# Screening Assessment for the Challenge

# Carbon Black

**Chemical Abstracts Service Registry Number**
**1333-86-4**

**Environment Canada**
**Health Canada**

**June 2013**

## Synopsis

Pursuant to section 74 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999), the Ministers of the Environment and of Health have conducted a screening assessment of carbon black, Chemical Abstracts Service Registry Number[1] 1333-86-4. Carbon black was identified as a high priority for screening assessment and included in the Challenge initiative under the Chemicals Management Plan because it was considered to pose greatest potential for exposure of individuals in Canada and had been classified by other agencies on the basis of carcinogenicity. This substance met the ecological categorization criteria for persistence, but not criteria for bioaccumulation potential and inherent toxicity to aquatic organisms.

Carbon black is not naturally produced in the environment; it is manufactured by the controlled vapour-phase pyrolysis and partial combustion of gaseous or liquid hydrocarbons. In 2006, according to information reported under section 71 of CEPA 1999, 227 900 000 kg of carbon black was manufactured in Canada and 26 400 000 kg was imported. Carbon black is used primarily in the rubber industry as a reinforcing filler and as a pigment in a variety of products including inks, paints and coatings, and plastics. In Canada, carbon black can also be present in a limited number of food products, cosmetics, pharmaceuticals and natural health products, pesticides, and in food packaging. According to information reported under section 71 of CEPA 1999, 10 000–100 000 kg of carbon black was reported to be released to land, 10 000–100 000 kg to air, and 1 000–10 000 kg to water in 2006.

The human health risk characterization focuses on scenarios in which the general population can be exposed to carbon black by inhalation, given the limited potential for exposure and uptake via the oral and dermal routes and the lack of reported acute or chronic toxicity via these routes. No empirical data were identified on the concentrations of carbon black in the environment. Accordingly, exposure from environmental media in the vicinity of a carbon black manufacturing facility was characterized using dispersion modeling. With respect to consumer products, carbon black is used in a large number of paints and coatings, some with potential for inhalation exposure and exposure estimates were derived for these scenarios. Carbon black is also an ingredient in certain costume spray hair dyes and an exposure estimate was derived for this scenario.

Based principally on the weight of evidence-based assessments of international agencies, a critical effect for characterization of risk to human health for carbon black is carcinogenicity. Increased incidences of lung tumours were observed in female rats exposed by inhalation to the only or lowest concentration tested in one 11-month and two 2-year bioassays. However, the weight of evidence indicates that the induction of lung tumours in rats following carbon black exposure is caused by an excessive lung burden

[1] Chemical Abstracts Service Registry Number; The Chemical Abstracts Service Registry Number (CAS RN) is the property of the American Chemical Society and any use or redistribution, except as required in supporting regulatory requirements and/or for reports to the government when the information and the reports are required by law or administrative policy, is not permitted without the prior, written permission of the American Chemical Society.

(i.e., particle overload) due to overwhelming and impairment of clearance mechanisms, resulting in an oxidative state. No evidence was available to indicate carcinogenicity by the oral or dermal routes of exposure. Genotoxicity data indicate that carbon black has the potential to cause DNA and chromosome damage. However, these effects are probably mediated by indirect mechanisms involving inflammation resulting from particle overload in the lung, resulting in generation of reactive oxygen species, oxidative stress, and oxidative DNA damage. As the tumours observed in animals are considered unlikely to have resulted from direct interaction with genetic material, a margin of exposure approach is used to characterize risk to human health.

The critical effect level for acute pulmonary non-cancer effects by inhalation is a lowest observed effect concentration (LOEC) of 1 $mg/m^3$ in male rats exposed to carbon black for 7 hours, based on a significantly higher prevalence of inflammation and oxidative stress compared with controls. The critical effect level for chronic pulmonary non-cancer effects by inhalation is a LOEC of 0.57 $mg/m^3$, based on increased respiratory symptoms and decreased lung function measurements in individuals (male) exposed to carbon black in an occupational setting.

The margins between upper-bounding estimates of airborne exposure to carbon black in the environment or from consumer products and levels associated with respiratory effects are considered to be adequate to address uncertainties in the health effects and exposure databases. On the basis of the adequacy of the margins between conservative estimates of exposure to carbon black and critical effect levels in animals, it is concluded that carbon black does not meet the criteria in paragraph 64*(c)* of CEPA 1999, as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger to human life or health in Canada.

Carbon black is not soluble, and when released into water it is expected to eventually settle into sediments. Carbon black is resistant to hydrolysis, photolysis and biodegradation; it is therefore persistent in the environment. Accumulation in the tissues of living organisms is not an ecological concern, as carbon black's physical and chemical properties do not make bioaccumulation possible. It is expected to have very low potential for toxicity to aquatic organisms. Although no environmental monitoring data were identified, conservative exposure concentrations were estimated in surface water near industrial sources. Conservative risk quotient analysis, comparing predicted environmental concentrations with a predicted no-effect concentration, resulted in risk quotients of less than one, indicating that carbon black is unlikely to cause harm to aquatic organisms.

Based on the information available, it is concluded that carbon black does not meet the criteria in paragraphs 64*(a)* and *(b)* of CEPA 1999, as it is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends. Carbon black has been determined to meet the persistence criteria but not the bioaccumulation potential criteria, as set out in the *Persistence and Bioaccumulation Regulations*.

Based on the information available, it is concluded that carbon black does not meet any of the criteria set out in section 64 of CEPA 1999.

This substance will be considered for inclusion in the Domestic Substances List inventory update initiative. In addition and where relevant, research and monitoring will support verification of assumptions used during the screening assessment.

# Introduction

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) (Canada 1999) requires the Minister of the Environment and the Minister of Health to conduct screening assessments of substances that have met the categorization criteria set out in the Act to determine whether these substances present or may present a risk to the environment or to human health.

Based on the information obtained through the categorization process, the Ministers identified a number of substances as high priorities for action. These include substances that:

- met all of the ecological categorization criteria, including persistence (P), bioaccumulation potential (B) and inherent toxicity to aquatic organisms (iT), and were believed to be in commerce in Canada; and/or
- met the categorization criteria for greatest potential for exposure (GPE) or presented an intermediate potential for exposure (IPE) and had been identified as posing a high hazard to human health based on classifications by other national or international agencies for carcinogenicity, genotoxicity, developmental toxicity, or reproductive toxicity.

The Ministers therefore published a notice of intent in the *Canada Gazette*, Part I, on December 9, 2006 (Canada 2006), that challenged industry and other interested stakeholders to submit, within specified timelines, specific information that may be used to inform risk assessment, and to develop and benchmark best practices for the risk management and product stewardship of those substances identified as high priorities.

The substance, carbon black, was identified as a high priority for assessment of human health risk because it was considered to present GPE and had been classified by other agencies on the basis of carcinogenicity. The Challenge for this substance was published in the *Canada Gazette* on December 26, 2009 (Canada 2009). A substance profile was released at the same time. The substance profile presented the technical information available before December 2005 that formed the basis for categorization of this substance. As a result of the Challenge, submissions of information pertaining to the substance were received.

Although carbon black was determined to be a high priority for assessment with respect to human health and it also met the ecological categorization criteria for persistence, it did not meet the criteria for bioaccumulation potential and toxicity to aquatic organisms. Therefore, this assessment focuses on information relevant to the evaluation of risks to human health but also considers ecological risks.

Screening assessments focus on information critical to determining whether a substance meets the criteria as set out in section 64 of CEPA 1999. Screening assessments examine

scientific information and develop conclusions by incorporating a weight-of-evidence approach and precaution[2].

This final screening assessment includes consideration of information on chemical properties, hazards, uses, and exposure, including the additional information submitted under the Challenge. Data relevant to the screening assessment of this substance were identified in original literature, review and assessment documents, and stakeholder research reports, and from recent literature searches up to August 2010 for ecological effects and up to May 2010 for human health effects and exposure. Key studies were critically evaluated; modelling results may have been used to reach conclusions.

Evaluation of risk to human health involves consideration of data relevant to estimation of exposure (non-occupational) of the general population, as well as information on health hazards (based principally on the weight-of-evidence assessments of other agencies that were used for prioritization of the substance). Decisions for human health are based on the nature of the critical effect and/or margins between conservative effect levels and estimates of exposure, taking into account confidence in the completeness of the identified databases on both exposure and effects within a screening context. The final screening assessment does not represent an exhaustive or critical review of all available data. Rather, it presents a summary of the critical information upon which the conclusion is based.

This final screening assessment was prepared by staff in the Air Health Science Division at Health Canada and the Existing Substances Program at Environment Canada and incorporates input from other programs within these departments. The ecological and human health portions of this assessment have undergone external written peer review and consultation. Comments on the technical portions relevant to human health were received from Dr. Pam Williams, E Risk Sciences; Dr. John Christopher, CH2M Hill; and Dr. Bernard Gadagbui, TERA. Additionally, the draft of this screening assessment was subject to a 60-day public comment period. While external comments were taken into consideration, the final content and outcome of the screening assessment remain the responsibility of Health Canada and Environment Canada. Approaches used in the screening assessments under the Challenge have been reviewed by an independent Challenge Advisory Panel.

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[2] A determination of whether one or more of the criteria of section 64 are met is based upon an assessment of potential risks to the environment and/or human health associated with exposures in the general environment. For humans, this includes, but is not limited to, exposures from ambient and indoor air, drinking water, foodstuffs, and the use of consumer products. A conclusion under CEPA 1999 on the substances in the Chemicals Management Plan (CMP) Challenge Batches 1–12 is not relevant to, nor does it preclude, an assessment against the hazard criteria specified in the Controlled Products Regulations, which is part of the regulatory framework for the Workplace Hazardous Materials Information System (WHMIS) for products intended for workplace use. Similarly, a conclusion based on the criteria contained in section 64 of CEPA 1999 does not preclude actions being taken under other sections of CEPA or other Acts.

The critical information and considerations upon which the final assessment is based are summarized below.

# Substance Identity

## Substance Name

For the purposes of this document, this substance will be referred to as carbon black. Information on the identity of carbon black is summarized in Table 1.

Carbon black should not be confused with black carbon, which is an entirely different substance. Black carbon is formed through the incomplete combustion of fossil fuels, biofuel, and biomass (e.g diesel exhaust) and is often referred to as soot, whereas carbon black is produced by the controlled vapour phase pyrolysis of gaseous or liquid hydrocarbons (IARC 1996; US EPA 2005). A clear difference between carbon black and black carbon is the quantity of organic compounds (including polycyclic aromatic hydrocarbons, PAHs) that they contain; while carbon black typically contains less than 1% of solvent-extractable organic material (which can vary between types of carbon black), soot contains up to 82% (IARC 1996; OECD 2006).

**Table 1. Substance identity for carbon black**

| | |
|---|---|
| **Chemical Abstracts Service Registry Number (CAS RN)** | **1333-86-4** |
| **DSL name** | **Carbon black** |
| **National Chemical Inventories (NCI) names** [a] | *Lampblack (TSCA);*<br>*Carbon black (DSL, EINECS, ENCS, AICS, ECL, SWISS, PICCS, ASIA-PAC, NZIoC);*<br>*Inorganic, carbon black (PICCS);*<br>*GRAY, CARBON BLACK PIGMENT (PICCS);*<br>*CARBON BLACK PIGMENT (PICCS);*<br>*C.I. PIGMENT BLACK 7, CARBON BLACK (PICCS);*<br>*C.I. Pigment Black 7 (PICCS);*<br>*BLACK, CARBON BLACK PIGMENT (PICCS);*<br>*ACETYLENE BLACK (PICCS);*<br>*BASIS PIGMENT BLACK 7 (PICCS)* |
| **Other names** | See Appendix I |
| **Chemical group (DSL Stream)** | UVCB- Inorganic [b] |
| **Major chemical class or use** | N/A |
| **Chemical formula** | n(C) [c] |
| **Chemical structure** | Amorphous |
| **SMILES** [d] | N/A |
| **Molecular mass** | N/A |

[a] National Chemical Inventories (NCI 2009): AICS (Australian Inventory of Chemical Substances); ASIA-PAC (Asia-Pacific Substances Lists); ECL (Korean Existing Chemicals List); EINECS (European Inventory of Existing Commercial Chemical Substances; ENCS (Japanese Existing and New Chemical Substances); NZIoC (New Zealand Inventory of Chemicals); PICCS (Philippine

Inventory of Chemicals and Chemical Substances); SWISS (Giftliste 1 and Inventory of Notified New Substances); and TSCA (*Toxic Substances Control Act* Chemical Substance Inventory).

[b] This substance is a UVCB (**U**nknown or **V**ariable Composition, **C**omplex Reaction Products, or **B**iological Materials); i.e., it is not a discrete chemical and may be characterized by a variety of physical structures.

[c] Number of carbon atoms is variable.

[d] Simplified Molecular Input Line Entry System

Carbon black is a powdered form of elemental carbon composed of particles and fused particle aggregates (McCunney 2001). The primary carbon black particle, also known as the nodule, is approximately 10 to 500 nm in diameter (Wang 2003; OECD 2006). The molecular structure of carbon black consists of a condensed aromatic ring system of carbon atoms arranged in large sheets of variable size and alignment. These sheets are randomly stacked around an axis, held together by van der Waals forces, and overlaid to form structures called nodules (IARC 1996; Wang 2003). During the production process, the carbon black nodules coalesce to form aggregates, the primary dispersible unit, which are about 80–810 nm in size and consist of a few up to hundreds of particles (McCunney 2001; OECD 2006). Further along the production process, electrical forces (e.g., van der Waals forces) promote the formation of agglomerates 1–100 μm in diameter that consist of hundreds to thousands of adhering aggregates. This is the form of carbon black often encountered in commerce (ICBA 2004). Thermal black, along with exhibiting the largest primary particle size and lowest surface area of the commercial carbon blacks, has the lowest degree of aggregation (IARC 1996; Wang et al. 2003).

Aggregates are held together by a multitude of van der Waals forces holding sheets together, whereas agglomerates are held together by relatively few van der Waals bonds (Wang 2003). Hence, while agglomerates may dissociate into aggregates under certain circumstances (Aitken et al. 2004), aggregation that spontaneously occurs in manufacturing processes produces aggregates of average size, generally over 100 nm, that are effectively unbreakable (ICBA 2004). For example, in a series of experiments where intense mechanical energy was applied to carbon black products via uniaxial compression, elastomer mixing, or ultrasonication, there was little or no release of nodules and only limited fracture of the largest agglomerates (Gray and Muranko 2006).

Although the aggregation that spontaneously occurs in manufacturing processes produces unbreakable aggregates of an average size that is generally over 100 nm (IBCA 2004), carbon black can include a limited fraction of materials that are smaller than this i.e., nano-scale materials. It should be noted that this screening assessment does not specifically address the potential nano-size fraction nor does it make a clear distinction between the fate, exposure and effects of the nano-scale aggregates from that of larger particles. This screening assessment addresses the entire formulation including the size fraction which may include a nano-size fraction. Because of the current level of understanding, it does not make a distinction between the fate, exposure and effects of the nano-scale particles from those of larger particles or aggregates. The Government of Canada is currently in the process of examining methods to assess risks posed by nano-

scale materials in Canada, and is also in the process of developing a regulatory framework for nanomaterials. As such, any potential risks posed by carbon black at the nano-scale may be subject to separate review at a future date.

## Physical and Chemical Properties

The experimental and estimated values for physical and chemical properties of carbon black that are relevant to its environmental fate are listed in Table 2. Few experimental data are available for carbon black. Prediction of physical and chemical properties using quantitative structure–activity relationship (QSAR) models is not suitable for carbon black because of its unique inorganic structural properties.

**Table 2. Physical and chemical properties for carbon black (CAS RN 1333-86-4)**

| Property | Type | Value | Temperature (°C) | Reference |
|---|---|---|---|---|
| Physical state | Experimental | Solid: powder | | ICBA 2004 |
| Melting point (°C) | Experimental | 3652–3697 (sublimation) | | Weast 1983 |
| Boiling point (°C) | Not available | | | |
| Primary particle size (nm) | See Table 3 | | | |
| Surface area ($m^2/g$) | See Table 3 | | | |
| Density ($kg/m^3$) | Experimental | 1800–1860 (Relative density: 1.80-1.86) | Not available | Kotlensky and Walker 1960; US EPA 1980 |
| Vapour pressure (Pa) | Professional judgement | Negligible | Not available | Personal communication from International Carbon Black Association, as cited in OECD 2006 |
| Henry's Law constant ($Pa \cdot m^3/mol$) | Professional judgement | Negligible | | |
| Log $K_{ow}$ (octanol–water partition coefficient) (dimensionless) | Not applicable | | | |

| Property | Type | Value | Temperature (°C) | Reference |
|---|---|---|---|---|
| Log $K_{oc}$ (organic carbon–water partition coefficient) (dimensionless) | Not applicable | | | |
| Water solubility (mg/L) | Experimental | Insoluble | Not available | IARC 1996 |
| Organic solvents solubility | Experimental | Insoluble | Not available | Hawley 1981; ITII 1988 |

## Sources

Carbon black is not naturally produced in the environment; it is manufactured by the controlled vapour-phase pyrolysis and partial combustion of gaseous or liquid hydrocarbons (IARC 1996; US EPA 2005). Depending on the specific process by which it is manufactured, carbon black can be further classified as acetylene black, gas black, channel black, furnace black, lampblack, or thermal black (see Table 3) (IARC 1996; OECD 2006).

Carbon black can be manufactured by a variety of industrial processes including the oil-furnace black process, the thermal black process, the lampblack process, the acetylene black process, the gas black process, and the channel black process (Wang 2003; OECD 2006). The two dominant processes presently used to manufacture the majority of carbon black are the oil-furnace black process and the thermal black process (Wang 2003; OECD 2006; Baan 2007).

The oil-furnace process involves partial combustion of residual aromatic oils at high temperatures (1400–1800°C), which allows for the production of a broad range of carbon blacks. The most common feedstock for this process is decant oil from gasoline (McCunney 2001; Wang 2003). The carbon black containing gases are quenched twice with water and filtered to separate the unagglomerated carbon black from the by-product tail gas (McCunney 2001; Wang 2003).

