbon black particles may be slightly irritating mechanically and may cause discoloration of lids and conjunctivae in humans. There is no information to suggest that carbon black might be a skin sensitiser.

After repeated inhalation of a high surface area carbon black for 13 weeks, no pathological or biochemical changes were found in the lungs of rats at 1.1 mg/m^3 (NOAEL, respirable fraction) but there were clear dose related increases in both biochemical and cellular markers of inflammation and lung damage at the next higher concentration of 7.1 mg/m^3 (respirable fraction). By 8 months post-exposure there was substantial clearance of the carbon black retained in the lungs of animals exposed to 1.1 mg/m^3, moderate clearance in the mid-exposure group (7.1 mg/m^3) and very little at 52.8 mg/m^3. Severe lung damage (including lung tumours) was seen in rats of both sexes exposed for 2 years to 2.5 mg/m^3 (16 hrs/day, 5 days/week).

In exposed carbon-black production workers, repeated inhalation exposure to carbon black can cause decrements in pulmonary function, increases in reported respiratory symptoms, and, possibly, chest film changes. Based on data from a large European multi-centre study covering 19 plants in 7 countries (UK , 2 plants; France, 3 plants; Germany, 5 plants; Holland, 2 plants; Italy, 3 plants; Spain, 3 plants; and Sweden, 1 plant), predictions suggest that after 40 years exposure to 1 mg/m^3 (inhalable fraction, 8-hr TWA) there would be minimal effects on lung function parameters. It has been estimated that exposure to a working lifetime of 40 years to inhalable carbon black at 1, 2 and 3.5mg/m^3 (8-hour TWA) would lead to mean decreases in FEV_1 of 48, 91 and 169 ml,

respectively. This may be compared to the average age-related decline in FEV_1 in adult males of about 1,200 ml over this 40-year period. A study of production workers in North America covering 22 plants (Canada, 2 plants; United States, 20 plants) yielded similar respiratory function results for 1 mg/m^3 40-year working-life exposures (FEV_1, 28 ml decrease).

In vitro, carbon blacks were non-mutagenic in various Ames tests, whilst organic extracts can exhibit a wide variety of activity, depending on the conditions of extraction. This activity is ascribed to mutagenic organic impurities (mainly polycyclic aromatic hydrocarbons) in the extract. Carbon black was tested negative in a mouse lymphoma assay, and did not induce sister chromatid exchanges in Chinese Hamster Ovary cells. The available evidence strongly suggests that Carbon Black is not directly mutagenic and that mutations are caused by secondary mechanisms such as oxidative stress; for these effects triggered by inflammatory processes, there is a threshold which has been shown to be above 1 mg /m³ respirable for high-surface Carbon Black (e.g. Printex 90). The threshold for low–surface Carbon Black is above this value.

In vivo, exposure of rats to doses of carbon black particles producing significant inflammation was associated with increased mutation in the hypoxanthine-guanine phosphoribosyl transferase gene (hprt) in alveolar type II epithelial cells. Addition of catalase inhibited the increase in mutation frequency implying a role for cell-derived oxidants in this reaction.

Animal carcinogenicity studies demonstrated that carbon black of respirable size could produce lung tumours in rats of both sexes, but not in mice or hamsters. Increases in the incidence of benign and malignant lung tumours were seen at the lowest concentration tested (2.5 mg/m^3, 16 hrs/day, 2 years). The lung tumours occurred under conditions that resulted in impaired lung clearance ("overload"). There is also evidence that inflammation and cell proliferation may have contributed to the development of rat lung tumours.

Skin painting studies in mice using a variety of commercial carbon blacks did not induce signs of skin cancer development. Limited lifetime oral studies showed no evidence of dermal carcinogenicity in rats and mice.
Studies of the carcinogenic potential of carbon black in workers generally suffer from limitations, and are considered not to reveal clear evidence for a causal role of carbon black in the development of human cancers (IARC 1996).

