Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies’ umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit: www.nicnas.gov.au

Disclaimer

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Acronyms & Abbreviations

Chemical Identity

| Synonyms                      | acetylene black  |
|                              | channel black    |
|                              | furnace black    |
|                              | lampblack        |
|                              | thermal black    |

Structural Formula

Molecular Formula: Unspecified

Molecular Weight (g/mol): 12.01

Appearance and Odour (where available): Odourless, fine black powder or black solid

SMILES: C

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

The chemical has reported domestic uses, including in:

- adhesives (binding agents);
- cleaning/washing agents;
- colouring agents;
- paints, lacquers and varnishes; and
- fillers.

The chemical has reported commercial uses, including:
The chemical has reported site-limited uses, including in:

- electrical and/or electronic devices; and
- manufacturing other chemicals.

The chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume of 10000–99999 tonnes.

**International**

The following international uses have been identified through European Union Registration, Evaluation, Authorisation and Restriction of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development (OECD) Screening Information Dataset Initial Assessment Report (SIAR); Galleria Chemic; Substances and Preparations in the Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (Cosing) database; United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) dictionary; and eChemPortal: OECD High Production Volume chemical program (OECD HPV), the US Environmental Protection Agency's Aggregated Computational Toxicology Resource (ACToR), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported cosmetic uses, (maximum concentrations specified in SCCS, 2013):

- as a colourant in skin products (0.001 %);
- in nail enamels and mascaras (5 %); and
- in eye decorative products (up to 10 %).

The chemical has reported use in tattoo inks (black pigments) (Hauri, 2011).

The chemical is widely available for cosmetic/domestic uses in the United States (Personal Care Products Council, 2011; US Household Products Database).

The chemical has reported domestic uses, including in:

- adhesives (binding) agents (<2 %);
- cleaning/washing agents;
- colouring agents;
- paints, lacquers, and varnishes (<1 %);
- corrosion inhibitors; and
- surface treatments (<2 %).

The chemical has reported commercial uses, including:

- in insulating materials;
- as a pigment in printing inks;
- in lubricants and additives;
- as an anti-set-off and anti-adhesive agent;
- as a filler, reinforcement and pigment in rubber products, tyres, belts, requiring abrasion resistance (80 %);
- in plastics;
- in construction materials;
- in explosives;
The chemical has reported site-limited uses, including as:
- in grinding materials;
- in impregnation materials;
- in photochemicals such as UV light absorbers and semiconductors (1%);
- as a process regulator; and
- in reprographic agents.

The chemical has reported non-industrial uses:
- an intermediate for manufacturing other substances; and
- a vulcanising agent.

The chemical has reported non-industrial uses:
- in agricultural and non-agricultural pesticides; and
- as a food preservative.

**Restrictions**

**Australian**

No known restrictions have been identified.

**International**

No known restrictions have been identified.

**Existing Work Health and Safety Controls**

**Hazard Classification**

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

**Exposure Standards**

**Australian**

The chemical has an exposure standard of 3 mg/m$^3$ time weighted average (TWA).

**International**

The following exposure standards are identified (Galleria Chemica):

**TWA**

- 3.5 mg/m$^3$ TWA in Canada (Alberta, Quebec, Saskatchewan, Yukon), Denmark, France, and the United States of America (California, Hawaii, Minnesota).

**Short-term exposure limit (STEL)**

- 7 mg/m$^3$ in Canada (Saskatchewan, Yukon), Egypt, Greece, Ireland, South Africa, the UK, and the USA (Hawaii, Washington).

**Health Hazard Information**
The chemical is a powder containing primary particles in the size range of 10–500 nm, fused together to form aggregates generally over 100 nm in size (EC-HC, 2013; OECD, 2006). The aggregates are chains of ‘primary carbon particles that are permanently fused together in a random branching structure’ (SCCS, 2013) and reported as unbreakable (ICBA, 2004). However, the chemical can also contain a limited fraction of primary particles <100 nm (EC-HC, 2013).

The commercial chemical contains >97% elemental carbon with different amounts of oxygen, hydrogen and sulphur. Less than 1 % by weight of the chemical consists of extractable organic materials absorbed onto the particle surface (SCCS, 2013). In the manufacturing process, the chemical is categorised as acetylene black, channel black, furnace black, lamp black, or thermal black. Furnace black is the predominant commercial form of this chemical (>95 % total world production) and has been used as a test material in the majority of the toxicological studies (IARC, 2010).