The thermal black process, which produces the relatively coarse thermal black, involves decomposition of natural gas, coke oven gas, or liquid hydrocarbons in the absence of air (Wang 2003). The hydrocarbon feedstock is decomposed at high temperatures producing carbon black, hydrogen, methane, and other hydrocarbons (McCunney 2001).

The four major types of carbon black can be characterized by size distribution of the primary particles, the degree of particle aggregation and agglomeration, the various chemicals adsorbed on the particle surface (Table 3), and the functional groups located at sheet extremities (OECD 2006). The majority of carbon blacks contain over 97–99% elemental carbon, with chemically bound hydrogen, oxygen, nitrogen, and sulfur; less than 1% of the finished product consists of organic material such as PAHs (Table 3; OECD 2006).

**Table 3. Typical ranges of properties for four types of carbon black (from IARC 1996 and OECD 2006 [modified])**

| Property | Acetylene black | Furnace black | Lampblack | Thermal black | Gas black |
|---|---|---|---|---|---|
| Average aggregate diameter | Not reported | 80–500 nm | Not reported | 300–810 nm | Not reported |
| Average primary particle diameter | 35–50 nm | 17–70 nm | 50–100 nm | 150–500 nm | 13–29 nm |
| Surface area ($m^2/g$) | 60–70 | 20–200 | 20–95 | 6–15 | 90–320 |
| Density (g/mL) | Not reported | 1.80 | 1.77 | Not reported | 1.20–1.80 |
| Oil absorption (mL/g) | 3.0–3.5 | 0.67–1.95 | 1.05–1.65 | 0.30–0.46 | 2.8–9.2 |
| pH | 5–7 | 5–9.5 | 3–7 | 7–8 | 2.5–4.5 |
| Volatile matter (%) | 0.4 | 0.3–2.8 | 0.4–9 | 0.10–0.50 | 5–6 |
| Hydrogen (%) | 0.05–0.10 | 0.45–0.710 | Not reported | 0.3–0.5 | Not reported |
| Oxygen (%) | 0.10–0.15 | 0.19–1.2 | Not reported | 0.00–0.12 | Not reported |
| Benzene extract (%) | 0.1 | 0.01–0.18 | 0.00–1.4 | 0.02–1.7 | <0.1–0.3 (toluene) |
| Ash (%) | 0.00 | 0.1–1.0 | 0.00–0.16 | 0.02–0.38 | 0.02 |
| Sulfur (%) | 0.02 | 0.05–1.5 | Not reported | 0.00–0.25 | 0.3–0.5 |

Most carbon black is shipped in pellets 1–2 mm in diameter that are formed by the wet pelletization process; less than 0.1% produced is delivered in fluffy powder form (OECD 2006).

As of 2008, furnace black accounted for nearly 81% of the Canadian production of carbon black, while the remainder was thermal black (Glauser 2008). Information on the quantity of carbon black produced in, imported to, and exported out of Canada from 1996 to 2007 is presented in Table 4. In addition, the apparent consumption is calculated based on these values.

**Table 4. Quantity of carbon black manufactured, imported, exported, and consumed (apparent) in Canada for the years 1996–2007 (modified from Glauser 2008)**

| Year | Canadian supply and demand for carbon black (millions of kilograms) | | | |
|---|---|---|---|---|
| | Production | Imports | Exports | Apparent consumption |
| 1996 | 185.0 | 57.9 | 76.9 | 166.0 |
| 1997 | 205.3 | 83.2 | 101.7 | 186.8 |
| 1998 | 217.3 | 81.2 | 111.4 | 187.1 |
| 1999 | 217.9 | 87.9 | 98.4 | 207.4 |

| Year | Canadian supply and demand for carbon black (millions of kilograms) | | | |
|---|---|---|---|---|
| | **Production** | **Imports** | **Exports** | **Apparent consumption** |
| 2000 | 228.7 | 103.0 | 107.5 | 224.2 |
| 2001 | 215.4 | 107.7 | 97.6 | 225.5 |
| 2002 | 215.3 | 113.3 | 101.3 | 227.3 |
| 2003 | 205.1 | 129.9 | 107.1 | 227.9 |
| 2004 | 223.4 | 132.8 | 128.9 | 227.3 |
| 2005 | 253.7 | 134.0 | 132.7 | 255.0 |
| 2006 | 225.3 | 115.3 | 125.1 | 215.5 |
| 2007 | 222.9 | 112.8 | 140.4 | 195.3 |

Based on the survey submissions received in response to the notice published under section 71 of CEPA 1999 (Environment Canada 2010a), in 2006, 227 900 000 kg of carbon black was manufactured in Canada and 26 400 000 kg of carbon black was imported into the country. Note that the term “manufacture” as defined in the section 71 notice includes the incidental production of a substance at any level of concentration as a result of the manufacturing, processing, or use of other substances, mixtures, or products (Canada 2009). Also note that manufacturers, importers, and/or users of carbon black were required to respond to the section 71 of CEPA 1999 survey only if the carbon black was intended for any type of use within a residence (whether alone or in a mixture, product, or manufactured item), considered available for inhalation exposure, manufactured or imported in a quantity above the reporting threshold of 100 kg, or used at a quantity above the reporting threshold of 1000 kg (Canada 2009). Hence, the section 71 data will in all likelihood underestimate the actual quantity that was manufactured and imported.

## Uses

According to submissions reported under section 71 of CEPA 1999, over 10 000 000 kg of carbon black was used in Canada in 2006. Carbon black was reported to be used in a variety of products including paints, inks, coatings, plastics (e.g., polyethylene), rubbers, polymer film sheeting, fibreglass, sealants (i.e., water-repellent wood sealant and coloured sealant for concrete floors), polyvinyl compounds, powder coatings, carpet cushions, polyurethane flexible foam, and packing materials (Environment Canada 2010a). As stated previously, manufacturers, importers, and/or users of carbon black were required to respond to the section 71 of CEPA 1999 survey only if the carbon black was intended for any type of use within a residence (whether alone or in a mixture, product or manufactured item), considered available for inhalation exposure, used or obtained in a quantity above the reporting threshold (Canada 2009). For this reason, the section 71 data in all likelihood underestimates the actual quantity that was used.

Information on the use of carbon black in Canada for the year 2007 – the most recent year for which data were available - is presented in Table 5. A total of 195 300 000 kg of

carbon black was used, with the majority of that in the manufacture of tires (64%) and other rubber products (23%); this is consistent with global usage patterns.

**Table 5. Use profile for carbon black in Canada for 2007 (Glauser 2008)**

| Use application | Percentage of annual use of carbon black |
|---|---|
| Tire manufacture | 64% |
| Other rubber products (e.g., belts, hoses, and other automotive products) | 23% |
| Plastics | 9% |
| Liquid systems | 3% |
| Other non-rubber applications | 1% |

Globally, approximately 90% of carbon black produced is used in the rubber industry as a reinforcing filler in a variety of products including tires, tubes, conveyor belts, cables, rubber profiles, and other mechanical rubber goods (Wang et al. 2003; ChemInfo Services, Inc. 2009; HSDB 2009). An additional 9% is used as a pigment in inks, paints and coatings, plastics, fibre, and ceramics. The remaining 1% is used in hundreds of diverse products, including batteries, high temperature insulating material, and thickeners for certain high temperature petroleum and synthetic greases (Wang et al. 2003; ChemInfo Services, Inc. 2009; HSDB 2009). In addition, carbon black is used to impart electrical conductivity in rubber and plastics (McCunney 2001).

According to the U.S. Household Products Database (HPD 2009), carbon black is used in a variety of household products including paints (liquid and aerosol), primers, stains, paint protectors (i.e., undercoating), rubber gaskets, caulking, concrete repair and sealants, cement colour pigments, fibreglass insulation, pipe seals, shoe polish, laserjet printer toners, inkjet printer cartridges, electronic sealants, and diaper ointment.

In Canada, carbon black is used as a pigment or combustible carrier in a variety of pest control products (PMRA 2007).

In Canada, carbon black is listed as an approved food additive under Division 16 of the *Food and Drug Regulations* for the purpose of colouring foods in accordance with levels consistent with Good Manufacturing Practice (GMP) in a number of foods (Canada [1978]). A survey of the food colour industry conducted in 1972 indicated that the food industry's use of carbon black was limited to a small number of food products, such as imitation spice, steak seasoning, licorice products, ice cream, and sandwich cream biscuits. Most recently, the Canadian Spice Association informed Health Canada that carbon black is not used today by its members (email from Food Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced).

In food-packaging applications, carbon black acts as a pigment, under the names Pigment Black 6 (lampblack) and Pigment Black 7 (furnace black and channel black), and is commonly found in colour concentrates for plastics, epoxy-based enamels, paperboard, inks, can-end cement and sealants for the packaging of food. In food plants, carbon black is also used in adhesives, sealants, and primers with incidental food contact (email from

Food Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced).

Carbon black is listed in the *Food and Drug Regulations* under section c.01.040.2(3)(a) as a colouring agent permitted in drugs for internal and external use under the name Carbon Black (C.I. No. 77266) (Canada [1978]). Thus, this colouring agent is permitted (as a non-medicinal ingredient) in human and veterinary drugs. Although permitted to be used in drugs, carbon black is not listed in the Therapeutic Products Directorate's internal Non-Medicinal Ingredients Database as being present in any currently licensed human drugs. There are also no current identified uses of this substance in veterinary drugs (2010 email from Therapeutic Products Directorate, Health Canada, to Risk Management Bureau, Health Canada; unreferenced). Carbon black is listed in the Natural Health Products Ingredients Database as an acceptable non-medicinal ingredient in natural health products where it can be used as a colourant (NHPID 2010) and in the Licensed Natural Health Products Database as a non-medicinal ingredient present in three currently licensed natural health products (LNHPD 2011).

According to Health Canada's Cosmetics Notification System, carbon black is used in a variety of cosmetics, including eye liner, eye shadow, blush, mascara, eyelash glue, hair dye, hair styling products, hair removal wax, nail polish, soap, skin moisturizer, cologne, tanning products, tattoo ink, and costume (theatrical) spray hair dye (CNS 2010). Carbon black is also approved for use in tattoo ink for animal ear tags (US EPA).

## Releases to the Environment

Based on the survey submissions received in response to the notice published under section 71 of CEPA 1999 (Canada 2009), 10 000–100 000 kg of carbon black was reported to be released to land in 2006, 10 000–100 000 to air, and 1000–10 000 kg to water (Environment Canada 2010a). Again, as noted earlier, the manufacturers, importers, and/or users of carbon black were required to respond to the section 71 of CEPA 1999 survey only if the carbon black was intended for any type of use within a residence, considered available for inhalation exposure, used or obtained in a quantity above the reporting threshold (Canada 2009). Therefore, in all likelihood these are underestimates of actual quantities of carbon black that are released to the environment in Canada.

During carbon black production (specifically from the oil-furnace process), emissions may occur from dryer vents, the transport system vents, the clean-up system vents, and from cleaning, spills, and leaks (OECD 2006). Carbon black production plants generally employ bag filters to reduce emissions into the atmosphere, and discharges from bag filters that are maintained and used under normal conditions reportedly contain less than 50 $mg/m^3$ carbon black (Johnson and Eberline 1978; IARC 1996).

Releases to the environment also occur during the use of carbon black in industrial processes. For the year 2006, the Canadian Chemical Producers' Association (now called Chemistry Industry Association of Canada) emissions inventory reported total carbon black emissions from member companies of 1140 kg (CCPA 2006). None of the companies that reported releases were producers of carbon black. Canada's National Pollutant Release Inventory (NPRI) and the United States Toxics Release Inventory (USTRI) databases do not contain environmental release information on carbon black (NPRI 2006; USTRI 2006)

A major source of carbon black to the environment from products is as a component of tire dust (also known as tire debris), where it is bound within an elastomer complex. Approximately 22% of a tire is composed of carbon black, and on average a tire loses from 10 to 20% of its weight during use over its service life (OECD 2006). As it is bound within the elastomer complex, carbon black is unlikely to be released from tires as an unbound particle through wear or abrasion (US EPA 1976; OECD 2006; ChemRisk, Inc. and DIK, Inc. 2008).

Tire manufacturing plants release limited amounts of particulate matter to the atmosphere, which presumably includes some carbon black (Environment Canada 2010a). Carbon black releases to the aquatic environment are limited to industrial wastewater resulting from handling the product before it is mixed with rubber compounds.

When they reach the end of their service life, scrap tires are retreaded and reused as whole tires, shredded tires, disposed of, or burned for energy production. The leachability

of carbon black from whole tires and shredded tires is very limited owing to its insolubility. Burning tires as a source of energy is a growing practice in Canada among cement plants (Cement Association of Canada 2010). Despite the presence of pollution control devices, some dust containing carbon black may escape into air. Cement plant furnace temperatures range between 1700°C and 2200°C (Cement Association of Canada 2010). Since its degradation temperature (3652–3697°C, Table 2) is higher than the furnace temperatures, carbon black will not be degraded during tire burning.

In smaller proportions, carbon black is also used as a pigment for printer inks and paint. Carbon black is manufactured and imported into Canada for pigment production (Environment Canada 2010a). Releases to the environment are essentially associated with industrial effluents. Inks are a consumer product used in commercial and individually owned printers. Ultimately carbon black on printed paper and in leftover ink cartridges and paint residues will be disposed of mainly in landfills, but could also be released to wastewater.

## Environmental Fate

QSAR models that are usually used by Environment Canada to evaluate a chemical's overall behaviour in the environment cannot be used for carbon black. A fate analysis based for example on log $K_{ow}$ and $K_{oc}$ is not applicable to carbon black. Typical fugacity modelling is also not applicable for this substance because, as for all non-volatile chemicals, this compound exerts zero partial pressure and fugacity in air (Diamond et al. 1992). Therefore, a qualitative approach based largely on professional judgement has been applied.

Carbon black has a negligible vapour pressure and is not expected to partition into air.

The fate of carbon black in the environment depends on the compartment into which it is released. Being a solid particle, carbon black is expected to eventually end up in sediments and soils. When released to soil, carbon black will mainly remain in the soil with some of the substance being transported by flowing water (runoff) to local surface waters.

If released to water, it is expected that carbon black will be present as suspended particulate matter, which will eventually settle into bottom sediments. However, if sources of continuous release to turbulent waters exist, it is reasonable to expect that suspensions of the substance might be continuously present. Owing to its stable C–C network, carbon black is not soluble and is not expected to breakdown under conditions typically found in surface waters, and in fact it will have a tendency to aggregate and agglomerate to avoid contact with water (Hawley 1981; IARC 1996). Depending on the process used to manufacture carbon black, the extremities of carbon ring sheets will bear different functional groups. However, none of these functional groups is expected to favour the solubilisation of the substance in water or in organic solvents (Hawley 1981; IARC 1996).

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# Persistence and Bioaccumulation Potential

## Environmental Persistence

Given that the majority of carbon black is expected to quickly reach soil or sediment due to settling of suspended particulates in air or water, its stability in these media is particularly relevant. However, no experimental biological degradation data for carbon black in these or other media have been identified. Since this substance is not amenable to modelling procedures relating to persistence, the following analysis was undertaken based on expert judgement and information gathered from a literature review.

According to OECD 2006, carbon black is neither photodegradable nor biodegradable. Hydrolysis, defined as the decomposition of a chemical compound by reaction with water, is not likely to occur because of carbon black's insolubility in water.

In the same way, for biodegradation processes to be effective, a substance must first be absorbed by a living organism. A substance needs to first cross the cell membrane to be absorbed, which means that it needs to be soluble either in water or in lipids so that transport can occur either through carrier proteins or directly through the cell membrane. Absorption and biodegradation is unlikely for carbon black because of its insolubility in both water and organic solvents.

Because carbon black is not degradable in any medium or by biota (Wang et al. 2003), it is considered persistent in the natural environment. Therefore, carbon black meets the persistence criteria in air, water, soil, and sediment as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

## Potential for Bioaccumulation

Bioaccumulation potential in aquatic exposures is typically quantified by determining either a bioconcentration factor (BCF) or a bioaccumulation factor (BAF). A BCF and BAF may be measured experimentally as ratios between concentrations in environmental media (typically water) and those in associated organisms at steady state. No such experimental data have been found from literature searches. Additionally, QSAR models cannot be used for carbon black; therefore, BCF and BAF values cannot be modelled.

Physical and chemical properties of carbon black do not indicate a potential to diffuse through membranes of aquatic organisms because of its low solubility in both water and organic solvents. The same conclusion is reached by OECD (OECD 2006) which noted that a relevant bioaccumulation potential of carbon black is not expected based on its insolubility in organic solvents and in water.

Furthermore, since the aggregate diameter of carbon black varies between 80 nm and 810 nm (Table 3), bioaccumulation of particulate carbon black is not likely owing to the large diameter of the solid aggregate particles.

Based on the available evidence relating to its chemical and physical properties, carbon black does not meet the bioaccumulation criteria (BAF or BCF ≥ 5000) as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000).

# Potential to Cause Ecological Harm

## Ecological Effects Assessment

As described previously, carbon black is expected to quickly reach soil and sediments when released into the environment. Settling of particles into soil and sediment from air and water is expected because of its particulate character, its high density as well as its low solubility and low volatility. However, carbon black may be present to some extent in the water column when kept in suspension, in turbulent river water for example. Carbon black is expected to have a low bioaccumulation potential. Experimental ecological effects data are mainly available for aquatic pelagic organisms and are summarized in Table 6. These data indicate that carbon black is not hazardous to these organisms.

Typical aquatic toxicity tests cannot be performed with this substance as carbon black is insoluble in water; hence modified test methods are required. Tests may be considered acceptable if the insolubility of the test compound is addressed by keeping the solid substance particles in contact with the organisms. One way to do so is to maintain suspensions of carbon black through continuous stirring or aeration; organisms are thus constantly in contact with agglomerated carbon black particles. Using this method, obstructive physical effects are not dissociable from chemical effects on organisms. Nominal concentrations in such tests (based on substance loadings) are used to establish exposure concentrations. Another method consists of preparing a carbon black suspension that is pH-adjusted and then filtered. In such tests the actual exposure concentrations are unknown because a variable amount of carbon black is captured on the filters. The advantage of this method is that it helps to maintain physical conditions (pH and homogeneity of the solution) and therefore avoids physical deleterious effects. Some types of carbon black diminish the pH of the test solution as a result of the dissociation of the acid functional groups located on the extremities of the carbon atom sheets. Large pH variations can be stressful for test organisms or can be lethal for others. Thus results from studies in which pH variations are expected to have been the cause of observed toxic effects have been rejected.