In relation to lung cancer, various cohort and case-control studies in the US did not show any increases in lung cancer risk in carbon black production workers. Cohort mortality studies of workers exposed to carbon black in the UK found an excess of lung cancer in some, but not all factories included in the study, and there was no association between duration of carbon black exposure and lung cancer mortality, nor were possible confounders such as smoking or past occupational histories taken into account.

A number of cases of skin cancer were identified in carbon black production workers in the US, whilst in a cohort of carbon black workers in the UK no excesses of skin cancer were found. Also, a study in the rubber and tyre manufacturing industry did not reveal an increased risk of squamous cell skin cancer in workers exposed to carbon black contaminated materials.

An excess number of bladder cancer cases were recently reported in dock workers with a history of manually unloading shipments of carbon black. As there is no information on potential confounding factors such as other chemical exposures in this workforce, and shipyard workers at the same harbour but not exposed to carbon black also showed an increase in bladder cancer, a role for carbon black in bladder cancer is unlikely.

Based on available data demonstrating a low bioavailability, the polycyclic aromatic hydrocarbons (PAHs) contained in carbon black are generally considered not play a role in lung cancer of laboratory rats. The lung cancers in rats are considered by some to be the result of a non-genotoxic mechanism secondary to cellular toxicity brought about by lung overloading, inflammation and oxidative stress. The relevance of carbon-black induced lung tumours in rats to human health is uncertain, but it appears that the rat is the most sensitive species to the effects of lung overload.

Carbon black has not been tested in guideline studies for its effects on fertility, reproduction and the developing organism. Based on the available toxicokinetic principles, it is very unlikely that carbon black particles will reach the reproductive organs, the embryo or the fetus under *in vivo* conditions. No adverse effects on reproduction and development would therefore be expected.

Environment

Carbon black is substantially elemental carbon. It has no functional groups that could bring about solubility in water and organic solvents. Its vapour pressure is negligible. It cannot be further degraded by hydrolysis, light or by photodegradation in air or in surface water. These physico-chemical properties are reason why important parameters like water solubility, octanol/water partition coefficient, dissociation constant or adsorption/desorption which are relevant for environmental fate and distribution cannot be analytically measured. Based on these properties it is expected that carbon black will not occur in air or water in relevant amounts. Also potential for distribution via water or air, respectively, can be dismissed. The deposition in soil or sediments is therefore the most relevant compartment of fate of carbon black in the environment, but carbon is widely distributed in nature and an essential element in the components of all living organisms.

Based on the physical-chemical properties of carbon black as an inert solid, its insolubility and stability in water and in organic solvents, diffusion through the gills or through the membranes of the body of the aquatic organisms and bioaccumulation is not expected.

As an inorganic compound with the chemical structure "C", carbon black will not be further biodegraded by microorganisms.

Since carbon black is not soluble in water and a difficult substance in aquatic toxicity testing, the preparation of the test mediums was not always in accordance with the OECD standard test guidelines but was carried out in accordance with the OECD Guidance Document on Aquatic Toxicity Testing of Difficult Substances and Mixtures, published in 2000. Its low toxicity, requiring high concentrations to be tested in order that toxicity might be detected, and the low pH, depending on the type of carbon black, of some aqueous suspensions makes the testing protocol even more difficult. Nevertheless, results with fish tests have established that a fish LC_{50} related to the nominal concentrations were greater than 5000 mg/L for aqueous suspensions, and greater than 10,000 mg/L for water accommodated filtrates. The results in the acute *Daphnia* test from water-accommodated filtrates indicated an EC_{50} of 5600 mg/L related to the nominal concentration which is attributed to the pH of the solution. The algal test results, also from water accommodated filtrates, showed no adverse effects at the highest tested concentration, from a nominal concentration of 10,000 mg/L. In addition, supporting tests with tyre dust filtrates showed LC_{50} >58,000mg/L, EC_{50} >69,000mg/L, and EC_{50} of >13,000mg/L for fish, *Daphnia*, and algae respectively, all related to the nominal concentrations. Because an analytical determination of the carbon black concentration is technically not feasible in the tests medium, and the test substance is present in a biologically unavailable form, the estimation of the true exposure concentration is difficult, therefore the PNEC calculation in the aquatic environment based on the nominal concentrations or loading rates respectively is not realistic. If the fish, *Daphnia*, and algal results were used to calculate a PNEC, an application factor of 1000 would be required. If this were applied to the fish LC_{50} of >5,000 mg/L, then a PNEC of >5 mg/L would result. However, the fish and invertebrate LC_{50} and EC_{50} data are dominated by physical and pH considerations, and treatment of these results by methodology appropriate to a chemical toxicity mechanism may not be appropriate.
Carbon black is not expected to interfere with the operation of sewage treatment plants, although it was not possible to carry out a sludge respiration study, due to the particulate nature of carbon black. The dehydrogenase activity of sewage treatment organisms has been tested, with an EC_{10} of approximately 800 mg/L nominal concentration of a suspension of carbon black particles.