Carbon black has been used widely for many decades in industrial applications. It has not been considered an industrial nanomaterial under the NICNAS working definition for nanomaterials as it was not manufactured intentionally to have particles in the nano scale (<100 nm) with unique properties (http://www.nicnas.gov.au/communications/issues/nanomaterials-nanotechnology/nicnas-working-definition-for-industrial-nanomaterial). However, this report used toxicological data available for the chemical, including the studies conducted using the chemical with primary particles in the nano scale as reported in the SCCS (2013). The primary particles in the nano scale are considered to be in aggregate/agglomerate form in the commercial chemical; the SCCS report (2013) stated that isolated carbon black nano particles could be rarely or not identified using electron microscopy techniques, in commercial formulations, cosmetic products or the test materials used in the toxicity studies. The aggregates/agglomerates observed were reported to be in the size range of 0.1–2500 µm (SCCS, 2013).

Recent information suggests that carbon black may now be produced intentionally to have particles in the nano scale, to have unique properties (e.g. to use in tyres to achieve increased durability and road grip) (OECD, 2013), allowing it to be considered as an industrial nanomaterial under the NICNAS working definition. This report does not specifically address any health risks anticipated from the intentionally produced nanomaterials of the chemical, as risk assessment of intentionally produced nanomaterials is out of scope of the IMAP framework.

**Toxicokinetics**

The chemical can be absorbed following inhalation exposure (SIAR, 2006; IARC, 2010; Health Canada, 2013; HSDB). Most studies have been conducted in rats, which is the most sensitive species for pulmonary toxicity. Inhalation studies in rats have shown that the majority of the chemical is cleared from the lungs primarily by the bronchial pathway, as well as by transepithelial passage via alveolar macrophage clearance mechanisms. A lower clearance efficiency was observed in hilar lymph nodes, where the chemical remains as aggregated deposits. At lung burdens of > 0.5–1 mg per lung, particle clearance was impaired (lung overload) and the retention half-time was approximately 70 days (OECD, 2006; EC-HC, 2013; HSDB).

Several studies in rats indicate that ultrafine particles penetrate the alveolar epithelial cell barriers and cellular membranes much more readily than an equal mass of larger particles or agglomerates, resulting in different toxicological properties (OECD, 2006; SCCS, 2013).

In vitro studies in human alveolar type II cell lines indicate that the high surface area particles (<100 nm) of the chemical have an increased potential to generate free radicals compared with low surface area particles (>100 nm), thus inducing greater oxidative damage to cells (OECD, 2006; IARC, 2010; REACH).

**Acute Toxicity**

**Oral**

The chemical has low acute toxicity in animals following oral exposure.

In an oral gavage study conducted similarly to OECD Test Guideline (TG) 401, Sprague Dawley (SD) rats (male and female) were exposed to the chemical up to 10000 mg/kg bw. No mortalities or clinical signs of toxicity were observed. The median lethal dose (LD50) in rats was therefore reported to be >10000 mg/kg bw (OECD, 2006; REACH).

**Dermal**

The chemical is considered to have low acute dermal toxicity. The insoluble chemical particles are not expected to penetrate through the skin sufficiently to cause systemic toxicity effects.

No dermal LD50 values are available. However, no dermal toxicity was observed in skin irritation studies conducted in New Zealand White rabbits (OECD, 2006).

**Inhalation**

The available data are insufficient to make a conclusion on the chemical’s acute inhalation toxicity. Primary particle sizes may contribute to varying degrees of acute inhalation toxicity, due to lung burden from the particles.

The OECD report (2006) stated, ‘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”).’
In an acute inhalation toxicity study (non-guideline), Wistar rats were exposed (nose only) to the chemical at 4.6 mg/m\(^3\) for four hours. No mortalities were reported and there were no effects on the blood pressure, body temperature or animal behaviour (OECD, 2006; REACH). The median lethal concentration (LC50) in rats was >4.6 mg/m\(^3\).

In an acute inhalation study, rats were exposed to ultrafine (20 nm) and fine (200 nm) particle sizes of the chemical at 1 mg/m\(^3\) for seven hours. Observed inflammatory effects for the ultrafine particles were significant neutrophil influx in the lungs (1 %), increased total bronchoalveolar lavage (BAL) leukocytes and reduced lung tissue glutathione (GSH) post-treatment (>7 hours). In contrast, fine particles did not cause significant inflammatory effects (OECD, 2006; HSDB, REACH).