Although both methods described above are not typical, their results may be considered acceptable. Exposure to carbon black suspensions helps in assessing physical deleterious effects while exposure to filtered solutions provides additional information on any chemical effects that this substance may have.

**Table 6. Empirical data for aquatic toxicity of carbon black**

| Test organism | Carbon black product; solubilization method | Carbon black type | Test type (duration) | Endpoint | Value (mg/L) | Reference |
|---|---|---|---|---|---|---|
| Zebrafish (*Brachydanio rerio*) | Special Black 4; suspension | Gas Black | Acute (96 hours) | No effect (morphological or behavioural anomalies) | ≥1000 | Degussa AG 1992a as cited in OECD 2006 |
| | Special Black 4; filtrate | Gas Black | | | ≥10 000 | |
| | Corax N220; suspension | Furnace Black | | | ≥1000 | Degussa AG 1991 as cited in OECD 2006 |
| Orfe fish (*Leuciscus idus*) | Printex U; filtrate | Channel Black | Acute (48 hours) | No effect | ≥8000 | Degussa AG 1979a as cited in OECD 2006 |
| | Special Black 4; suspension | Gas Black | Acute (96 hours) | No effect | ≥1000 | Degussa AG 1979b as cited in OECD 2006 |
| | Printex G; suspension | Channel Black | Acute (96 hours) | No effect | ≥1000 | Degussa AG 1979c as cited in OECD 2006 |
| | Printex 400; suspension | Channel Black | Acute (96 hours) | No effect | ≥1000 | Degussa AG 1979d as cited in OECD 2006 |
| | Printex G, Printex U, Printex 400 and Special Black 4; suspension | Gas Black and Channel Black | Acute (14 days) | No effect | ≥5000 | Degussa AG 1978 as cited in OECD 2006 |
| Water flea (*Daphnia magna*) | Special Black 4; filtrate | Gas Black | Acute (24 hours) | No effect (mobility) | 3200 | Degussa AG 1992b as cited in OECD 2006 |
| | | | | LOEC [a] (change in mobility) | 5600 | |

| | | | | | | |
|---|---|---|---|---|---|---|
| Algae (*Scenedesmus subspicatus*) | Printex 30; filtrate | Furnace Black | Chronic (72 hours) | No effect (growth inhibition) | ≥10 000 | Degussa-Hüls 1999 as cited in OECD 2006 |

[a] LOEC: lowest no observable effect concentration

As noted in the Sources section, furnace black and thermal black are the most common manufactured forms of carbon black worldwide. But, because of the limited amount of data available all toxicity data were considered—regardless of the form tested—when the predicted no-effect concentration (PNEC) was derived.

The ability of carbon black to modify solution pH is a chemical property that varies between carbon black types. Added to water, gas black has the potential to acidify a solution to pH levels as low as 3.5–4.5 (Table 3). Effects are observed from a pH of 5.5 and below; certain insects and crustaceans are likely affected. When the pH drops below 5.0, many fish species don't survive (Tremblay and Richard 1993). At the opposite end of the spectrum, furnace black can increase solution pH up to 9–10 when massively added to water (Evonik Industries 2009). These differences are probably due to differences in the functional groups (acidic or basic) attached at the ends of the carbon layers. Deleterious physical effects related to pH are unlikely to be observed in Canadian aquatic ecosystems because the forms with the most potential to reduce pH, such as gas black, are used in negligible amounts in Canada (Glauser 2008). Furthermore, the quantities of carbon black needed to reach effect-level pH are several orders of magnitude higher than the quantities of this substance estimated to be released to Canadian water bodies (see Ecological Exposure Assessment section). Consequently, as previously noted, results from studies in which pH changes are expected to have been the cause of observed toxic effects have been rejected.

A conservative PNEC was derived from the lowest no-effect toxicity value identified for carbon black in suspension, since results from studies with suspensions are the most environmentally realistic. Test organisms that were exposed to suspensions are zebrafish and orfe fish. Gas black, furnace black, and channel black were tested under the same experimental conditions. Carbon black in water was allowed to equilibrate for 20 hours under continuous stirring. The exposure duration was between 14 and 96 hours for all tests. No effects were observed for both zebrafish and orfe fish at 1000 mg/L. This value was selected as the critical toxicity value, and was divided by an assessment factor of 50 to account for uncertainties related to interspecies and intraspecies variability of aquatic organisms in sensitivity and extrapolation from results for short-term laboratory studies to a long-term no-effects concentration in the field (aquatic media). This calculation for carbon black in suspension in water resulted in a PNEC of 20 mg/L.

In cases where filtrate from extensively stirred suspensions was used, no toxic effect was observed for *Daphnia* exposed to up to 3200 mg/L of carbon black. At 5600 mg/L, some of the animals were still mobile but swam slower. An effect concentration inducing a change in mobility in 50% of the organisms and plants ($EC_{50}$) was estimated to be between 5600 mg/L and 10 000 mg/L. The test was conducted according to OECD Test

Guideline 202 (OECD 1984). However, although the protocol suggests to avoid adjusting pH, the pH range resulting from this test was outside that for natural water pH (6–9) and below the recommended pH (7–9) for *Daphnia* (Kring and O'Brien 1976). Gas black was used for this study, so the pH decreased as the substance concentration increased. Since the pH for the studies was below that recommended for *Daphnia*, and since pH can have a significant influence on test organisms, the study was not considered acceptable for derivation of the PNEC.

Studies on blue mussel (*Mytilus galloprovincialis*) hemocytes showed biochemical effects at concentrations of a few orders of magnitude below those reported in the studies mentioned in Table 6 (between 1 and 10 mg/L) (Canesi et al. 2008; Canesi et al. In press). Biochemical effects observed were increased lysozyme activity and nitrite production, higher reactive oxygen species production, and decreased mitochondria number. Direct correlations between carbon black concentration and inflammatory effects were suggested by the authors. However, acute and chronic tests relating to mortality, growth, or reproduction are more typically used as a basis for determining a critical toxicity value than are biochemical studies because of the difficulty of relating biochemical effects to effects at higher levels of biological organization (e.g., whole organism). Hence, this study was not considered to derive the PNEC for aquatic organisms.

Carbon black is widely used in tires and will typically be found at approximately 30% in tire dust (OECD 2006). As a result of tire abrasion, carbon black could reach roadsides through run-off from roads. Carbon black in tires is expected to be bound in the rubber material and not to be readily bioavailable. Toxicity tests performed with tire dust are described below as evidence of limited effect – due to low bioavailability or to low toxicity-of carbon black from tire dust. Exposure concentrations in these tests are expressed as the mass of tire/tire dust by volume of water added; the concentrations tested are not necessarily representative of the concentrations that could be expected in run-off from roads.

A study from the Basel Convention (Basel Convention 1999) examined the effects of tire powder on fish, daphnids and algae to assess the potential effects of traffic on water bodies located near busy roads. Carbon black constituted 21.5% of the tires studied (30% of the tire dust used in the experiment). No signs of toxicity (mobility, morphological anomalies, and growth) were observed at exposure concentrations of up to 58 000 mg of tire dust/L of water for zebrafish, 68 000 mg of tire dust/L of water for *Daphnia*, and 13 000 mg of tire dust/L of water for algae. Experiments were not carried out at higher concentrations.

Toxicity tests have been performed on tire dust in aqueous suspensions, filtrate from suspensions of tire dust, and water taken after soaking used and new tires (Smith et al. 1969; Abernethy 1994; Gualtieri et al. 2005; Wik et al. 2006). These studies used different methodologies which resulted in variable effects that are difficult to interpret. In particular, it was found that the tire brand tested has a large effect on toxicity (Wik et al. 2006). In addition, some tests found new tires to have a greater toxicity to aquatic

organisms, while other tests showed the opposite, depending on the material tested. Also, studies were performed either on new and used tires, tire dust and chips, or whole tires that had been soaked in water for periods varying between 24 hours and 32 days. These variations in the test material used partly explain the observed variations of tire toxicity. As the content of carbon black in the tires tested varied, it is not possible to know with certainty whether the toxicity demonstrated in these studies is due to carbon black, other substances that could have been present in or on the tires, or both. Therefore, no toxicity conclusion can be reached regarding the effects of carbon black in tires and tire dust.

As mentioned earlier, if carbon black reaches a water body, it will sink to bottom sediments due to its low water solubility and high density. Thus, sediment-dwelling organisms could be exposed to this substance. However, no toxicity data specific to sediment-dwelling organisms are available for carbon black.

Carbon black will also deposit from air to surface soil. No toxicity data for tests conducted with terrestrial organisms exposed directly to carbon black are available. However, results of tests conducted with earthworms have been reported for filtered extractions of tire dust. The tire dust contained approximately 21% carbon black. The tests, in which 100 g of tire dust was shaken in one litre of water for 24 hours and then filtered, showed no effects on earthworm survival (Basel Convention 1999).

## Ecological Exposure Assessment

No data concerning concentrations of carbon black in water, sediments or soil in Canada or elsewhere have been identified.

### *A – Industrial Release*

Information on Canadian releases of carbon black to the environment is presented in the previous section Releases to the Environment.

A site-specific exposure analysis was conducted for the aquatic compartment at seven industrial sites where carbon black was used for manufacturing tires or rubber, used as a pigment, or manufactured for sale for various industrial purposes (Environment Canada 2010d). These sites represented the top seven industrial users or manufacturers identified from over 50 companies that responded to the CEPA section 71 Notice (Environment Canada 2010a). Each user or manufacturer reported an annual consumption or production quantity of carbon black in the range of 1 000 000 to 100 000 000 kg. The selection of these sites is therefore expected to represent a set of realistic worst-case release scenarios across Canada, based on a general assumption that the quantity released is proportional to the quantity consumed or produced.

In this site-specific exposure analysis, each site included one facility, a wastewater treatment plant, and a receiving water body. The predicted environmental concentration (PEC) in the receiving water body was estimated by dividing the concentration of carbon

black in the treated wastewater effluent by a maximum dilution factor of 10 provided by the receiving water. The concentration in the treated wastewater effluent was preferably based on data reported under section 71 of CEPA. Where reported data were missing, this concentration was calculated based on: 1) an estimated fraction of the carbon black lost from the facility where it was used to a local municipal wastewater treatment plant; 2) an estimate of the wastewater treatment plant's removal efficiency; and 3) the wastewater treatment plant effluent flow rate. The loss fraction was conservatively estimated (based on expert judgement) to be 0.5% that resulted from chemical container handling operations and the industrial processes relevant to the facilities under consideration. The carbon black removal efficiency from influent resulting from the wastewater treatment process was conservatively estimated to be 50% where lagoons or primary treatments exist. The effluent flow of a local wastewater treatment plant was estimated if this information was not otherwise publically available. An assumption for the frequency of release from the facility of 250 days/year was used when estimating PECs. Scenarios also assumed that the municipal wastewater treatment plant that receives industrial wastewater is in operation all year-long (350 days) in most locations.

Based on the above assumptions, the PECs are estimated to be in the range of 0.08 to 7.5 mg/L for water bodies receiving releases from the top seven industrial users or manufacturers of carbon black in Canada. Since only the top seven users or manufacturers were considered in this site-specific analysis, along with certain upper bound assumptions, the PEC values obtained are considered to represent worst case exposure.

***B – Consumer Release***

As carbon black is found in consumer products and can be released to water, the tool Mega Flush (Environment Canada 2009) was employed to estimate carbon black concentration in multiple water bodies receiving wastewater treatment plant effluents that may contain printer ink and paint (Environment Canada 2010c). Inks and paints were considered in this scenario because they are more likely than rubber products to be subject to "down-the-drain" releases of carbon black.

The Mega Flush tool predicted carbon black concentrations in surface water at approximately 1000 release sites across Canada based on several mostly very conservative assumptions that include:

- 100% release of the substance from inks and paints
- wastewater treatment plant removal rate of 0 % in all cases
- number of annual release days of 365 days/year
- receiving water dilution factor in the range of 1 to 10.

Conservative PECs for carbon black in the receiving water bodies were thus estimated to be as high as 6.6 mg/L. This estimate is based on a total of 4 336 447 kg/year for the quantity of the substance used as a pigment, dye, or ink by consumers based on information provided by industries that produce or import carbon black for this purpose

(Environment Canada 2010a). The equation and inputs used to calculate the PECs are described in Environment Canada (2010d).

## Characterization of Ecological Risk

The approach taken in this ecological screening assessment was to examine relevant scientific and technical information and develop conclusions based on a weight-of-evidence approach and using precaution as required under CEPA 1999. Lines of evidence considered include results from a conservative risk quotient calculations, as well as information on persistence, bioaccumulation, toxicity, sources, and fate of the substance.

Carbon black is expected to be persistent in air, water, soil, and sediment. It is also expected to have a very limited bioaccumulation potential. The high manufacturing and importation volumes of carbon black in Canada, along with information on its uses, indicate potential for widespread release into the Canadian environment. Once released into the environment, carbon black will end up mainly in sediment and soil. However, it may be present in the aquatic compartment when it is kept in suspension—in turbulent river water, for example.

Carbon black has also been demonstrated to have low potential for toxicity to aquatic organisms. Physical effects related to a change in the pH of the water column are unlikely to be observed in Canadian aquatic ecosystems because the forms of carbon black having the highest potential to reduce pH, gas black and lampblack, are used in negligible amounts in Canada (Glauser 2008). Furthermore, the quantities of carbon black needed to reach effect-level pH changes are several orders of magnitude higher than what is estimated to be in contaminated Canadian receiving waters.

Conservative risk quotient calculations (PEC/PNEC) for carbon black indicate that exposure values are unlikely to be high enough to cause harm to aquatic organisms in Canada. Using the PNEC based on toxicity data for carbon black suspensions, risk quotients for the top seven industrial users or manufacturers of carbon black are estimated to be 0.004, 0.05, 0.05, 0.13, 0.14, 0.14, and 0.38. For the consumer use and release scenario, the maximum exposure concentration estimated based on highly conservative assumptions is 6.6 mg/L, which results in a risk quotient of 0.33.

As mentioned earlier, carbon black is expected to partition to sediments. However, no environmental monitoring data for sediments, or toxicity data specific to sediment-dwelling organisms are available for this substance. Considering that no effects were observed on pelagic aquatic organisms at concentrations up to 10 000 mg/L, effects in sediment-dwelling organisms from exposure to carbon black in Canada are unlikely. Carbon black will also deposit on roadside surface soil. No effects on survival were seen for one soil organism (earthworm) when exposed to filtered extractions of tire dust. Although the dust contained 30% carbon black, this substance may have been tied up in an unavailable form in the tire matrix. Hence, uncertainty remains as to whether carbon black is harmful to terrestrial organisms.

For wildlife, inhalation exposure from industrial releases of carbon black can occur. No ecotoxicological studies by this route were identified. However, as presented in the Characterization of Risk to Human Health section, conservative estimates of concentrations near manufacturing sources are several orders of magnitude less than the lowest acute and chronic LOECs reported in laboratory rodents. Therefore, airborne exposures in the vicinity of industrial sources are unlikely to be high enough to cause harm to wildlife.

In surface water, sediments, soil and air, carbon black is thus unlikely to cause ecological harm in Canada.

**Uncertainties in Evaluation of Ecological Risk**

Given that more than one structure (i.e., multiple forms of carbon black) exist, it is recognized that structure-related uncertainties exist for this substance. Toxicity data for as many forms of carbon black as possible were considered to address uncertainties related to its physical structure.

The experimental conditions used in aquatic toxicity tests were not standard. Because of the very low solubility of carbon black, standard tests could not be performed. However, methods that involve stirring or aeration to maintain suspensions of material and filtering to remove the suspended matter were used to address this problem. The suspension method likely increases the potential toxicity to aquatic organisms because physical effects may contribute to any toxicity observed.

Also regarding ecotoxicity, based on the expected partitioning behaviour of this chemical in the environment, the significance of soil and sediment as important media of exposure is not well addressed by the effects data available. Indeed, most of the effects data identified apply primarily to pelagic aquatic organisms, although the water column may not be the medium of primary concern based on the partitioning of carbon black. However, considering that no effects are observed for aquatic organisms at very high exposure concentrations, it is unlikely that effects would be observed for soil and sediment dwelling organisms.

The overall assessment conclusion was reached despite the conservative assumptions that were made in response to uncertainties encountered in the assessment. A key uncertainty relates to the lack of empirical data on environmental concentrations of carbon black in Canada, which was addressed by predicting conservative concentrations in water using an industrial exposure model. There is also uncertainty associated with the PNEC used in the risk quotient calculation, because of the lack of measurable deleterious effects in toxicity tests. Also, final concentrations to which test organisms were exposed are unknown because carbon black concentrations were not measured in filtrated test waters, and some

carbon black would remain on the filter. This uncertainty was partly addressed by dividing the critical toxicity value by an assessment factor of 50.

Available information is currently not sufficient to derive a quantitative estimate that would help determine the importance of carbon black entering the Canadian market as a component of manufactured items and/or consumer products. However, it is anticipated that because of the conservative approaches adopted in this assessment, the quantities of carbon black released to the various environmental media would not be significantly larger than those estimated here. It is also recognized that releases from waste disposal sites are possible and could contribute to overall environmental exposure, but any resulting increases in exposure concentrations are expected to be slight.

# Potential to Cause Harm to Human Health

## Exposure Assessment

This exposure assessment focuses on scenarios in which the general population can be exposed to carbon black by inhalation, rather than via the dermal or oral routes. Although carbon black is used in some cosmetics, including some personal care products with potential skin contact (see section on Uses), as a particle that is not soluble in either water, organic solvents, or biological fluids, carbon black would not be expected to be absorbed through the skin (US EPA 2005). Its use as a tattoo ink also indicates that any appreciable systemic absorption via the skin is unlikely, because tattoos are stable in the skin for many years. With respect to oral exposure, carbon black is approved or notified for use as a pigment or colourant in a number of food products, pharmaceuticals, and natural health products, but in most cases it is only used in a very limited number of these products, or not at all (see section on Uses). Moreover, as discussed further in the subsequent section (Health Effects Assessment) and in Appendix III, inhalation exposure to carbon black is clearly associated with pulmonary effects, whereas no effects have been reported after acute or chronic exposure via the oral or dermal routes. This was despite the fact that in the oral studies, the administered doses were approximately three orders of magnitude greater than those that induced effects in inhalation studies.