Although no reliable tests on terrestrial organisms have been carried out with carbon black, earthworm tests have been reported for filtered extractions of tyre dust. These tests, on filtrate from 100g of material, shaken for 24 hours in one litre of water, showed no toxicity. This supports the expected low toxicity of carbon black to terrestrial organisms.

Exposure

Worldwide production capacity of carbon black is of the order of 8 million tonnes (1996) with North America contributing about 1,815,000 tons; Western Europe 1,310,000 tons; Eastern Europe 1,545,000 tons; Asia 2,630,000 tons; South America 480,000 tons, and Africa and Australia 185,000 tons.

Approximately 90% of carbon black is used in rubber applications (70% is used as a reinforcement in tyres for automobiles and other vehicles, 20% for other rubber products such as hoses, gaskets, mechanical and molded goods, and footwear), 9% is used as a pigment in printing inks and surface coatings, and the remaining 1% as an ingredient in hundreds of diverse applications, for instance, in the manufacture of dry-cell batteries. End users of rubber, ink or paint products are not exposed to Carbon Black *per se*, it is bound within the product matrix

(IARC 1996). Releases into the environment may occur from production and processing. Both shredded tyres, and carbon black have been shown to significantly adsorb organic pollutants. Also, aqueous desorption of adsorbed organics of various polarities from carbon black has been shown to be negligible.

Occupational exposure may occur during production and processing. Geometric mean personal exposure, measured in carbon black-producing plants in Western European countries as the respirable and inhalable dust fractions (approximately 7400 and 8000 samples, respectively) and North America as total, respirable, and inhalable carbon black dust fractions (approximately 4100, 2500 and 1000 samples, respectively) is on average less than 1 mg/m³ on an 8-hour time-weighted average basis. A recent study has demonstrated that occupational exposure to airborne carbon black in carbon black-producing plants is not in the ultrafine or nanoparticle range.

RECOMMENDATION AND RATIONALE FOR THE RECOMMENDATION AND NATURE OF FURTHER WORK RECOMMENDED

Human Health: The chemical is currently of low priority for further work.

The chemical possesses properties indicating a potential long-term hazard for human health. The relevance of carbon black induced lung tumours in rats to human health is uncertain. IARC classified carbon black as Group 2B ` *possibly carcinogenic to humans´* (1996).

It is noted that from other chemical studies [TiO_2, talc, etc.] that the rat is a species sensitive to lung overload. Judgment as to the interpretation of these data relative to human risk is clearly beyond the SIDS program. The SIAM is aware that such work is in progress. Carbon black, TiO_2 and talc were re-evaluated by IARC in February 2006.

Based on data presented by industry to the Sponsor country, exposure is controlled in occupational settings. Geometric mean personal exposure, measured in production factories in western European countries as respirable and inhalable and North America as total, respirable, and inhalable carbon black is on average less than 1 mg/m³ 8-hr TWA. In most products, carbon black is bound into a matrix. Therefore, exposure is negligible for consumers. Countries may wish to investigate any exposure scenarios that were not represented by the Sponsor country.

Environment: The chemical is currently of low priority for further work because of its low hazard profile.