**Corrosion / Irritation**

Respiratory Irritation

Inhalation toxicity studies indicated respiratory effects in rodents following inhalation of the chemical. However, the available data are insufficient to classify the chemical as a respiratory irritant.

Several available inhalation toxicity studies in rats and mice reported inflammatory effects indicating respiratory irritation at concentrations >1.5 mg/m\(^3\) (EC-HC, 2013).

Skin Irritation

The chemical is not a skin irritant.

The chemical produced no skin irritation in New Zealand White rabbits (n = 3) in a test conducted according to the OECD TG 404. The intact and scarified skin of rabbits was treated with 0.5 g of the moistened chemical for four hours under occlusive conditions and observed for 72 hours. No signs of skin irritation (erythema, oedema) were observed (OECD, 2006; REACH).

Eye Irritation

The chemical is not considered to be irritating to the eyes. However, mechanical irritation from the insoluble particles is possible.

The chemical produced no eye irritation in New Zealand White rabbits (n = 6) in a test conducted according to the OECD TG 405. The left eye of each rabbit was instilled with 100 mg of the chemical and monitored up to 96 hours. No effects were observed (OECD, 2006; REACH).

SCCS (2013) stated that, at most, the chemical may be a slight eye irritant, based on a Bovine Cornea Opacity and Permeability (BCOP) test.

OECD (2006) stated that 'As superficial foreign bodies, carbon black particles may be slightly irritating mechanically'.

**Sensitisation**

Respiratory Sensitisation

The available data indicate that the chemical is not a respiratory sensitisier. However, it may have some adjuvant activity, similar to other particulate materials.

A test conducted in mice using various particles ('Kanto loam dust, fly ash, diesel exhaust particles [DEP], aluminum hydroxide [positive control], no particles [negative control]') and carbon black (30–200 nm; surface area 20.4 m\(^2\)/g), showed that all particles, including carbon black, increased immunoglobulin E (IgE) induction suggesting adjuvant activity (SCCS, 2013).

In a non-guideline study, female mice (n = 6/group) were exposed (via intranasal application) to ovalbumin alone or co-administered with ultrafine (<30 nm) or fine (>200 nm) particles of the chemical (total dose = 200 µg chemical, 3.3 mg/mL). Airway inflammation and adjuvant activity in the lungs were only observed with the ultrafine particles. No systemic antibody response was observed with exposure to the chemical. Adjuvant activity was indicated by the induction of Th2 immune response to ovalbumin and increased levels of cytokines (SCCS 2013; REACH). Although the study authors reported the chemical to be non-sensitising, the SCCS (2013) concluded that 'Carbon black was demonstrated to be able to act as Th2 adjuvant when used in combination with an antigen'.

Skin Sensitisation
The chemical is not considered to be a skin sensitiser. Negative results were reported for the chemical in a guinea pig test and a local lymph node assay (LLNA) for skin sensitisation.

The SCCS Opinion on the chemical (2013) considered all carbon black materials as nano-structured materials. It stated that therefore it was difficult to make a conclusion on the skin sensitisation potential of the chemical, as the chemical particles were unlikely to have 'penetrated the skin to reach the cellular targets of the immune system' in the studies evaluated.

The chemical was not found to induce dermal sensitisation in a Buehler test (OECD TG 406) in guinea pigs (SCCS, 2013).

In an LLNA test (OECD TG 429), the chemical (20–30 nm particle size) was topically applied to the ears of female CBA mice (n = 4/group, 25 µL/ear) at concentrations of 0, 0.25, 0.5, 1.0, 2.5 or 5.0 % (w/v) in propylene glycol for three consecutive days. No treatment-related symptoms were observed in any of the treated groups. The stimulation indices (SI) obtained were below three at all concentrations (1.13, 1.20, 1.23, 1.26, and 1.24 for the tested concentrations, respectively), indicating that the chemical was not a skin sensitiser (SCCS, 2013).

**Repeated Dose Toxicity**

**Oral**

The chemical is not considered to cause serious damage to health from repeated oral exposure.

In a 13-week study (OECD TG 408), Wistar rats (n = 10/sex/dose) were administered the chemical (20–30 nm particle size) via oral gavage, at doses of 0, 100, 300 or 1000 mg/kg bw/day. The no observed adverse effect level (NOAEL) was reported to be the highest dose tested (1000 mg/kg bw/day), as there were no adverse effects observed in this study.