In this section, exposure to carbon black in the general environment is discussed first, followed by exposure from consumer products. Details of the exposure estimates summarized in this section are presented in Appendix II.

### *Environmental Media*

In the available literature and in government reports, no empirical data were identified regarding directly measured concentrations of carbon black in environmental media (air, water, soil, and sediment) in Canada or elsewhere. In addition, environmental concentrations have not been estimated using fugacity models because carbon black is not suitable for such modelling, since it is an inorganic substance that has negligible volatility and is insoluble in both water and organic solvents.

Environmental exposures are considered to be greatest in the vicinity of industrial facilities that manufacture or use carbon black. In dispersion modelling that was conducted in 2001 as part of the application for a provincial Certificate of Approval for a facility in Alberta that manufactures carbon black, using the methodology approved by the province and the results of stack testing of the facility, the estimated maximum 24-hour $PM_{10}$ concentration at the nearest residence was 0.0016 mg/m$^3$, or 0.00037 mg/kg-body weight (bw) per day as an intake for an adult (Environment Canada 2010a; details in Appendix II). The report indicates that most of the particulate emissions from this facility is carbon black. The annual average concentration at the same location was predicted to be 0.0001 mg/m$^3$, or 0.000023 mg/kg-bw per day as an intake. For several reasons, these values are considered upper-bounding estimates of the concentrations to

which the general population in the vicinity of a manufacturing facility may be exposed. First, the short-term value assumes the maximum single-day concentration of carbon black predicted for a 5-year period, whereas the annual average is 16-fold less; hence, typical daily exposures would be much lower. As well, the estimate assumes that the concentration of carbon black originating from this manufacturing facility is the same indoors as it is outdoors, which is considered a conservative assumption. Finally, after the dispersion modelling was conducted, pollution control equipment was installed in 2004 and stack emissions were reduced by 99% on average; however, this reduction has not been factored into the estimated concentrations, because it is not known whether the other two manufacturing facilities in Canada use similar pollution control technology.

Carbon black comprises approximately 22% of the content of vehicle tires (OECD 2006). Release of tire particles from tire wear on the roadway could therefore represent a potential source of exposure to carbon black-containing particles, especially along transportation corridors. No studies have identified that quantify carbon black in atmospheric particulate matter from tire wear in Canada or elsewhere. However, this source is not expected to contribute appreciably to exposure of the general human population to carbon black, for the following reasons. For one thing, carbon black is physically bound within elastomeric complexes in tires and is unlikely to be released as unbound particles through wear or abrasion (US EPA 1976; OECD 2006; ChemRisk, Inc., and DIK, Inc. 2008), or through migration given its negligible solubility and volatility. Also, most of the material worn from tires is released as non-suspendable particles deposited on or beside the road, and as a result tire debris contributes only a small percentage (1 to 10%) to airborne particulate matter near roads (Pierson and Brachazek 1974; ChemRisk, Inc. and DIK, Inc. 2008). In addition, virtually the entire particle mass released from tire wear is too large to enter the respiratory system (>10 μm) (Kreider et al. 2010). Because carbon black in tire wear particles will be almost entirely bound in elastomeric particles, most of which are non-suspendable and non-respirable, inhalation exposure of the general population to unbound carbon black from tire wear is considered to be low. There is also potential exposure to carbon black-containing particles related to secondary uses of tires (recycling, energy production), or from accidental fires at disposal sites where tires are stored, however there are insufficient data to characterize potential bystander exposure from these sources.

Confidence in the exposure characterization for environmental media is considered to be low due to the lack of measured data on the levels of carbon black in environmental media. Estimated exposures are considered to be upper-bounding limits as they are based on conservative assumptions. Confidence is high that inhalation exposure to carbon black from releases to ambient air is minimal.

### *Consumer Products*

Based on the available information on uses of carbon black in Canada, consumer products represent a possible source of direct exposure to users, as carbon black is present in a wide range of consumer products. For the reasons outlined in the introductory part of this exposure assessment, this section focuses on potential exposures to carbon black in

consumer product sources where there appears to be some potential for inhalation exposure, and does not consider the dermal or oral routes of exposure.

Carbon black is used in a large number of paints and coatings. To estimate inhalation exposure to carbon black from use of these products, the scenario of spray painting of wall paints with an airless spray gun was selected as the application scenario because it would generate aerosol particles and the surface area and duration of painting are greater than for other application scenarios. While the use of spray guns by consumers is uncommon, these tools are nonetheless readily available to rent or purchase at various popular do-it-yourself (DIY) stores; thus, there is some use by consumers. Exposure to respirable paint aerosol was estimated using measurements from controlled studies in which the entire walls of poorly ventilated test rooms were painted by professional painters using an airless sprayer (NPCA 2004). The concentration used to estimate exposure was 1% w/w, the maximum concentration in paints other than black paint in the U.S. Household Products Database (HPD 2009). Although some black interior paints contain several-fold higher concentrations of carbon black (HPD 2009), it was considered exceedingly unlikely that homeowners would both use a spray gun to apply wall paint *and* paint large wall surfaces black, so the scenario is for non-black paint. The exposure estimates also assume that the consumer uses recommended personal protective equipment. The specific inputs and assumptions used in deriving the estimate are presented in Appendix II.

Based on the maximum concentration of respirable paint aerosol measured in these controlled studies, and the maximum concentration of carbon black identified in non-black paints, the upper-bounding estimate of exposure to carbon black from spray painting of walls is 0.00257 mg/m$^3$ as a concentration during painting, or 0.0000734 mg/kg-bw per event (see Appendix II). This is a conservative estimate of potential inhalation exposure via spray painting of walls because the measurements and assumptions are considered the worst-case scenario with respect to such factors as ventilation, use patterns, and bioavailability of carbon black from the paint aerosol.

Airborne exposure to carbon black from applying wall paints would be negligible for most consumers, since homeowners typically use rollers to apply interior paints. Painting with a roller does not produce large amounts of spray, and most of the resultant droplets would be too large to be respirable; as a result, roller painting is expected to generate negligible amounts of respirable particles.

Once paints have dried, the carbon black is contained within the cured paint. Decorating and renovating activities such as sanding are known to generate respirable particles (Koponen et al. 2009). Based on the maximum breathing zone concentration of respirable dust generated by professionals sanding in controlled studies (NPCA 2004), and the maximum concentration of carbon black in non-black paint, the upper-bounding estimated concentration of carbon black from sanding of walls is 0.0015 mg/m$^3$ during sanding, or 0.00011 mg/kg-bw per event (see Appendix II). It should be noted that this estimate is conservative. The controlled wall-sanding studies were designed to provide a worst-case estimate of potential exposures in that they involved professional painters

sanding walls in a poorly ventilated room for a several hours. In addition to the inadequate ventilation, the maximum measured concentration of respirable dust was used to calculate these estimates, and it was assumed that no personal protective equipment was used. Finally, most of the carbon black would be expected to remain bound in the paint matrix within these particles.

Carbon black was also reported to be present at a weight fraction of 2.27% in a single costume spray hair dye notified to the CNS (October 2010 email from Consumer Product Safety Bureau, Health Canada to Water, Air and Climate Change Bureau, Health Canada; unreferenced). Exposure estimates for costume spray hair dye containing this concentration were derived using ConsExpo version 4.1 software (RIVM 2007). The scenario modelled is of an individual dyeing his/her hair in the bathroom and then remaining within the bathroom for the subsequent 5 minutes. The specific inputs and assumptions used the modelling are presented in Appendix II. The predicted mean concentration of carbon black in the hair dye spray droplets is 0.000169 mg/m$^3$ during the application and shortly after, or 0.000000134 mg/kg-bw per event as an intake.

Carbon black is used as a pigment in toners for laser printers and photocopiers and in inks for inkjet printers. While in operation, printers and photocopiers emit respirable particles, largely in the ultrafine size fraction (Destaillats et al. 2008), and it is sometimes assumed that these emissions arise from the toner. However, Wensing et al. (2008) tested a specially built printer with no toner and no paper and found that about the same number of particles was produced as from an identical printer with toner and paper, indicating that ultrafine size emissions originate from sources other than toners. In addition, in printers and photocopiers, the toner or ink is typically contained in a sealed system of cartridges (OECD 2006) and the carbon black in the toner powder is strongly bound in a plastic resin (Smart Computing Encyclopedia 2010). Based on the above information, inhalation exposure of the general population to unbound carbon black from operating printers and copiers is not expected to be appreciable. It is also likely that there is some loss of print from the surface of the paper, particularly in the case of some poorer quality machines; however, most of the resultant particles would be expected to be too large to be respirable and to be encapsulated in the toner resin.

Confidence in the numerical estimates of exposure from consumer products is moderate given the absence of empirical measurements. The estimates presented are considered to be overestimates as they are based on conservative assumptions. Therefore, there is confidence that the exposure estimates are conservative and upper-bounding.

## Health Effects Assessment

An overview of health effects information for carbon black is presented in Appendix III.

The International Agency for Research on Cancer (IARC) has classified carbon black as Group 2B, *possibly carcinogenic to humans* (IARC 1996). They concluded that sufficient evidence exists in experimental animals for the carcinogenicity of carbon black and

carbon black extracts, while the evidence in humans is inadequate. An IARC Monographs Working Group re-evaluated carbon black in 2006 and re-affirmed this classification (Baan 2007), though the full report was not available at the time of this screening assessment.

The main route of exposure to carbon black relevant to human health is inhalation; as carbon black is insoluble, dermal absorption is unlikely to occur (OECD 2006). Due to its small particle size, carbon black has the ability to reach the alveolar region of the lung (IARC 1996). The majority of studies on deposition and clearance of carbon black have been performed in the rat due to its sensitivity in regards to carbon black-induced pulmonary toxicity (OECD 2006). Carbon black, both free and phagocytosed, is cleared from the lung primarily by the bronchial tree, as well as by transepithelial passage via alveolar type I cells (IARC 1996). Impairment of alveolar macrophage-mediated clearance can occur through volumetric overloading (i.e., particle overload), which can result in a failure of active movement of the carbon black towards the mucociliary escalator (Oberdorster 2002; OECD 2006). Retention half-time of poorly soluble particles of low toxicity is approximately 70 days in rat lung, with delayed clearance occurring at lung burdens equal to or greater than 0.5–1.0 mg carbon black per lung (Oberdorster 2002; OECD 2006).

The presence of PAHs on the carbon black particle is not considered relevant in terms of carcinogenicity, due to limited bioavailability. More specifically, little or no elution of PAHs has been observed from the surface of carbon black by biological fluids, while carbon black exposure does not appear to result in the formation of PAH-DNA adducts (JECFA 1987; Borm et al. 2005; OECD 2006).

The carcinogenicity of carbon black (Printex 90) was investigated in a 24-month inhalation study in female Wistar Crl (WI) BR rats, at sequential whole-body concentrations of 7.4 mg/m$^3$ for 4 months and 12.2 mg/m$^3$ for 20 months, for 18 hours per day, 5 days per week, followed by clean air conditions for a final 6 months (Heinrich et al. 1995). Histopathological investigations of the nasal and paranasal cavities, larynx, trachea, and lung were performed. There was a marked significant increase in lung tumours; the incidences of adenomas, adenocarcinomas, and squamous-cell carcinomas were 13, 13, and 4%, respectively, compared with one adenocarcinoma (0.5%) and no adenomas or squamous-cell carcinomas in 217 control rats. In addition, 20 benign cystic keratinizing squamous cell (CKSC) tumours were seen in the 100 treated females compared with none in the controls. The lung particle burden, defined as quantity of carbon black particles present in the lung tissue, was 44 mg/lung. No evidence of pulmonary carcinogenicity was seen in mice exposed to carbon black at sequential whole-body exposure concentrations of 7.4 mg/m$^3$ for 4 months and 12.2 mg/m$^3$ for 9.5 months, 18 hours per day, 5 days per week, followed by clean air conditions for a final 9.5 months (Heinrich et al. 1995).

The carcinogenicity of carbon black (Printex 90) was investigated in a 2.5-year inhalation study in female Wistar Crl (WI) BR rats, at two whole-body concentrations of 6 mg/m$^3$ for 43 weeks and clean air conditions for 86 weeks or 6 mg/m$^3$ for 86 weeks and clean air

conditions for 43 weeks, for 17 hours per day, 5 days per week (Heinrich et al. 1994). Lung tumours were observed in 0%, of the rats exposed to clean air alone (controls), 17% (12/72) in those exposed to carbon black for 43 weeks, or and 8% (6/72) of those exposed to carbon black for 86 weeks(with no statistically significant difference between the two treatment groups). In the 43-week group, the tumours included two adenomas, four adenocarcinomas, one squamous-cell carcinoma, and seven benign CKSC tumours; in the 86-week group tumours included one adenoma, one squamous-cell carcinoma, and four benign CKSC tumours. Six rats in the 86-week exposure group also showed marked hyperplasia or marked squamous-cell proliferation.

The carcinogenicity of Elftex-12 carbon black was investigated in a 2-year whole-body inhalation study with both female and male rats, at concentrations of 0, 2.5, or 6.5 mg/m$^3$ for 16 hours per day, 5 days per week (Nikula et al. 1995). Exposed females showed a clear, dose-related, statistically significant increase in benign and malignant lung tumours (mainly adenomas and adenocarcinomas), with combined tumour incidences of 0% (0/105; i.e., out of 105 rats), 7% (8/107), and 27% (28/105) in control, low-dose, and high-dose groups, respectively. No such effect was observed in males [equivalent incidences of 3% (3/109), 2% (2/106), and 4% (4/106), respectively]. Squamous cysts (a type of non-neoplastic lesion) were seen in 0% (0/91), 9% (8/90), and 15% (13/87) of control, low-dose, and high-dose females, and in 0% (0/86), 1% (1/73), and 5% (4/74) of the control, low-dose, and high-dose males, respectively.

Data from studies of carbon black exposure by inhalation were supported by intratracheal instillation studies. In several chronic intratracheal studies, adenomas, adenocarcinomas, squamous cell carcinomas, and benign CKSC tumours were observed in the lungs of rats exposed to carbon black at doses of 1–3 mg/week for 27–36.5 months but not in controls (Pott et al. 1994; Dasenbrock et al. 1996).

A dermal carcinogenicity study of three carbon blacks (furnace, thermal, and channel black) was performed in CFW and C3H male mice, with a dosing regime of 10 or 20% carbon black suspended in cottonseed oil, mineral oil, or 1% carboxymethyl cellulose thrice weekly for 12–18 months (Nau et al. 1958b). No local skin carcinogenicity was observed.

No increase in tumour incidence was observed in rats and mice treated with 2.05 g/kg feed of carbon black (equivalent to 103 and 267 mg/kg-bw per day for rats and mice, respectively [Health Canada 1994]) for 2 years compared with controls (Pence and Buddingh 1985). All tissues were examined for gross pathology. IARC (1996) noted several deficiencies in this study, including small numbers of animals used and incomplete histopathological examinations.

A number of epidemiological studies examining an association between lung cancer and carbon black exposure have been conducted with data from workers in the carbon black production and use industries (i.e., manufacturing involving carbon black) (see Appendix III for study summaries and references). In 1995, the IARC working group on the evaluation of carcinogenic risks to humans (IARC 1996) evaluated carbon black,

highlighting four occupational epidemiological studies that provide the greatest potential for elucidating human cancer risk (Robertson and Ingalls 1980; Hodgson and Jones 1985; Blair et al. 1990; Siemiatycki 1991).

According to IARC (1996), the study that was considered the most informative was conducted by Hodgson and Jones (1985). The study consisted of 1422 male workers in five UK carbon black production factories, and a borderline excess risk of death from lung cancer was observed (standardized mortality ratio [SMR]: 1.5, 95% CI: 1.0–2.2) (Hodgson and Jones 1985). This effect was most evident in one of the five factories, although no evidence indicated a correlation with exposure level. Issues noted by IARC (1996) were possible confounding factors due to unavailability of smoking data and problems with the completeness of the cohort. In a population-based case control study that was conducted in Montreal, a positive association between carbon black exposure and lung cancer (52 cases; odds ratio: 1.6, 95% CI: 1.1–2.3) was observed (Siemiatycki 1991). This was only observed in the subgroup of "high" exposure individuals, and was only significant when a cancer series control group (selected from non-lung cancer cases), but not a population control group, was used for the analysis. An analysis of a carbon black production employee cohort in the United States found no excess incidence or mortality due to lung cancer compared with state vital statistics (relative risk: 0.9, 95% CI: 0.5–1.5) and no increased mortality with increased employment length (Robertson and Ingalls 1980). In a study primarily examining lung cancer risk of formaldehyde exposure in an industry that uses carbon black, a slight but non-statistically significant risk of lung cancer was observed (relative risk: 1.3, 95% CI: 0.8–2.0) (Blair et al. 1990). IARC (1996) also reported methodological limitations and confounding issues with these three studies.

Overall, IARC (1996) concluded that primarily because of inconsistent results, issues of confounding factors, and study design limitations, there was inadequate epidemiological evidence to demonstrate the carcinogenicity of carbon black in humans.

In 2006, an IARC Monographs Working Group re-evaluated the carcinogenic potential of carbon black (Baan 2007) and considered three studies of production workers in the UK, Germany, and the USA to be most informative (Sorahan et al. 2001; Dell et al. 2006; Wellmann et al. 2006). Among the UK cohort of workers initially examined by Hodgson and Jones (1985), and followed until the end of 1996, an excess risk of mortality for lung cancer was observed (SMR: 173, 95% CI: 132–222) (Sorahan et al. 2001). The risk of lung cancer did not increase with increasing cumulative exposure to carbon black (Baan 2007). In a cohort study of German male workers in a carbon black manufacturing facility, a statistically significant increase in the risk of mortality from lung cancer was observed (SMR: 218, 95% CI: 161–287) (Wellmann et al. 2006). IARC (Baan 2007) noted that no dose-response relationship was observed and smoking data were incomplete. In a study of 5011 employees at 18 U.S. carbon black production facilities, no excess risk for mortality from lung cancer was observed (Dell et al. 2006). In fact, the number of deaths observed were slightly less than the numbers expected based on national rates (SMR: 97, 95% CI: 82–115). IARC (Baan 2007) noted that the risk of lung cancer was not assessed based on level of exposure, and smoking status was also not

considered. The re-evaluation concluded that the evidence remained inadequate for the determination of carbon black as a human carcinogen (Baan 2007). Since the latest IARC evaluation, no more recent studies were identified that could provide additional evidence of the human carcinogenicity of carbon black.