Oral toxicity studies in CWF or C3H mice fed with the chemical for 12–18 months (10 % in diet), or for two years (2.05 g/kg diet) produced no treatment-related effects in organs or tissues (OECD, 2006; REACH).

**Dermal**

Based on the limited information available, the chemical is not considered to cause serious damage to health through repeated dermal exposure.

In a repeated dose dermal toxicity study, the chemical at 20 % (as an emulsion) was painted on the backs of male C3H mice, three times per week for 41 weeks (details not available). No changes in the organs or tissues were observed (OECD, 2006; REACH).

**Inhalation**

Based on the available data, the chemical is considered to cause serious damage to health from repeated inhalation exposure, warranting hazard classification. However, these effects were considered to be due to particle overload, rather than inherent chemical toxicity.

High exposure to the chemical may cause particle overload in the lungs of rats (Health Canada, 2013). Female rats, female mice and female hamsters inhaling the chemical (primary particles of 14 nm) at 1 or 7 mg/m³ repeatedly showed similar effects. A no observed adverse effect concentration (NOAEC) of 1 mg/m³ was established in mice and rats, based on lung effects at 7 and 50 mg/m³ for primary particles of 14 nm and 70 nm, respectively. These effects were persistent even after the recovery period of 11 months (SCCS, 2006).

In a repeated dose inhalation toxicity study, male Fischer 344 (F344) rats were exposed to the chemical (reported as respirable primary particles of 16 nm size with a specific surface area of 220 m²/g) at 0, 1, 1, 7.1 or 52.8 mg/m³, six hours/day, five days/week for 13 weeks. The rats were sacrificed immediately after the treatment period, and after three- or eight-month recovery periods. The NOAEC was established at 1.1 mg/m³ based on dose-related increases in lung damage and, above, 7.1 mg/m³. Prolonged retention of the chemical in the lungs was observed even after eight months. Lung inflammatory effects were evident based on neutrophil and macrophage accumulation within the alveolar region, epithelial hyperplasia and fibrosis (US EPA, 2005; OECD, 2006; EC-HC, 2013; REACH).

In another study, F344 rats (n = 135–139/sex/dose) were exposed (whole body) to the chemical (particle size (MMAD) reported as 1.95 µm (67 %) and 0.10 µm (33 %)) at 0, 2.5 or 6.5 mg/m³, 16 hours/day, five days/week for 24 months. Three males and three females were selected randomly for sacrifice after 3, 6, 12, 18 or 23 months. At ≥2.5 mg/m³, severe lung damage was observed, including lung tumours that increased linearly with exposure time (see Carcinogenicity). Exposure-related lesions such as alveolar epithelial hyperplasia and inflammation were also identified (OECD, 2006; IARC, 2010; REACH).

**Observation in humans**

A few studies investigated non-malignant effects in workers exposed to the chemical.
A large-scale study was conducted in workers in European production plants between 1987 and 1995, in three phases, with participation rates >90 % (>2000 workers) in the last two. The workers were assessed for a range of respiratory symptoms and lung function measurements. All data were adjusted to account for environment, age, height and smoking habits. Based on cumulative measurements of inhalable chemical (particle sizes not available), repeated exposure for 40 years at 1.0 mg/m³ TWA for eight hours was reported to cause minimal effects on lung function parameters. Multiple linear and logistic analyses of the data revealed lung and respiratory effects. The modelled data indicate the forced expiratory volume in one second (FEV1) of 48, 91 and 169 mL at 1, 2 and 3.5 mg/m³ exposure, respectively, were reduced in non-smoking males, indicating pulmonary effects. Lung effects have been associated with fibrogenic irritation (OECD, 2006; HSDB). Biomonitoring investigations involving physical examination of exposed workers in this study showed exposure-related symptoms such as cough and phlegm, lethargy, chest pains, skin irritation, reduced senses of smell and hearing, and discoloured sputum and stools. Respiratory effects included bronchitis, pneumosclerosis and myocardial dystrophy. These effects were found more frequently in workers with 2–4 years of exposure (EC-HC, 2013; HSDB).

An epidemiological study conducted in 1971 (location not stated) examined the oral mucosa of 600 workers exposed to the chemical (particle sizes not reported). The exposure concentrations were 2–3 times the maximum allowable concentration (MAC) in the furnace area and 4–11 times the MAC in the trapping and collecting areas. Increased keratosis and leukoplakia (thickened white patches) of the mouth (oral mucosa), cheeks, gums and tongue were reported (NIOSH 1978; REACH).

Limited data were available on ocular exposure in humans. After long-term use (at least for two years) of eye cosmetics containing the chemical, tiny pigment particles (size not indicated) were reported to cause black pigmentation of palpebral conjunctivae at the upper tarsal border of the eye. Two other reports indicated contradictory results—one reported chronic follicular-papillary reaction of the eyes, and the other reported no symptoms related to exposure (HSDB; REACH).

### Genotoxicity

The chemical is not considered to be directly genotoxic. The genotoxicity test results varied with the specific particle sizes tested and some in vitro assays with negative results were not considered relevant for the specific particle sizes tested (in the nano range) (SCCS, 2013). However, this assessment is not evaluating the primary nanoparticle effect of the chemical, as the particles are expected to exist as aggregate or agglomerate form. Therefore, genotoxic effects due to the nano particle nature of the chemical (as discussed in the SCCS, 2013 report) were not considered in this assessment.

In the SCCS report (2013), genotoxicity of the chemical was explained as being based on two principal modes considered ‘primary’ and ‘secondary’ (SCCS, 2013). Genetic damage induced by particles in the absence of pulmonary inflammation was defined as primary genotoxicity. The in vivo data suggested that secondary genotoxicity was due to various mechanisms such as inflammatory responses, oxidative stress and the generation of reactive oxidative species (ROS) or reactive nitrogen species (RNS) (SCCS, 2013). However, the SCCS report (2013) characterised the DNA strand breaks induced in the livers of mice (see Reproductive and developmental toxicity) and positive results in some in vitro studies as primary genotoxic effects (SCCS, 2013).

The chemical (particle size reported as 20–30 nm with a surface area of 200–260 m²/g and 98 % purity) gave negative results in two in vitro assays (OECD, 2006; SCCS, 2013; HSDB):

- the chemical did not induce mutations at the hypoxanthine-guanine phosphoribosyl transferase (hprt) locus of L5178Y mouse lymphoma cells; however, the SCCS report (2013) stated that an appropriate exposure time might not have been used; and
- the chemical did not induce micronuclei in cultured Chinese hamster ovary (CHO) cells, with or without metabolic activation.

The same test substance gave mixed results in Ames tests, with or without metabolic activation. The positive results observed in Ames tests were attributed to mutagenic impurities (polycyclic aromatic compounds, nitropyrenes) present in some of the chemical extracts (OECD, 2006; EC-HC, 2013).

Depending on the particle sizes tested, the chemical gave positive or negative results for single strand DNA breaks (negative results for 260 nm particles and positive results for 14 nm particles) in a comet assay with A549 human type II lung alveolar epithelial cells (SCCS, 2013). In another comet assay, the chemical (particle size = 12.3 ± 4.1 nm) induced DNA damage in primary mouse embryo fibroblast cells (SCCS, 2013).

In vivo assays in *Drosophila melanogaster* gave negative results for gene mutations and chromosomal aberrations (OECD, 2006; EC-HC, 2013; HSDB).

Several studies investigated DNA adduct formation in the lungs of rodents. Rats exposed to the chemical by inhalation at 6.2 mg/m³ for 12 weeks, showed a four-fold increase of DNA adducts in alveolar type II cells. The dose was high enough to elicit localised inflammation in the lungs (EC-HC, 2013; REACH).

In an in vivo genotoxicity study (non-guideline), male F344 rats were exposed via inhalation to the chemical (particles size reported as 16 nm with a 220 m²/g surface area) at 0, 1.1, 7.1, or 52.8 mg/m³, six hours/day, five days/week for 13 weeks. Increased mutations at the hprt locus were observed in alveolar type II cells at doses of 7.1 mg/m³ and above, and also after three- and eight-month recovery periods in the highest dose group. These mutations were triggered following lung tissue injury and inflammation, epithelial hyperplasia and pulmonary fibrosis. Furthermore, the addition of an antioxidant enzyme (catalase) inhibited mutations, indicating the role of cellular oxidants in mutagenesis. No adverse lung effects were detected at 1.1 mg/m³ (OECD, 2006; EC-HC, 2013; SCCS, 2013; REACH).