A number of *in vivo* and *in vitro* genotoxicity studies have examined carbon black's mechanism of action as a toxicant and carcinogen.

*In vitro*, mixed results have been reported for bacterial mutagenicity assays of carbon black, with positive outcomes generally attributed to impurities, including PAHs and nitropyrenes, in the carbon black extract tested (IARC 1996; OECD 2006). In mammalian cells, an assay for mutations at the *tk* locus in mouse lymphoma cells was negative (Kirwin et al. 1981), but carbon black was considered "weakly mutagenic" in a mouse epithelial cell line, in the *lacZ* and *cII* transgenes (Jacobsen et al. 2007).

There is some *in vitro* evidence in mammalian cells of carbon black-induced DNA damage (strand breaks) and chromosome damage (micronuclei). A sister chromatid exchange assay in Chinese hamster ovary cells was negative (Kirwin et al. 1981). One cell transformation assay was negative (Kirwin et al. 1981) while another was positive (Riebe-Imre et al. 1994).

*In vivo*, DNA damage was observed by the comet assay in a lung cell suspension from male mice following intratracheal instillation of Printex 90 at 0.2 mg per mouse for 3 and 24 hours compared with controls (Totsuka et al. 2009). Carbon black was negative for several types of mutations, aneuploidy, and chromosomal aberrations in *Drosophila melanogaster* (Kirwin et al. 1981).

*In vivo* and *in vitro* assays on DNA adduct formation were mainly negative (Wolff et al. 1990; Gallagher et al. 1994; Borm et al. 2005); however, in one study DNA adducts were detected in alveolar type II cells isolated from rats after 12 weeks of exposure to carbon black (Bond et al. 1990). The exposure concentration in this study was sufficiently high (6.2 $mg/m^3$) that significant localized inflammation was induced in the rat lung.

The *in vivo* data indicate that carbon black-induced mutagenicity only occurs in the presence of inflammation. The frequency of *Hprt* mutations was significantly increased, compared with controls, in alveolar type II cells isolated from rats exposed by inhalation to furnace black for 13 weeks at 7.1 and 52.8 $mg/m^3$, but not at 1.1 $mg/m^3$ (Driscoll et al. 1996). The exposure concentrations at which mutation frequencies were increased also caused significant inflammation, hyperplasia, and fibrosis in rat lung. The addition of an antioxidant enzyme (catalase) inhibited the mutation frequency increase, indicating that cellular oxidant concentration also plays a role in mutagenesis.

There was no difference in the number of K-*ras* or *p53* mutations observed in pulmonary carcinomas from rats exposed by inhalation to Elftex-12 compared with controls (Swafford et al. 1995), although very few tumours were examined and the frequency of these mutations in rat lung tumours is expected to be low (OECD 2006).

Numerous *in vitro* studies have demonstrated that exposure to carbon black induces an increase in reactive oxygen species (ROS), as well as other indicators of oxidative stress and inflammation (see Appendix III for details and study summaries). Jacobsen et al. (2007) detected oxidative DNA damage (oxidized purines) in mouse epithelial cells after exposure to carbon black. *In vivo*, increased 8-hydroxydeoxyguanosine (8-OHdG; marker of oxidative DNA damage) residues were detected in rat lung DNA after 13 weeks of inhalation exposure to Printex 90 at 1 to 50 mg/m$^3$ (Gallagher et al. 2003).

Although there is evidence indicating that carbon black can cause DNA and chromosome damage both *in vitro* and *in vivo*, the weight of evidence of the genotoxicity data indicate that carbon black is not mutagenic as a direct DNA-reactive compound, but rather mutagenicity is secondary to oxidative stress and inflammatory processes. Other national and international agencies have also concluded that carbon black is not directly mutagenic (IARC 1996; OECD 2006). The *in vivo* and *in vitro* data support an important role for inflammation, oxidative stress, and formation of ROS in carbon black toxicity.

Carbon black-induced rat lung tumours are proposed to be associated with particle overload (IARC 1996; ILSI 2000; Oberdorster 2002; Borm et al. 2004). For poorly soluble low-toxicity particles, such as carbon black, particle overload can be defined as a retained lung burden of particles greater than the steady-state burden predicted from the deposition rates and clearance kinetics of the particle (ILSI 2000). Carbon black is considered insoluble and durable, as it requires other clearance mechanisms, such as phagocytosis, for removal. Therefore, rapid local accumulation can occur upon sustained exposure, resulting in an inflammatory state in the lung (ILSI 2000). Tumourigenicity results from inflammation in a particle overload-induced state by: (1) ROS-induced DNA damage that is fixed and propagated by increased epithelial cell proliferation and (2) ROS-induced elevated cytokine and growth factor production acting as promoters of epithelial and neoplastic growth (IARC 1996; ILSI 2000; Oberdorster 2002; OECD 2006).

Thresholds for rat lung particle overload and risk for subsequent neoplastic events proposed in the literature for particles such as carbon black are 1 mg/g of lung tissue or 1–3 mg per rat lung. These values appear to be 10-fold higher than what is required to induce neutrophilic inflammation (ILSI 2000; Tran et al. 2000; Oberdorster 2002; Borm et al. 2004).

Consistent with the mechanistic and carcinogenicity data, the non-neoplastic effects of carbon black involve local inflammation-related pulmonary and cardiovascular toxicity following inhalation in both experimental animals and humans.

Available studies on the respiratory effects of carbon black in humans are based on occupational exposure from both carbon black production and product manufacturing facilities (see Appendix III for references).

In a study consisting of two phases, 2342 and 1994 male workers in 19 and 16 carbon black European factories in the earlier and later phase, respectively, were assessed for various respiratory symptoms and lung function measurements (Gardiner et al. 2001). Positive statistically significant associations were observed between current carbon black exposure and cough, sputum production, cough with sputum production, chronic bronchitis, forced expiratory volume, forced mid-expiratory flow, and forced expiratory volume: forced vital capacity ratio. Similarly, positive significant and borderline significant associations were also observed for cumulative exposure. Mean duration of employment (for cumulative exposure) was almost 15 years, and all data were adjusted for the effects of factory, age, height, and smoking. This study demonstrated the lowest carbon black concentrations correlating with an effect in humans, with the mean current inhalable dust concentrations being 0.77 and 0.57 $mg/m^3$ for the two phases of the study, respectively. Consistent with the decrease in carbon black exposure, respiratory morbidity in the second phase of the study was reduced in relation to current exposure as compared with the first phase, although the association with cumulative exposure remained consistent in both phases.

Additional epidemiological data have also demonstrated reduced lung function, chest radiograph abnormalities, and respiratory symptoms in workers exposed to higher mean measured inhalable and respirable carbon black dust concentrations (see Appendix III for references). It has been hypothesized that these findings are attributable to a slight non-specific irritant effect of heavy occupational dust exposure, or possibly a fibrous tissue reaction in the lung parenchyma surrounding carbon deposits (IARC 1996).

The lowest inhalational LOEC in laboratory animals has been identified as 400 $\mu g/m^3$ of carbon black from a 4-day study in male mice (3 hour exposure per day), based on increased left ventricular diameter, right ventricular and pulmonary vascular pressure, ROS, and matrix metalloproteinase (MMP) 2 and 9 in heart tissue, along with decreased fractional shortening and ejection fraction (Tankersley et al. 2008).

Available data support the formation of cardiovascular inflammation and ROS following high-dose carbon black exposure by inhalation, resulting in altered cell signalling. It has been hypothesized that cardiovascular toxicity following carbon black exposure can result from either an inflammatory response due to larger particles and/or aggregates of fine particles in the lung, or the translocation of ultrafine particles into the pulmonary circulation upon inhalation (Tankersley et al. 2007, 2008; Niwa et al. 2008).

In an acute inhalation study, neutrophil influx, increased epithelial permeability, and oxidative stress (reduction of total GSH) was observed in the lungs of male rats exposed to 1 $mg/m^3$ of carbon black for 7 hours (Li et al. 1997; OECD 2006). In a short-term, repeated-dose inhalation study, increased blood pressure and levels of inflammatory markers (circulating monocyte chemotactic protein-1, IL-6, and C-reactive protein) were observed in rats exposed to carbon black at 15.6 $mg/m^3$ for 6 hours/day, 5 days/week, for 4 weeks (i.e 120 hours total exposure, Niwa et al. 2008).

In a subchronic inhalation study, inflammation and oxidative stress (TNF-α, inflammatory protein-2, ROS, and reactive nitrogen species) were observed in the bronchioalveolar lavage fluid from rats exposed to carbon black at 7 mg/m$^3$ and 50 mg/m$^3$, 6 hours per day, 5 days per week, for 13 weeks (390 hours total exposure, Carter et al. 2006). In an additional study with an identical dosing protocol, pulmonary inflammation (polymorphonuclear leukocyte infiltration) was observed in rats and mice exposed to carbon black at 7 mg/m$^3$ and 50 mg/m$^3$ (Elder et al. 2005). In a subchronic to chronic inhalation study, morphological changes in the respiratory tract and decreased resistance to infection were observed in mice exposed to carbon black at 1.5 mg/m$^3$, 3 hours per day, 5 days per week, for 4 to 20 weeks (60 to 300 hours total exposure, Fenters et al. 1979).

In a chronic inhalation study in rhesus monkeys, alveolar wall destruction (which was classified as moderate to severe emphysema) and hypertrophy of the right ventricular septum were observed after exposure to carbon black at 53 mg/m$^3$ for 6 hours per day, 6 days per week, for 3 years (Nau et al. 1976).

Carbon black did not influence the number of implantation sites or pups per litter, survival rate, pup weight, or pup length in a developmental study in female Fischer 344 rats exposed by inhalation to carbon black at 100 µg/m$^3$ for 4 hours per day on days 11–20 of pregnancy (Archibong et al. 2002). However, only one relatively low concentration was used and no information on the carbon black was provided. In a 10-week intratracheal instillation study, partial vacuolation of the seminiferous tubules, elevation of serum testosterone concentration, and decreased daily sperm production were observed in male ICR mice exposed to 0.1 mg carbon black (Printex 90, Printex 25, and Flammruss 101) once a week (Yoshida et al. 2008). In addition, *in vitro* exposure of mouse Leydig TM3 cells to carbon black (Printex 90) decreased cell viability 24 hours after exposure to 1000 µg/mL and enhanced steroidogenic acute regulatory mRNA expression 48 hours post-exposure at 10 and 30 µg/mL (Komatsu et al. 2008). No oral or dermal reproductive or developmental studies were identified. Overall, there is little evidence that carbon black is a developmental toxicant. Limited evidence in mice indicates carbon black may affect the male reproductive system, although the effect on male fertility was not examined in the previously mentioned studies (Yoshida et al. 2008). The OECD concluded that, based on the toxicokinetic data and information from toxicity studies with other similar-sized particles, it is unlikely that carbon black will reach the reproductive organs, embryo, or fetus under *in vivo* conditions and therefore no adverse effects on reproduction and development are expected (OECD 2006).

No liver toxicity was observed in C57BL/6 mice following exposure to 20 mg/m$^3$ carbon black (Printex 90) for 90 minutes per day for 4 days (Saber et al. 2009).

No gross pathological or microscopic changes, as well as no effects on survival rate, were observed in mice and rats after oral exposure to 10% carbon black or 2.05 g/kg of carbon black in feed (equivalent to 103–500 and 267–1300 mg/kg-bw per day for rats and mice, respectively [Health Canada 1994]) (Nau et al. 1976; Pence and Buddingh 1985). Following 123 applications of 20% emulsions of carbon black to the skin of mice over a

period of 41 weeks, no effect on body weight or detectable pathological changes were observed (Nau et al. 1976).

Carbon black is considered to be possibly irritating to the skin, eye, and/or respiratory system (OECD 2006). Grant (1986) concluded that carbon black may be slightly irritating mechanically and may cause discoloration of lids and conjunctivae, but is chemically inert (as cited in HSDB 2009).

Confidence in the toxicity data set is considered to be moderate. Data in experimental animals are available for acute toxicity, repeated-dose toxicity, chronic toxicity, carcinogenicity, and genetic toxicity. Most studies are in rodents, and one limited study has been conducted in non-human primates. Limited data are available on reproductive and developmental toxicity. The available toxicity studies were conducted at high carbon black exposure levels resulting in a particle overload state in the lung, while the effects of exposure to lower levels of carbon black have not been examined fully. The relevance of rat particle-overload pulmonary toxicity to humans has yet to be elucidated. A limited number of toxicity studies have demonstrated potential cardiovascular effects in rodents resulting from carbon black exposure, which must be investigated further. Substantial epidemiological data exist examining the association between worker exposure to carbon black and respiratory toxicity and carcinogenicity; however, the results are somewhat inconsistent, and there are issues of potential confounding effects from other chemicals and from smoking, as well as methodological limitations.

## Characterization of Risk to Human Health

Carcinogenicity is considered in the health effects assessment for carbon black, as the substance was classified by IARC as possibly carcinogenic to humans (group 2B) (IARC 1996; Baan 2007). This classification was based on increases in benign and malignant tumours reported in female rats following long-term inhalation and intratracheal exposure in several studies, whereas the evidence in humans was deemed inadequate (IARC 1996; Baan 2007). More recent studies do not provide additional evidence of the carcinogenicity of carbon black in humans that would affect IARC's conclusions.

The available genotoxicity data indicate that carbon black is not likely to be mutagenic or to be tumourigenic through direct interaction with genetic material. Although carbon black can cause DNA damage both *in vitro* and *in vivo*, these effects are considered to be secondary to increases in oxidative stress, inflammation, and generation of reactive oxygen species. In the rat lung, pulmonary inflammation resulting from carbon black inhalation or intratracheal instillation causes oxidative DNA damage and altered cell signalling leading to tumourigenesis. More specifically, it appears that the induction of lung tumours in rats after carbon black exposure is caused by an excessive lung burden (i.e., particle overload) owing to overwhelming and impairment of clearance mechanisms, resulting in an oxidative state (Oberdorster 2002). Limited information regarding particle overload in humans and non-human primates also suggests more

interstitialization of the deposited particles and less inflammation and epithelial cell proliferation responses than reported in the rat (ILSI 2000).

As carbon black is not directly genotoxic, a margin of exposure approach is used to assess risk to human health.

For inhalation exposure, the lowest reported LOEC is 0.4 mg/m$^3$, based on cardiovascular toxicity observed after short-term repeated-dose exposure (vascular remodelling and altered function, along with oxidative stress, in male mice) in a 4-day study (Tankersley et al. 2008). The lowest reported LOEC resulting in pulmonary toxicity is 1 mg/m$^3$, based on non-neoplastic effects observed after acute inhalational exposure (inflammation and oxidative stress in male rats) in a 7-hour study (Li et al. 1997; OECD 2006).

The lowest reported acute LOEC for pulmonary toxicity (1 mg/m$^3$ in rats, Li et al. 1997; OECD 2006) has been selected as the acute critical effect level for the margin-of-exposure analysis, rather than the lower effect level for cardiovascular toxicity in mice reported by Tankersley et al. (2008), for the reasons outlined in the following paragraphs.

There is a stronger weight of evidence for pulmonary toxicity and for an effect level of 1 mg/m$^3$ in the toxicological and epidemiological literature. Respiratory effects, including inflammation, have been observed in several studies in rats and mice after single or repeated exposures to concentrations of carbon black of 1.5 mg/m$^3$ and greater (Fenters et al. 1979; Dungworth et al. 1994; Driscoll et al. 1996; Elder et al. 2005; Carter et al. 2006). With respect to humans, Gardiner et al. (2001) observed statistically significant associations between respiratory symptoms and lung function changes in workers with mean exposure to inhalable dust concentrations of carbon black of as little as 0.57 mg/m$^3$, a level similar to those that induced respiratory effects in rats.

In contrast, there is some question with respect to generalizing the results of the cardiovascular effects observed in mice by Tankersley et al. (2008). For example, a 4-hour exposure of Wistar rats to 5 mg/m$^3$ carbon black did not result in effects on heart rate, blood pressure, or plasma endothelin levels (Vincent et al. 2001), although increased blood pressure was observed in rats exposed to a higher concentration of 15.6 mg/m$^3$ for 4 weeks in another study (Niwa et al. 2008). The cardiovascular effects of carbon black in the mouse also appear to be strain-specific; 0.536 mg/m$^3$ exposure for 3 hours resulted in an increase in $O_2$ pulse (oxygen uptake per heartbeat at rest) in B6 mice, but not OuJ or HeJ mice (Hamade et al. 2008). In epidemiology studies of carbon black workers, exposure to carbon black was not related to cardiovascular morbidity or mortality (Robertson and Ingalls 1980, 1989; Robertson and Inman 1996).

Finally, the acute exposure regime in the study with rats by Li et al. (1997) is considered more relevant to the single exposures that are characteristic of the consumer products modelled in the exposure assessment than that for the mice studied by Tankersley et al. (2008), in which exposures were intermittent and spread roughly over 1 week.

The lowest reported LOEC for chronic toxicity is 0.57 mg/m$^3$, based on increased respiratory symptoms and decreased lung function measurements in male workers exposed to carbon black for an average of almost 15 years (Gardiner et al. 2001).

For environmental exposures, the acute upper-bounding estimate of exposure is the highest short-term airborne concentration predicted at the residence nearest to a carbon black manufacturing facility by dispersion modelling of emissions from the facility (i.e., 24-hour $PM_{10}$ of 0.0016 mg/m$^3$). Comparison of the critical effect level for acute inhalation toxicity (i.e., 1 mg/m$^3$ for non-systemic lung inflammation) with the upper-bounding estimate of environmental exposure results in a margin of exposure of 625. For chronic exposure, the estimate of exposure is the annual average airborne concentrations predicted at the nearest residence, i.e., 0.0001 mg/m$^3$. Comparison of the critical effect level for chronic inhalation toxicity (i.e., 0.57 mg/m$^3$) with the chronic estimate of exposure results in a margin of exposure of 5700. As discussed in the Exposure Assessment section, the exposure estimates are upper-bounding in several respects.