In another genotoxicity study, groups of female F344 rats (n = 3 or more/dose) were exposed to the chemical via inhalation at 1, 7 or 50 mg/m³ (particle size reported as 16 nm with a 300 m²/g surface area) or at 50 mg/m³ (particles size reported as 70 nm with a 37 m²/g surface area), six hours/day, five
days/week for 13 weeks. The lung DNA was extracted after the last exposure and after a 44-week recovery period in clean air. No DNA adducts were observed in lung homogenates of rats. An increased level of the oxidative DNA damage marker (8-hydroxydeoxyguanosine, 8-OHdG) was observed after prolonged exposure to the chemical (16 nm particles only) at 50 mg/m$^3$ (OECD, 2006; SCCS, 2013; HSDB; REACH). The lack of particle penetration into the lung epithelial cells or the small number of particles in the cell nuclei may have contributed to the lack of formation of DNA adducts (SCCS, 2013).

The SCCS report (2013) concluded that various secondary mechanisms (such as ROS generated by reactive particle surfaces, and impurities such as polycyclic aromatic hydrocarbons producing reactive metabolites to form DNA adducts) may have contributed to some positive genotoxic effects.

Carcinogenicity

The available data indicate carcinogenic effects in rats (lung tumours) following inhalation or intratracheal exposure to the chemical, but not from other routes of exposure. The neoplastic effects observed in rodents were attributed to the particle nature of the chemical and were more prominent with smaller particle sizes. High lung burdens from particle overload may have impaired the clearance mechanisms, leading to tumour formation.

Following its evaluation in 2006, the International Agency for Research on Cancer (IARC) classified the chemical as ‘Possibly carcinogenic to humans’ (Group 2B), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity in animals (IARC, 2010).

No neoplastic effects were observed in rats exposed to the chemical in the diet, for 15 months or two years. However, these oral studies were reported to be incomplete due to a lack of histopathological examinations and due to the small numbers of test subjects used (IARC, 1996; OECD, 2006).

In several dermal studies, the chemical was applied as a pure substance and in six concentrations in the range 6–60 % in acetone (doses not specified) to Swiss mice, twice weekly for 9–24 months. No skin tumours were observed (IARC, 1996; OECD, 2006).

Long-term inhalation or intratracheal exposure to the chemical caused carcinogenic effects in rats. In a carcinogenicity study (OECD TG 451), Fischer 344 rats were exposed to the chemical (particle sizes reported as 14 nm and 37 nm) via inhalation (whole body) at 2.5 or 6.5 mg/m$^3$, 16 hours/day for two years. Increased incidences of benign and malignant lung tumours were observed at 2.5 mg/m$^3$ due to high lung burdens. At 6.5 mg/m$^3$, based on logistic regression modelling, female rats developed lung tumours (adenomas and adenocarcinomas) at a significantly higher rate when compared with male rats (OECD, 2006; IARC, 2010; EC-CH, 2013; HSDB).

In two carcinogenicity studies, female Wistar rats had the chemical intratracheally instilled (particle sizes reported as 14 nm and 95 nm) at 1–3 mg, once a week for 15–17 weeks. Increased lung tumours and benign lesions (cystic keratinising squamous cell tumours) were observed in treated rats. Carcinogenic potency increased with smaller particle sizes and larger surface areas (OECD, 2006; IARC, 2010; EC-CH, 2013; HSDB). This was consistent with the results of inhalation studies, which relate particle sizes to toxicity effects (See Acute toxicity: Inhalation).

No evidence of carcinogenicity was observed in mice and, therefore, it was reported that the tumour effects might be species-specific (OECD, 2006).

The SCCS report (2013) concluded that the chemical ‘can induce malignant tumors in female rats after inhalation exposure or intratracheal instillations’. The carcinogenic potency was high with small particles (14 nm) compared with large particles (95 nm). A dose threshold could not be derived from the available animal carcinogenicity studies (SCCS, 2013).

Three cohort studies were conducted in production facilities for the chemical in the UK, USA and Germany. An increased risk of lung cancer was observed in workers in the UK and Germany but not in the USA. However, the data were reported to be inconsistent, as most of the studies did not account for the levels of exposure, smoking habits and occupational history (OECD, 2006; IARC, 2010; EC-CH, 2013).

The OECD report (2006) concluded that ‘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, and it appears that the rat is the most sensitive species to the effects of lung overload. At present the potential of the chemical to induce lung tumours in humans cannot be ruled out on theoretical grounds, although the epidemiological evidence does not suggest such a causal link’.