Exposure from consumer products was estimated for those products where there was potential for inhalation exposure to unbound carbon black (in most consumer products, carbon black is bound in a matrix and unavailable for exposure). The upper-bounding estimate of exposure from consumer products is 0.00257 mg/m$^3$ (adjusted for removal of respirable paint aerosol by the respirator) of carbon black during the spray application of wall paints. Comparison of the critical effect level for acute inhalation toxicity (i.e., 1 mg/m$^3$) with the upper-bounding estimate for spray painting of wall paints results in a margin of exposure of 389. (The margins of exposure for carbon black from sanding paint [667] or from costume spray hair dye [5917] are greater as a consequence of the much lower exposures from these activities.) The margin of exposure for spray painting of wall paints depends on the use of suitable respiratory protection, which is widely recommended and readily available (Appendix II). As discussed under Exposure Assessment, the exposure estimate for spraying wall paint is also considered conservative and upper-bounding in a number of respects. It should also be noted that airborne exposure to carbon black from applying wall paints would be negligible for most consumers, since homeowners typically use rollers to apply interior paints, in which case little, if any, respirable paint particles would be generated.

Increases in tumour incidence were only observed following long-term inhalation exposure of male rats to carbon black at concentrations that were greater (i.e., 2.5 mg/m$^3$ or more) than the critical effect levels used in this risk characterization, and that resulted in particle overload (Heinrich et al. 1994, 1995; Nikula et al. 1995). The weight of evidence indicates that chronic inflammation induced by particle overload is a prerequisite for carbon black-induced tumourigenicity. The concentrations that induce tumours in the rat and those that result in particle overload are both approximately three or more orders of magnitude greater than the estimated exposures in the general environment or from consumer products.

In the Tankersley et al. (2008) study, cardiovascular effects parameters were affected in very old mice (28 months old), but not in younger mice (18 months old). This is

consistent with the extensive literature indicating that elderly humans have heightened susceptibility to the cardiovascular effects associated with exposure to a variety of types of inhaled particles (US EPA 2009) and indicates that the elderly may be more vulnerable to inhalation of carbon black than are other segments of the general population.

In summary, the upper-bounding estimated exposure concentrations that the general population is exposed to are between 389 and 625 times or more lower than the acute critical effect level, and 5700 times less than the critical effect level for chronic effects. In addition, the estimated exposure concentrations are several orders of magnitude below those associated with particle overload and tumourigenicity in animals. The available data, while limited, also indicate that the effect levels associated with pulmonary effects appear to be similar in animals and humans, and that the elderly of several species may be the segment of the population most sensitive to the effects of inhaled carbon black (as for other particle types). Based on these considerations, the margins of exposure for carbon black near point sources and from consumer products are expected to be adequate to address uncertainties in the health effects and exposure databases.

**Uncertainties in Evaluation of Risk to Human Health**

A number of differences exist between the rat and human lung, which may lead to different forms of pulmonary toxicity and lung cancer. They include differences in cell types, tissue histology, airway geometry and ventilation, clearance rates, and damage or repair mechanisms (Levy 1996; ILSI 2000). These differences support the hypothesis that the severity of carbon black toxicity, through oxidative stress mechanisms, may vary between species, especially in regard to dose–response (Levy 1996). The particle overload phenomenon in the rat lung, resulting in lung lesions and tumours, may be a rat-specific effect with questionable relevance to human lung cancer (Levy 1996). This is consistent with the absence of lung tumours after long-term inhalation exposure of non-human primates to 53 $mg/m^3$ carbon black (Nau et al.1976), and of mice to sequential concentrations of 7.4 $mg/m^3$ for 4 months and 12.2 $mg/m^3$ for 20 months (Heinrich et al. 1995).

Although a carcinogenic response in the rat model following particle overload has been observed, the toxicity profile of carbon black at concentrations that do not result in lung overload is unknown. Further research involving doses of carbon black below those that result in particle overload in rodent and non-human primate models would lead to further understanding of the biological effects of carbon black and risk to human health.

With respect to exposure to carbon black in the general environment, no data were identified regarding measured concentrations of carbon black in environmental media, particularly in ambient air. In the absence of measurements of carbon black in outdoor and indoor air, the assessment was based on modelled estimates of concentrations near point sources, which are expected to present a greater potential for exposure than to the general population. In addition, conservative assumptions, which would overestimate exposure, were made in using these predicted concentrations.

In most consumer products carbon black is bound in a matrix and unavailable for exposure, for example, as a pigment in plastics and rubbers. However, there is uncertainty about the biological availability of carbon black in products where it is not firmly encased in a solid matrix, such as uncured paints. In the absence of data, it has been assumed that the carbon black in such non-solid matrices is entirely bioavailable. Confidence is moderate that the derived environmental and consumer product exposure estimates are adequately protective of the general population of Canada, as conservative estimates and assumptions and upper-bounding scenarios were used when data were unavailable.

## Conclusion

Based on the information presented in this screening assessment, it is concluded that carbon black does not meet the criteria in paragraphs 64*(a)* and *(b)*, as it is not entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long-term harmful effect on the environment or its biological diversity or that constitute or may constitute a danger to the environment on which life depends. Carbon black meets the criteria for persistence as set out in the *Persistence and Bioaccumulation Regulations* (Canada 2000) but does not meet bioaccumulation potential criteria.

On the basis of the adequacy of the margins between estimated exposures to carbon black and critical effect levels, it is concluded that carbon black does not meet the criteria in paragraph 64*(c)* of CEPA 1999, as it is not entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health.

It is therefore concluded that carbon black does not meet any of the criteria under section 64 of CEPA 1999.

This substance will be considered for inclusion in the Domestic Substances List inventory update initiative. In addition and where relevant, research and monitoring will support verification of assumptions used during the screening assessment.

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# Appendix I. Other names for carbon black

*10B; 150T; 1K01967; 20B; 2200B; 2300CB; 2400B; 2500B; 25B; 25B (pigment); 25BS; 25CB; 2700B; 3030B; 3050B; 30B; 30B (carbon black); 3230B; 3250B; 3350B; 3400B; 3500B; 35G; 3750B; 376M; 40B; 40B2; 44CB; 45B; 45B (carbon black); 50HG; 5110B; 550B; 550B (carbon); 600JD; 63B1 Black; 650B; 7270M; 7400SB; 750B; 7550F; 7550SB/F; 7861D; 850B; 9100SH3; 9100SH4; 95CB; 960B; A 1-1000; AB 6 (carbon black); ACARBP; ACET; ACET (carbon black); Acetylene Black AD 100; Acetylene Black HS 100; Acetylene CB; AcryJet Black 357; Acticarbon AC 35; AD 200; AD 200 (carbon); AG-FW 2V; Ajack 2056; Ajack Black 5021; AM Black; AM Black 9700; AMBK 2; AMBK 7; Americhem 11793F1 Black; Animal bone charcoal; Aqua Black HA 3; Aqua-Black 001; Aqua-Black 162; Aquablak; Aquablak 15; Aquablak 235A; Aquablak 245; Aquablak 320; Aquadisperse Black CB-EP; Aquafine AF Black E 2B; Aquafine Black E 2B; Aqualour Black; Aquis II KW 3729; AR-D 2; Arosperse 15; Arosperse 15-213; Arosperse 15-239; Asahi 15; Asahi 15HS; Asahi 35; Asahi 35G; Asahi 50; Asahi 500G; Asahi 50H; Asahi 50HG; Asahi 55; Asahi 55G; Asahi 60; Asahi 60G; Asahi 60H; Asahi 60HG; Asahi 60HN; Asahi 60UG; Asahi 65; Asahi 66; Asahi 70; Asahi 70HAF; Asahi 70L; Asahi 70NP; Asahi 75N339; Asahi 80; Asahi Black 3078; Asahi Black HS 500; Asahi Thermal Black FT; Asahi Thermal FT; Asahi Thermal MT; Asahithermal; ASM; ASM (carbon); ATG 60; ATG 70; ATR 077; Aurasperse W 7017; Austin Black;AX 015 ;AX 3023; B 326M; Baydur Blackpaste DN; Bayscript VPSP 20016; BF 31150; BK 6; BK 6 (carbon black); BK 8200; BKhPO; Black 40; Black 6B7; Black BLN; Black DCF 50; Black FW; Black No. 2; Black Pearls; Black Pearls 1000; Black Pearls 1100; Black Pearls 120; Black Pearls 130; Black Pearls 1300; Black Pearls 1300A73; Black Pearls 160; Black Pearls 2000; Black Pearls 280; Black Pearls 3200; Black Pearls 3500; Black Pearls 3550; Black Pearls 3700; Black Pearls 420; Black Pearls 430; Black Pearls 4350; Black Pearls 4560; Black Pearls 460; Black Pearls 4750; Black Pearls 480; Black Pearls 490; Black Pearls 6100; Black Pearls 700; Black Pearls 800; Black Pearls 8500; Black Pearls 880; Black Pearls 900; Black Pearls L; Black Y 200; Blackhole Bonjet Black 850L; Bonjet Black CW 1; Bonjet Black CW 2; BP 1000; BP 130; BP 1300; BP 2000; BP 280; BP 3200; BP 3500; BP 3700; BP 4302; BP 450; BP 480; BP 700; BP 800; BP 8001; BP 880; BPL; C 100; C 100 (carbon); C 1000; C 1000 (carbon); C 1216P85; C 198; C 311; C 44; C 44 (carbon); C.I. 77266; C.I. 77268:1; C.I. Food Black 3; C.I. Pigment Black 6; C/B-G-SVHCa 21; CA 4395; Ca 54; Ca 54 (adsorbent); Cabot 330; Cabot 607; Cabot Black Pearls 4350; Cabot N 326; Calblack N 220; Calgon RBDA; Carbex 330; Carbocolor; Carbodis 100; Carbodis 80; Carbolac 1; Carbolac 2; Carbon black; Carbon Black 100; Carbon Black 2000; Carbon Black 2300; Carbon Black 25; Carbon Black 25B; Carbon black 2600; Carbon Black 2650Carbon Black 30; Carbon Black 32; Carbon Black 40; Carbon Black 4350; Carbon black 44; Carbon Black 45; Carbon Black 45L; Carbon Black 50; Carbon Black 52; Carbon Black 850; Carbon Black 960; Carbon Black 980; Carbon ECD; Carbon ECP 600JD; Carbon ISAF; Carbon MA 7; CB 24; CB 24 (carbon black); CB 30; CB 3750B; CB 5264; CB 850; CB 950; CB 960; CB 970; CC 1150U; CC 40-220; CC-N 880; CC-N 990UP; CD 1001; CD 1003; CD 1006; CD 2005; CD 2005HT1000; CD 2005HT1500; CD2005HT2000; CD 2013; CD 2019; CD 2038CD 2041; CD 6206; CD 7055 Ultra; CDX 975; CECA 4S; Celgreen HBMD-D; CF 31; CF 9; CF Black PC; CFP-FF 949K;*

*Channel black; Channel Black 100; Char, from refuse burner; Chesacarb; Chesacarb E; Chesacarb EC; Chesacarb ECAChesacarb K 2; Chezacarb A; Chezasorb; CK 3; CK 4; CK 4 (carbon black); CMB 561; CMF 88; COAL SOOT; Codispersion 30R20; Colanyl Black N; Collophite; Color Black FW 1; Color Black FW 18; Color Black FW 18G; Color Black FW 2; Color Black FW 200; Color Black FW 220; Color Black FW 285; Color Black S 160; Color Black S 170; Colormatch DR 20845; Colormatch DR 20942; Colormatch VS; 20519; Columbian Raven 350; Conductex 40-200; Conductex 40-220Conductex 900; Conductex 950; Conductex 975; Conductex 975 Ultra; Conductex 975U; Conductex CC 40-220; Conductex N 472; Conductex SC; Conductex SC Ultra; Conductex SC-U; Condutex XC 72; Continex N 330; Continex N 356; Corasol C 30; Corax 234; Corax 3HS; Corax A; Corax L; Corax L 29; Corax L 6; Corax N 100; Corax N 110; Corax N 115; Corax N 121Corax N 220; Corax N 234; Corax N 234G; Corax N 326; Corax N 330; Corax N 339; Corax N 358; Corax N 375; Corax N 539Corax N 550; Corax N 600; Corax N 762; Corax N 772; Corax N 990; Corax P; CRX 1416B; CS-BK 100Y; CSF; CSF (carbon black); CSX 147; CSX 150A; CSX 150A2; CSX 156; CSX 174; CSX 200ACSX 292; CSX 320; CSX 333; CSX 362; CSX 440; CSX 440L; CSX 99; CW 2; CW 2 (pigment); CX-GLT 20; D and C Black No. 2; D&C Black No. 2; DAB 50; DB 40R; DC Black 7100; DCF 50; Degussa AG-FW 2V; Degussa Black FW; Degussa FW 200; Denka Acetylene Black; Denka Black AB 12; Denka Black AB 6; Denka Black AB 7; Denka Black DH; Denka Black FX 35; Denka Black HS 100; Denka Black HS 200; Denka Black NC 75; Denka Black OAB 100; Denka Black ST 100; Denka HS 100; Denkablack; Dequssa FW 2V; Dermmapol Black G; Derussol P 130; DeSK 008; DeSK 009; DG 100; Diablack; Diablack 2350Diablack 30; Diablack 3030; Diablack 3030B; Diablack 3050B; Diablack 3150B; Diablack 3230B; Diablack 3250; Diablack 33Diablack 3500B; Diablack 3950; Diablack 45L; Diablack 52Diablack 960B; Diablack A; Diablack E; Diablack G; Diablack H; Diablack HA; Diablack HS-SAF; Diablack I; Diablack L; Diablack LH; Diablack LI; Diablack LM-SFR; Diablack LR; Diablack MA 100; Diablack MA 14; Diablack MA 220; Diablack MA 230; Diablack MA 40; Diablack MA 650; Diablack MA 70; Diablack MA 77; Diablack MA 8; Diablack MA 800; Diablack MA 8B; Diablack N 220; Diablack N 234; Diablack N 234M; Diablack N 339; Diablack N 550M; Diablack SA; Diablack SF Diablack SH; Diablack UX 10; Disperse Black SD 9020; Disperse HG 935; DMG 105a; DR 0217; Durex O; Dymic MBR 717; DZ 13E 153; E 1670; E 1720; E 1830; E 1830 (carbon black); E 1990E 330R; EB 095; EB 109; EB 111; EB 118; EB 122; EB 122; (carbon black); EB 123; EB 136; EB 137; EB 167; EB 169; EB 171; EB 172; EB 174; EB 204; EB 204 (carbon black); EC 300EC 600; EC 600J; Ecorax; Ecorax 1670; Ecorax 1720; ECPECP (carbon black); ECP 04; ECP 110; ECP 600JD; ECX-A 304NW; ECX-Z 501; EDO; EDO (carbon black); EG Black G 30; Eldic EC 8013; ELF 415; ELF 78; ELF-O; Elftex; Elftex 115; Elftex 12; Elftex 120; Elftex 150; Elftex 180; Elftex 225; Elftex 254; Elftex 280; Elftex 285; Elftex 415; Elftex 435; Elftex 460; Elftex 470; Elftex 495; Elftex 5; Elftex 8; Elftex Oil Pellets; Elftex P 100; Elftex TB; Elftex TP; EM Black K 16; EM Color Black K 16; Emacol NS Black 4901; Ensaco 150; Ensaco 150G; Ensaco 200; Ensaco 210G; Ensaco 23MM; Ensaco 250; Ensaco 250G; Ensaco 260G; Ensaco 350; Ensaco 350G; EP 510 Black EP 564 Black; EPC; EPC (carbon black); Euderm Black D-B EX 3-3; EXP; EXP (carbon black); EXP 1; EXP 2; F 122 F 122 (carbon black); F 200; F 200 (carbon black); F 30940M F 35X; Farbruss 200; Farbruss FW 1; Farbruss FW 18; Farbruss S 160; FCB 010; FCB*

*025; FD 0721; FEF; FEF 550; FEF-LS FK 35; FK 45; FK 45 (carbon black); Flame Black; Flammruss 101; Flexiverse Black 7; Flexobrite Black 43/77VB; FT 239; Fuji AS Black; Fuji AS Black 810; Fuji SP Black 8306; Fuji SP Black 8922; Fuji VL Black; Furex N 772; Furnace black; Furnace Black 101; Furnace black 2300; Furnace Black 3050; Furnace Black 3600B; Furnal 500; Furnex; Furnex N 765; FW 1; FW 100; FW 101; FW 18; FW 1P; FW 2; FW 200; FW 200B; FW 200P; FW 285; FW 2V; FX-GBI 015; G 2; G 2 (carbon black); GA 50; GA Black 1; GA Black 12031; Gas black; GCH 200; GF 20; GF 20 (carbon black); GP Black 4613; GPF 660; Grand Black YT 100; Graphon C; Graphtol Black BLN; H 950; H 960; H 960 (carbon black); HA 3; HA 3-20S; HAF; HAF-HS; HAF-N 330; HCB-A; HCF; HCF 2300; HCF 2600; HCFox 1; HG 1; HG 1 (carbon black); HG 1B; HG 1P; HG 3; HG 3 (carbon black); HG 4; HG 4 (carbon black); HG 4B; HG-CB; Hi-Black 150B; Hi-Black 160B; Hi-Black 40B2; Hi-Black 41; Hi-Black 420B; Hi-Black HI; Highblack 40B1; Himicron K Black 0542; HM 00-02; HMK 7360; Holcobatch C Black 93909; Hostafine Black T; Hostafine Black TS; Hostajet BLK-VP 2676; HP 180; HPL 41 Powder; HS 100; HS 25; HS 45; HS 500; HS 5009; HS-N 100; HT 1000C; HT 1100; HT 1500C; HTC 100; HTC 20; HTC 20S; HTC-G; HTC-S; HTC-SH; HTC-SL; Huber N 990; HV 3396; ICB 0510; IDIS 25K; IDIS 31K; IDIS 40; IJX 56; IMC 10; IP 200; IP 600; IRA 2; IRB 7; Irgafin Black CN; IRX 1046; ISAF; ISAF-LS; ISAF-N 220; JAS 220; JAS 330; JAS 550; JE 2105; JE 32; JE 4200; JE 6300; JE 6500; K 354; K 354M; K 534; K 615; KB 600JD; Ketjen 600JD; Ketjen Black A 8; Ketjen Black EC-P 600; Ketjen Black W 310A; Ketjen EC-DJ 600; Ketjenblack; Ketjenblack 300; Ketjenblack 300J; Ketjenblack 600JD; Ketjenblack BC; Ketjenblack EC 300; Ketjenblack EC 300N; Ketjenblack EC 310; Ketjenblack EC 310NW; Ketjenblack EC 3750; Ketjenblack EC 600; Ketjenblack EC 600DJ; Ketjenblack EC 600J; Ketjenblack EC 8002; Ketjenblack EC-DJ 500; Ketjenblack EC-DJ 600; Ketjenblack EC-P; Ketjenblack EC-P 600JD; Ketjenblack EC-X; Ketjenblack EP-C 600JD; Ketjenblack EP-DJ 600; Ketjenblack ES; Ketjenblack FC; Ketjenblack KC; Ketjenblack W 310A; KGO 250; KM 9051 KOG 5CB; KOG-CLS; Kosmas 40; KTU 3; L 1/8 Black MA 100 L 6; Lamp Black; Lamp Black 101; Lamp Black 888-9907B; Lamp Black LB 101 Pigment I; LB 101; Levanyl B-LF; Levanyl Black A-SF; Levanyl Black B-LF; Levanyl Black BZ; Levanyl Black N-LF; Levanyl N-LF; Liojet WD Black 002C; Lion Paste W 310A; Lion Paste W 311N; Lion Paste W 376R; LPT; LPT (carbon); LS-N 700; Luconyl Black 0060; Luconyl Black 0066; M 1000 (carbon black); M 2; M 2600; M 5; M 8; M 800; MA 10; MA 10 (carbon); MA 100; MA 100R; MA 100S; MA 200RB; MA 220; MA 230; MA 285; MA 6; MA 6 (carbon black); MA 60; MA 60 (carbon); MA 600; MA 600 (carbon black); MA 600B; MA 7; MA 7 (carbon black); MA 77; MA 78; MA 7A; MA B; MA 8; MA 800; MAF-LS; Magecol 888; MB 45; MB 45 (carbon black); MC; MC (carbon black); MC Black 082E; MCF 88; MCF 88B; MCF 950; MCF 970; MCF-HS 78; MCF-LS 74; ME 4; ME 4 (carbon); Metanex D; Methane black; MHI 201; MHI 220; MHI 273; MHI 5732; MHI Black 209; MHI Black 217; MHI Black 220; MHI Black 4962M; MHI Black 8704M; MHI Black 971; MHI-K 220; Microdis I; Microjet Black CW 1; Microjet Black M 800; Microjet C; Microlith Black C-K; Microlith Black C-T; Microlith Black C-T 85095; Microlith Black C-WA; Microlith Black CA; Microlith Black M; Micropigmo Black WM-BK 5; Micropigmo WM-BK 5; Microsol Black 2B; Mikuni 0542; Mitsubishi 1000; Mitsubishi 20B; Mitsubishi 2400; Mitsubishi 2400B; Mitsubishi 258; Mitsubishi 260; Mitsubishi 2770B; Mitsubishi 30; Mitsubishi 3030; Mitsubishi*

*3050; Mitsubishi 30B; Mitsubishi 3150; Mitsubishi 3400; Mitsubishi 40; Mitsubishi 44; Mitsubishi 45; Mitsubishi 47 Mitsubishi 50; Mitsubishi 650; Mitsubishi 900; Mitsubishi Carbon 10; Mitsubishi Carbon 25; Mitsubishi Carbon 40 Mitsubishi Carbon 44; Mitsubishi Carbon 45; Mitsubishi Carbon 50; Mitsubishi Carbon 52; Mitsubishi Carbon Black 2000; Mitsubishi Carbon Black 2600; Mitsubishi Carbon Black 3050; Mitsubishi Carbon Black 33; Mitsubishi Carbon Black 44 Mitsubishi Carbon Black 900; Mitsubishi Carbon Black 950; Mitsubishi Carbon Black 990; MM 192; Mogul; Mogul (carbon black); Mogul A; Mogul E; Mogul L; Mogul L 3; Molacco; Monaprin Black XBE; Monarch 1000; Monarch 1100; Monarch 120; Monarch 1300; Monarch 1400; Monarch 1500; Monarch 280; Monarch 4; Monarch 460; Monarch 4750; Monarch 580; Monarch 700; Monarch 71; Monarch 800; Monarch 81; Monarch 880; Monarch 900; Monarch Black 1300; Monarch Fluffy 435; Monarch M 800; Monocol 35T; Monocol 37T; Monocol MX 230; MPC Black; MPS 1100 Black (T); MPS 1504 Black (T); MSC 30; MT; MT (carbon black); MT Carbon MT-C; MT-CI; MT-N 990; MTCI; Multilac A 903 Black N 103; N 110; N 115; N 121; N 121HT1000; N 121HT1500; N 134 N 134 (filler); N 135; N 135 (carbon black); N 200; N 205; N 205 (carbon black); N 219; N 219 (carbon black); N 220; N 229; N 230; N 230 (carbon black); N 231; N 234; N 240; N 242; N 293; N 294; N 296; N 296 (carbon black); N 299; N 326; N 326M; N 326MP; N 326N; N 326T; N 330; N 335; N 339; N 343; N 347; N 351; N 351H; N 356; N 358; N 375; N 472; N 539; N 550; N 550G; N 550M; N 550U; N 582; N 630; N 630 (carbon black); N 650; N 650 (carbon black); N 650H; N 660; N 660 (carbon black); N 667; N 683; N 705; N 754; N 760; N 760 (carbon black); N 762; N 76225; N 76230; N 765; N 769; N 770; N 770 (carbon); N 772; N 774; N 785; N 787; N 834; N 908UP; N 990; N 990 (carbon black); N 991; Nanom Black FB-S; Neo-Spectra Beads AG; Neo-Spectra Mark I; Neo-Spectra Mark II; Neocon 600P; Neotex 100H; New Lacqutimine Black FLPR; New Lacqutimine Color Black FLTR Conc.; Nicabeads PC 0520; Nicabeads PC 1020; Nichilon 105; Nigros F; Nigros G; Nigros I; Nigros K; Nipex 150; Nipex 160IQ; NIPex 170IQ; Nipex 18; Nipex 180; Nipex 180IQ; NIPex 35; Nipex 60; Nipex 70; Nipex 90; NIPX 35; NIPX 60; Niteron 10; Niteron 10K; Niteron 200; Niteron 2000; Niteron 200B; Niteron 200H; Niteron 200IS; Niteron 200LG; Niteron 200MP; Niteron 300; Niteron 300B; Niteron 300MP; Niteron 3350; Niteron 410; Niteron 55; Niteron 55A; Niteron 55G; Niteron 75; Niteron FEF 10; NU 12-2-06; Nylofil Black BLN; OAB 100; OB 44; Oil 9B; Oil Furnace Black 44B; OneSource 9292B3546 Tint; Orient Black N 330; OTS OTS (carbon black); OTS-S; OTS-S/A; OTS-S/B; P 1250; P 145 P 154; P 226M; P 234; P 2410; P 243-0; P 245; P 245 (carbon) P 257E; P 267; P 267E; P 268E; P 324; P 33; P 33 (carbon black) P 357E; P 366E; P 367E; P 36G-E; P 399E; P 514; P 514 (carbon); P 701; P 701 (carbon black); P 702; P 705; P 803 P 803 (carbon black); P 814; P 814 (carbon black); PAF 50; Panther 17FB; PAU 1; PB 115; PBK 7; PCM-DH 1012; PE 2272; PE 3324; Peach black; Pearl 2000; Pearl Black 2000; Pearls 800; Peerless MK II; Pelletex; Pelletex SRF; PEM 8080BKMB; Peony Black 30940; Peony Black F 30940; Peony Black FF 30940MM; Permablack 2847A; Permablack 900; Permablak 663; PEX 986020; PEX 998004 Black; PEX 998023 Black; PF 300; PGM 33; PGM 40; Philblack; Philblack N 220; Philblack N 550; Philblack N 765; Philblack O; Picachem B 9; Pigmatex Black T; Pigment Black 07; Pigment Black 6; Pigment Black 7; Pigment Black FW 200 Beads; PK 7; Plasblak 3037; Plasblak EV 1755; Plasblak PE 1371; Plasblak PE 1851; Plasblak PE 2614; Plasblak PE 2640; Plasblak PE 2642; Plasblak PE 2648;*

*Plasblak PE 2662; Plastblak EV 1755; PLWTEXG; PM 100; PM 100 (carbon); PM 100A; PM 100KrSZ; PM 100V; PM 105K; PM 136.5; PM 15; PM 15 (carbon black); PM 15RVDM; PM 30V; PM 50; PM 50 (carbon black); PM 70; PM 70 (carbon black); PM 75; PM 80V; PM 90e; PME 100V; PME 70V; PME 80V; PMG 33; PMN 130; PMN 130N; PMO 130; PMTK 90; Pollux Black PM-B; Pollux Black PP 8TO85; Pollux Black PP-B; Porousblack; PP 8T1106; PPM 77255; Printex; Printex 140; Printex 140T; Printex 140U; Printex 140V; Printex 150T; Printex 200; Printex 25; Printex 30; Printex 300; Printex 35; Printex 40; Printex 400; Printex 45; Printex 55; Printex 60; Printex 70; Printex 75; Printex 75R; Printex 80; Printex 85; Printex 90; Printex 95; Printex A; Printex Alpha; Printex EC 2; Printex F 80; Printex Falpha ; Printex G; Printex L; Printex L 1; Printex L 40; Printex L 6 ; Printex P; Printex U; Printex V; Printex XA; Printex XE; Printex XE 2; Printex XE 2B; Printex XE-II; Printex XPB 080; Pritex 75; Product 11793F1 Black; PSB 183; PSM Black; Pureblack SCD 205; Pureblack SCD 530; Pureblack SCD 545; Pureblack SCD 550; Pureblack SCD 555; Purex HS 25; Purex HS 45; PV 817; Pyroblack 3S; Pyroblack 5AF; Pyroblack 5F; Pyroblack 7007; Pyroblack 7F; R 250; R 250 (pigment); R 250R; R 300; R 300 (carbon black); R 330; R 330R; R 400R; R 760; R 760 (carbon); R 880; Raven; Raven 1000; Raven 1020; Raven 1035; Raven 1040; Raven 1060; Raven 1060B; Raven 1080; Raven 11; Raven 1100; Raven 1100 Ultra; Raven 1170; Raven 1190 Ultra; Raven 1200; Raven 12200; Raven 125; Raven 1250; Raven 1255; Raven 1255B; Raven 14; Raven 15; Raven 150; Raven 1500; Raven 16; Raven 200; Raven 2000; Raven 22D; Raven 2500; Raven 2500 Ultra; Raven 30; Raven 3200; Raven 35; Raven 350; Raven 3500; Raven 360; Raven 3600 Ultra; Raven 40; Raven 410; Raven 420; Raven 420 Dense; Raven 430; Raven 430 Ultra; Raven 450; Raven 50; Raven 500; Raven 5000; Raven 5000UIII; Raven 520; Raven 5250; Raven 5720; Raven 5750; Raven 7000; Raven 760; Raven 760 Ultra; Raven 760B; Raven 780; Raven 780 Ultra; Raven 8000; Raven 860; Raven 860 Ultra; Raven 880 Ultra; Raven 890; Raven Beads; Raven Black; Raven C; Raven P-FE/B; RCC 6; RCF 10; RCF 10B; RCF 30; RCF 44; RCF 45L; RCF 50; Rebonex H; Rebonex HS; Rebonex I; Rebus 1106; Rebus Carbon Black 1106; Rega 199; Regal; Regal 1250R; Regal 250 Regal 250R; Regal 300; Regal 300R; Regal 330; Regal 330R; Regal 350R; Regal 400; Regal 400R; Regal 415R; Regal 500R; Regal 600; Regal 660; Regal 660R; Regal 700; Regal 85 Regal 99; Regal 99R; Regal Black 250R; Regal L; Regal R 330 Regal SRF; Regal SRF-S; Renol Black R-HW; Renol Black RT-HW; RL 00-02; Royal Spectra; RU 0262; Ryudye W Black RC S 160; S 160 (carbon black); S 170; S 170 (carbon black); S 2400 Black 4; S 300; S 300 (carbon black); S 315; SA Black DY 6; SAB 1; SAF; SAF (carbon); SAF-HS; SAF-LS; Sagal 3; SAGN 110; Sakap 10; Sakap 6; Sandye Black P; Sandye Black P Paste 2904; Sandye Black P Paste SL; Sandye DP Black P 2904; Sandye Super Black C; Sanylene Black EMA; Sapex 20; Sashinekka E; SB 100; SB 250; SB 4; SB 5; SB 5 (carbon); SB 500; SB 500 (carbon black); SB 6; SB 6 (carbon); SB4A; SBF-T 1683; SCBK 22; SCD 205; SCD 530; SCD 545; SCD 550; SCD 555; SD 10M; SD 9020; SD 9020(carbon black); SD 9134; SD 9139; Seast 116; Seast 116HM; Seast 116MAF; Seast 3; Seast 300; Seast 300HAF-LS; Seast 3H; Seast 5H; Seast 6; Seast 600; Seast 6T; Seast 7H; Seast 7HM; Seast 9; Seast 900A; Seast 9H; Seast 9M; Seast F; Seast FM; Seast FY Seast G 116; Seast G-SO; Seast G-SVH; Seast GS; Seast ISAF; Seast KH; Seast N; Seast N 211; Seast N 300; Seast NB; Seast NH; Seast S; Seast S-SRF; Seast SO; Seast SOP; Seast SP Seast SP-SRF-LS; Seast SY-SRF-HS 8500;*

*Seast V; Seika Seven Servacarb; Sevacarb MT; Sevacarb MT-CI; Sevacarb MT-CL; Sevacarb MT-LS; Sevacarb SC-N 990; Sevalco; SFC 4350 Shawinigan ABC 55-22913; Shawinigan Black; Shawinigan Black C 55; Shoblack; Shoblack FEF; Shoblack IP 200; Shoblack IP 600; Shoblack MAF; Shoblack N 110; Shoblack N 200; Shoblack N 220; Shoblack N 2201; Shoblack N 234; Shoblack N 326; Shoblack N 326M; Shoblack N 330; Shoblack N 330T; Shoblack N 335; Shoblack N 339; Shoblack N 351; Shoblack N 550; Shoblack N 660; Shoblack N 762; Shoblack S 118; SHPA 817; Sicoflush L Black 0054; Sicoflush L Black 0063; Sicoflush P Black 0054; SL 10; SL 10 (carbon black); SL 30; SL 30 (carbon black); Sohn Black; Soot FR 101; SOOTS (COAL SOOT EXTRACTS); Sorbead H; SP 250; SP 250 (carbon black); SP 350; SP 350 (carbon black); SP Black 8922; SP Black AS 1192; SP Black AS 1193; SPAB 8K500; Special black 100; Special black 15; Special Black 250; Special Black 250P; Special Black 350; Special black 4; Special Black 4A; Special black 5; Special black 6; Special Black Bayer A-SF; Special Black S 160; SPF 35; Spheron 4000A; Spheron 5000; Spheron 5000A; Spheron 6; Spheron 9; SR 129; SR 401; SRB 1; SRB 1 (carbon black); SRB 3; SRB-N 762; SRF; SRF 772; SRF Black; SRF Carbon; SRF-HS; SRF-L 35; SRF-N 770; SS Fujikura Black; Statex 160; Statex 550CBL; Statex B; Statex B 12; Statex G; Statex GH; Statex M 70; Statex MRG; Statex MT; Statex MT 90; Statex N 200; Statex N 550; Statex N 650; Sterling 1120; Sterling 142; Sterling 2320; Sterling 4620; Sterling 6630; Sterling 6630A; Sterling FT; Sterling FT-FF; Sterling MT; Sterling MTG; Sterling N 550; Sterling N 765; Sterling NS; Sterling NS 1; Sterling R; Sterling R-V 7688; Sterling RX 76; Sterling SO; Sterling SO 1; Sterling SO-N 550; Sterling SR; Sterling V; Sterling VH; Sterling VL; Sun Black LHD 9303; Sunblack 250; Sunblack 605; Sunblack 970; Sunblack X 15; Sunblack X 25; Sunblack X 45; Sunsperse Black LHD 9303; Supandai PLR-FC 121; Super Black 205; Super Colloid 6; Super P-Li; Super S; Super S (filler); Supercarbovar; Superjet LB 1011; Suprapal Black 2XS8A734; Suprapal Black X 60; SZ 7740; T 10189; T 1375 Black (R) EC; T 4; T 4 (carbon black); T 900; T 900 (carbon black); T 9068G; T 990; T 990 (carbon black); T-NS; T-NS (carbon black); TACK 1; TB 4300; TB 4501; TB 4550; TB 510; TB 5500; TB 575 Black S-T 2; TB 7240F; TB 7550F; TB-A 700F; TBK-BC 3; TC 415; TC-N 550; TeG 10; TeG 10 (carbon); Termax N 990; Termoks 277KhIT; TET 1999; TG 10; TH 110; Thai Black N 339; Thermal Black; Thermal MT-CB; Thermax; Thermax 907; Thermax Floform CC-N 990; Thermax Floform N 990; Thermax Medium Thermal Black MT; Thermax MT; Thermax N 990; Thermax N 991; Thermax Stainless; Thermax Ultra Pure N 991; Tintacarb 300; Tintacarb 435; TM 15; TM 30; TM 30 (carbon black); TM 50; TM 50 (carbon black); TM 70; TM 75; Toka Black 3800; Toka Black 3855; Toka Black 3885; Toka Black 4400; Toka Black 4400F; Toka Black 4500; Toka Black 4500F; Toka Black 4550F; Toka Black 5400; Toka Black 5500; Toka Black 7100; Toka Black 7100F; Toka Black 7240; Toka Black 7350; Toka Black 7350F; Toka Black 7360SB; Toka Black 7550; Toka Black 7550F; Toka Black 7700; Toka Black 8300; Toka Black 8500; Toka Black 8500F; Toka Black A 700F; Tokai Carbon 600A; Tokai Carbon ESR; Toner Black 020; Toral AS 1; TPH 0012; TS 1; TS 1 (carbon); TVP 0623 BLACK; UK-Vulcan P; Ultrabond Black K; UM 66; UM 76; UM 85; UN 1361; UN 1993 (DOT); UNA 4; Unipure Black LC 902; Unisperse Black B-PI; Unisperse Black C-E 2N; Unisperse Black C-S; United 3017; United SL 90; UX 10; V 1391; V4; V4 (carbon black); ValKan 72XC; VC Black; Vcx 500; VPSP 20016; Vulcan; Vulcan (carbon black); Vulcan 10H; Vulcan 1345; Vulcan 1380; Vulcan 1391; Vulcan 6; Vulcan 66; Vulcan 72; Vulcan 9;*