Reproductive and Developmental Toxicity

Based on the information available, no reproductive or developmental toxicity effects are expected from exposure to the chemical.

Two inhalation studies (non-guideline) using diesel exhaust in SD rats (exposure during gestational days (GD) 6–15) and New Zealand White rabbits (exposure during GD 6–18) did not show any maternal toxicity or developmental toxicity effects (OECD, 2006).

Pregnant mice exposed to the chemical (14 nm primary particles) at 42 mg/m$^3$ via inhalation for 10 hours had higher levels of DNA strand breaks in the liver, five and 24 days after exposure compared with control animals. Offspring from these mice also had higher levels of DNA strand breaks in the liver at weaning and in adolescents, compared with controls. Exposure did not affect gestation or lactation (SCCS, 2013). SCCS (2013) stated that, ‘oral and dermal exposure to carbon black is of little concern in relation to reproductive toxicity, however, inhalation exposure should be avoided’.

The OECD report (2006) concluded that, ‘Based on the available toxicokinetic principles, it is very unlikely that carbon black particles will reach the reproductive organs, the embryo or the foetus under in vivo conditions’.

**Risk Characterisation**

**Critical Health Effects**

The critical health effects for risk characterisation include potential inhalation toxicity from repeated exposure, due to the possible presence of small primary particle sizes. High lung burden from particle overload can impair the clearance mechanism, causing adverse health effects including possible induction of lung tumours.

The impurities in the chemical such as polycyclic aromatic hydrocarbons may cause various secondary mechanisms of toxicity by producing reactive metabolites.

**Public Risk Characterisation**

The chemical is reported to be used in domestic products in Australia. Risks are not expected if the chemical is used in formulations, such as paints, which bind the primary particles.

International uses indicated cosmetic use of the chemical. The cosmetic products containing the chemical are not expected to have free primary particles in the nano particulate size (up to 100 nm), but rather aggregates or agglomerates of particles (SCCS, 2013). Even if some primary particles are present or dislodged from aggregates or agglomerates, the inhalation risk from these is expected to be low. Cosmetic powder or spray formulations containing the chemical are not expected to be used in quantities that will cause particle lung overload. Therefore, the risk to the public is not considered to be unreasonable from using cosmetic products containing the chemical.

Considering the possible presence of toxic polycyclic aromatic hydrocarbons as impurities of the chemical, the SCCS report (2013) recommended that the purity of the chemical used in cosmetic products should be >97 %.

The domestic products containing the chemical are not expected to use a dry powder form of the chemical that could cause excessive lung exposure resulting in lung overload. Therefore, the risk to the public is not considered to be unreasonable.

**Occupational Risk Characterisation**

During product formulation, dermal, ocular and inhalation exposure of workers to the chemical might occur, particularly where manual or open processes are used. These can include transfer and blending activities, quality control analysis, and cleaning and maintaining of equipment. Worker exposure to the chemical at lower concentrations can also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic long-term health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise inhalation exposure to the chemical are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine appropriate controls.

The data available support an amendment to the hazard classification in HSIS (refer to Recommendation section).

**NICNAS Recommendation**

Assessment of the chemical is considered to be sufficient, provided that the recommended amendment to the classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

**Regulatory Control**

**Public Health**

Products containing the chemical should be labelled in accordance with state and territory legislation (SUSMP, 2015).

**Work Health and Safety**

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical hazards and environmental hazards.

<table>
<thead>
<tr>
<th>Hazard</th>
<th>Approved Criteria (HSIS)</th>
<th>GHS Classification (HCIS)</th>
</tr>
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<tbody>
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<thead>
<tr>
<th>Hazard</th>
<th>Approved Criteria (HSIS)</th>
<th>GHS Classification (HCIS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repeat Dose Toxicity</td>
<td>Harmful: danger of serious damage to health by prolonged exposure through inhalation (Xn; R48/20)</td>
<td>May cause damage to organs through prolonged or repeated exposure through inhalation - Cat. 2 (H373)</td>
</tr>
</tbody>
</table>

a Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].


* Existing Hazard Classification. No change recommended to this classification

**Advice for consumers**

Products containing the chemical should be used according to the instruction on the label.

**Advice for industry**

**Control measures**

Control measures to minimise the risk from inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical if valid techniques are available to monitor the effect on the worker’s health;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

**Obligations under workplace health and safety legislation**

Information in this report should be taken into account to assist with meeting obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((m)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (m)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice and Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.
References


Last update 24 April 2015