*Vulcan 9A32; Vulcan C; Vulcan J; Vulcan K; Vulcan M; Vulcan P; Vulcan PF 300; Vulcan VX 72; Vulcan X 72; Vulcan XC; Vulcan XC 200; Vulcan XC 272; Vulcan XC 305; Vulcan XC 605; Vulcan XC 72; Vulcan XC 72R; Vulcan XC 72R-GP3820; VXC 200; VXC 305; VXC 500; VXC 605; VXC 7; VXC 72; VXC 72R; W 311N; W 356A; W 9793; WA Black A 250; Witcoblack 100; X 1303; X 1341; X 1396; X 55; XC 3017L; XC 305; XC 37; XC 500; XC 605; XC 72; XC 72R; XE 2; XE 2B; XE 37; XF 72; Xfast Black 0066; XLH 81; XLH 82; XPB 080; XPB 171; XPB 255; XPB 289; XPB 297; XPB-AT 1234; Y 200; Y 50A; Y 70; Y 70 (carbon black); YML 100; YP 17; Z 271; Z 281; Z 312; ZCP; ZCP (carbon black)*

# Appendix II. Upper-bounding estimates of exposure to carbon black via inhalation

| Scenario | Data/Assumptions | Estimated exposure |
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| Airborne exposure near carbon black manufacturing plant | Dispersion modelling of particulate emissions from main stack, for a period of 5 years, conducted to comply with Certificate of Approval. Most of the particulate emissions were reported to be carbon black (Environment Canada 2010a)<br><br>Used US EPA ISCST3 dispersion model, local digital terrain elevation data, meteorological data from nearby airport, and August 2001 main stack measurements.<br><br>Sum of emissions measured from 15 other dust collector exhausts on site in 2000 were much less than those from the main stack described above (0.4% of total).<br><br>The maximum $PM_{10}$ predicted at the nearest residence was used to estimate exposure because this was considered more relevant to exposure of local receptors than was the concentration at the point of impingement<br><br>**General assumptions:**<br>- Body weight: 70.9 kg (Health Canada 1998)<br><br>**Inhalation route:**<br>- Inhalation rate: 16.2 $m^3$/day (Health Canada 1998)<br>- Breathing zone concentration of carbon black: 0.0016 mg/$m^3$ (acute) and 0.0001 mg/$m^3$ (chronic) (Environment Canada 2010a)<br>- Indoor concentrations of carbon black assumed to be the same as outdoors (considered conservative)<br><br>Acute exposure = (16.2 $m^3$/day × 0.0016 mg/$m^3$) ÷ 70.9 kg<br><br>Chronic exposure = 16.2 $m^3$/day × 0.0001 mg/$m^3$) ÷ 70.9 kg<br><br>A bag house was subsequently installed (in 2004) to reduce emissions; in testing conducted in 2006, mean stack emissions were reduced 99%. This decrease has not been incorporated in estimating exposure because it is not known whether the other two manufacturing facilities in Canada use similar pollution control technology. | **Concentration**<br>Maximum $PM_{10}$, nearest residence<br>Acute<br>- 24 h average (avg): 1.6 µg/$m^3$ (0.0016 mg/$m^3$)<br>Chronic<br>- Annual avg: 0.1 µg/$m^3$ (0.0001 mg/$m^3$)<br><br>**Acute intake** = 0.00037 mg/kg per day<br>**Chronic intake** = 0.000023 mg/kg per day |

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| Painting walls using an airless sprayer[a] | Maximum weight fraction identified in non-black paints in the US Household Products Database: 1% w/w (HPD 2009)<br><br>**General assumptions**<br>- Exposure frequency: 1/year (RIVM 2006a)<br>- Body weight: 70.9 kg (Health Canada 1998)<br><br>**Inhalation route**<br>- Inhalation rate: 0.675 $m^3$/hour (Health Canada 1998)<br>- Breathing zone concentration of respirable paint aerosols: 5.14 mg/$m^3$ (NPCA 2004)[b]<br>- Exposure duration: 3 hours (NPCA 2004)<br>- Respirator with particle filter is used, removes 95% of respirable paint aerosol (PMRA 2000, 3M Occupational Health and Safety Division 2010). The use of suitable respiratory protection is recommended both in the manuals for airless sprayers and at DIY shops that sell and rent these sprayers, as well as for any spray application of paint. The assumed respiratory protection is for a recommended respirator. Such units are modestly priced and readily available at DIY stores. Other types of readily available protection, such as N95 masks, afford a similar degree of protection to that assumed in the assessment.<br><br>Acute exposure =<br>(0.675 $m^3$/hour × 5.14 mg/$m^3$ × 1% × 5% × 3 hours) ÷ 70.9 kg | **Concentration**<br>0.00257 mg/$m^3$ during event, adjusted for respirator use<br><br>**Intake**<br>= 0.0000734 mg/kg per event |
| Sanding paint | Maximum weight fraction identified in non-black paints in the US Household Products Database: 1% w/w (HPD 2009)<br><br>**General assumptions**<br>- Exposure frequency: 1/year (RIVM 2006a)<br>- Body weight: 70.9 kg (Health Canada 1998)<br>- Weight fraction non-volatile in paint: 0.3 (RIVM 2007)<br>- No Personal Protective Equipment (PPE) used<br><br>**Inhalation route**<br>- Inhalation rate: 0.675 $m^3$/hour (Health Canada 1998)<br>- Breathing zone concentration of total respirable dust from sanding: 0.15 mg/$m^3$ (NPCA 2004)[c]<br>- Exposure duration: 2.4 hours (NPCA 2004)<br><br>Acute exposure =<br>(0.675 $m^3$/hour × 0.15 mg/$m^3$ × 1% × 2.4 hours) ÷ (70.9 kg × 0.3) | **Concentration**<br>0.0015 mg/$m^3$ during event<br><br>**Intake**<br>= 0.00011 mg/kg per event |

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| Dyeing hair with costume spray hair dye | Weight fraction in the sole costume spray hair dye containing carbon black reported to the Cosmetics Notification Database system: 2.27 % w/w (October 2010 email from Consumer Product Safety Bureau, Health Canada to Water, Air and Climate Change Bureau, Health Canada; unreferenced)<br><br>**General assumptions**<br>- Exposure frequency: 6/year (RIVM 2006b)<br>- Body weight: 70.9 kg (Health Canada 1998)<br><br>**Inhalation route: exposure to spray, spraying toward exposed person**<br>- Inhalation rate: 0.675 $m^3$/hour (Health Canada 1998)<br>- Exposure duration 5 minutes, room volume 10 $m^3$, ventilation rate 2/hour, mass generation rate 0.47 g/second, spray duration 0.24 minute, airborne fraction 1, weight fraction non-volatile 0.03, density non-volatile 1.5 $g/cm^3$, weight fraction non-volatile 0.03, room height 2.5 m, cloud volume 0.0625 $m^3$ (RIVM 2006b)<br>- Inhalation cutoff 10 µm (modified from RIVM 2006b)<br><br>Acute exposure = (0.675 $m^3$/hour × 0.000169 mg/$m^3$ ×5 minutes) ÷ (70.9 kg × 60 minutes/hour) | **Concentration**<br>0.000169 mg/$m^3$ during event<br><br>**Intake**<br>= 0.000000134 mg/kg per event |

[a] Spray painting would not be the method of choice for most homeowners/consumers to paint large areas owing to the potential for contaminating non-target surfaces.

[b] The maximum breathing zone concentration of respirable aerosol from test painting of walls in controlled studies (NPCA 2004) was selected.

[c] The maximum breathing zone concentration of total respirable dust from sanding by professional painters in controlled studies (NPCA 2004) was selected to represent an upper bound for do-it-yourself (DIY) projects by homeowners.

# Appendix III. Summary of health effects information

| Endpoint | Lowest effect levels[a]/results |
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| **Laboratory animals and *in vitro*** | |
| Acute toxicity | *Lowest oral $LD_{50}$*<br>>10 g/kg body weight in male and female rats (Henry and Kaufman 1973; OECD 2006)<br><br>[Additional acute oral studies: studies cited in OECD 2006; studies cited in CCOHS 2009]<br><br>*Lowest dermal $LD_{50}$*<br>>3000 mg/kg in rabbits (US EPA 2005; CCOHS 2009)<br><br>*Lowest inhalation effect levels*<br>1 mg/m$^3$ of 20 nm carbon black for 7 hours in male HAN rats, small (1%) but significant neutrophil influx in lung, marked increase in epithelial permeability and reduction of total lung GSH. No effect with 200 nm carbon black. Aggregate/agglomerate size would have been larger but was not reported (Li et al. 1997; OECD 2006).<br><br>[Additional acute inhalation studies: Ford et al. 1998; Vincent et al. 2001; Gilmour et al. 2004; OECD 2006; Hamade et al. 2008]<br><br>[Acute intratracheal instillation studies: Muller et al. 2005; OECD 2006; Yamamoto et al. 2006; Bachoual et al. 2007; Chang et al. 2007; Yokohira et al. 2007; Jacobsen et al. 2009;]<br><br>[Pharyngeal aspiration study: Zhao et al. 2009] |
| Short-term repeated-dose toxicity | *Lowest inhalation effect levels*<br>400 µg/m$^3$ carbon black (Regal 660) 3 hours for 4 days in groups (n = 15) of male C57BL/6, C3H/HeJ, and B6C3F1 mice age 18 and 28 months. Increase in left ventricular diameter, right ventricular and pulmonary vascular pressure, ROS, and MMP2 and MMP9 (indicates cardiac stress and remodelling). Decrease in fractional shortening and ejection fraction. Toxicity not as apparent in 18-month-old mice as in 28-month-old mice (Tankersley et al. 2008).<br><br>[Additional short-term inhalation study: Niwa et al. 2008]<br><br>[Short-term intranasal study: Shwe et al. 2006]<br><br>No cardiovascular toxicity was observed in C57BL/6, HeJ, and OuJ mice exposed by inhalation to approximately 0.556 mg/m$^3$ carbon black (Regal 660) for 3 hours/day for 3 days (Hamade and Tankersley 2009).<br><br>No effects on the liver were observed in C57BL/6 mice exposed |

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| | to 20 mg/m$^3$ carbon black (Printex 90) for 90 minutes/day for 4 days (Saber et al. 2009). |
| Subchronic toxicity | **Inhalation toxicity in rats, mice, and hamsters**<br><br>Groups of female F344 rats, B63F1 mice, and F1B Syrian golden female hamsters were exposed to carbon black (Printex 90) as follows: 1, 7, or 50 mg/m$^3$ for 6 hours/day, 5 days/week, for 13 weeks and killed 1 day, 3 months, and 11 months post-exposure; bronchioalveolar lavage fluid was analyzed. Levels of tumour necrosis factor-α and macrophage inflammatory protein-2 were elevated in a dose-dependent manner (significance seen at 7 mg/m$^3$), greatest in rat. Rats produced significantly higher ROS and reactive nitrogen species levels (7 mg/m$^3$) than did either the mouse or the hamster (hamster the lowest). Therefore greatest effect with 50 mg/m$^3$, LOEC 7 mg/m$^3$, greatest impact of carbon black in rat (Carter et al. 2006). In another study with identical protocol (Printex 90 and Sterling V), prolonged carbon black lung retention was found in rats and mice exposed to 7 mg/m$^3$. Lung inflammation and histopathology more severe and prolonged in rats than in mice and hamsters. NOEL of 1 mg/m$^3$ for all species (Elder et al. 2005).<br><br>[Additional subchronic inhalation study in hamsters: Snow 1970] |
| Chronic toxicity/ carcinogenicity | **Non-cancer inhalational effects**<br><br>CD-1 mice were exposed to carbon black at 1.5 mg/m$^3$ for 4, 12, or 20 weeks for 3 hours/day, 5 days/week. There were morphological changes in the respiratory tract and decreased resistance to infection (Fenters et al. 1979).<br><br>NOEL – 1.1 mg/m$^3$ (Driscoll 1996)<br><br>[Additional chronic inhalation studies: Snow 1970; Nau et al. 1976; Dungworth et al. 1994; Mauderly 1994; Heinrich et al. 1995]<br><br>*Primates*<br>Rhesus monkeys were exposed to carbon black (thermal black): 53 mg/m$^3$ for 6 hours/day, 6 days/week, for 3 years. Massive accumulations of carbon black particles in the lungs. Evidence of moderate to severe emphysema and hypertrophy of the right ventricular septum (Nau et al. 1976).<br><br>**Non-cancer oral and dermal effects**<br><br>No toxicity was observed in rats and mice following oral exposure of 10% carbon black in feed for 72 weeks or 2.05 g/kg carbon black in feed for 2 years on survival rate or gross pathology/microscopic changes in a wide range of tissues (Nau et al. 1958a, 1976; Pence and Buddingh 1985). |

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| | No dermal toxicity was observed in 240 mice after 41 weeks (123 applications) of 20% emulsions of thermal blacks or control vehicles. No detectable changes were observed, and carbon black did not have an effect on body weight (Nau 1976).<br><br>**Inhalation carcinogenicity**<br>Groups of 100 females Wistar Crl (WI) BR rats were exposed whole-body to sequential doses of carbon black as follows: 7.4 $mg/m^3$ for 4 months, 12.2 $mg/m^3$ for 20 months, and clean air conditions for 6 months for 18 hours/day, 5 days/week. There was a marked increase in lung tumours, the incidences of adenomas, adenocarcinomas, and squamous-cell carcinomas being 13, 13, and 4%, respectively, compared with one adenocarcinoma in 217 controls. In addition, 20 benign cystic keratizing squamous-cell tumours were seen in the treated females as compared with none in the controls. The lung particle burden was 44 mg/lung (Heinrich et al. 1995).<br><br>Groups of 72 female Wistar Crl (WI) BR rats were exposed whole-body to carbon black as follows: 6 $mg/m^3$ for 43 weeks and clean air for 86 weeks *or* 6 $mg/m^3$ for 86 weeks and clean air for 43 weeks for 17 hours/day, 5 days/week, compared with clean air controls. Lung tumours were observed in 17% (12/72) and 8% (6/72) of the rats exposed for 43 and 86 weeks, respectively (no statistically significant difference between the two groups), compared with none in the clean-air controls. In the 43-week group the tumours included 2 adenomas, 4 adenocarcinomas, 1 squamous-cell carcinoma, and 7 benign CKSC tumours; in the 86-week group they included 1 adenoma, 1 squamous-cell carcinoma, and 4 benign CKSC tumours. Six rats in the 86-week exposure group also showed marked hyperplasia or marked squamous-cell proliferation (Heinrich et al. 1994).<br><br>Groups of 135–136 female and 138–139 male Fischer 344/N rats were exposed whole-body to carbon black (furnace black, Elftex 12) at 0, 2.5, or 6.5 $mg/m^3$ for 16 hours/day, 5 days/week, for up to 24 months. Three rats of each sex from each group were sacrificed after 3, 6, 12, 18, or 23 months for histopathological examination. Exposed females showed a clear dose-related increase in benign and malignant lung tumours (mainly adenomas and adenocarcinomas), with incidences of 0, 7.5, and 26.7% in control, low-dose, and high-dose groups, respectively. No such effect was observed in males (equivalent incidences 2.8, 1.9, and 3.8% respectively). Squamous cysts (a type of non-neoplastic lesion) were seen in 0/91, 8/90, and 13/87 in control, low-dose, and high-dose females, respectively, and in 0/86, 1/73, and 4/74 control, low-dose, and high-dose in males, respectively. (Nikula et al. 1995)<br><br>No pulmonary carcinogenicity was observed in mice following |

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| | inhalation exposure to carbon black as follows: 7.4 mg/m$^3$ for 4 months, 12.2 mg/m$^3$ for 20 months, and kept under clean air conditions for 6 months for 18 hours/day, 5 days/week. Fewer lung tumours in exposed animals compared with clean air controls (Heinrich et al. 1995).<br><br>**Intratracheal carcinogenicity**<br>Female Wistar rats were instilled with either 3 mg carbon black suspended in 0.9% saline (n = 37) or 0.4 mL 0.9% saline (n = 39) once a week for 15 weeks. Animals were observed for 131 weeks. In the exposed group, 65% rats had primary lung tumours (adenomas, adenocarcinomas, squamous cell carcinomas, and CKSC tumours) compared with none in the control rats (Pott et al. 1994).<br><br>Groups of 48 treated (or 47 control) female Wistar Crl:(WI)BR rats were treated with either furnace black or lamp black as follows: 1 mg carbon black once a week for 16–17 weeks suspended in a mixture of 0.9% NaCl and 0.25% Tween 80. Animals were observed for up to 27 months. Lung tumours were observed in 10 females exposed to furnace black (one adenoma, four carcinomas, and nine CKSC tumours) and in four females exposed to lamp black (all CKSC tumours), compared with none in the controls. The lung particle burden 1 day after the last treatment was 11 mg/lung (Dasenbrock et al. 1996).<br><br>[No additional inhalation/intratracheal studies identified in rats]<br><br>**Oral carcinogenicity**<br>No carcinogenicity was observed in both mice and rats exposed orally to 0 or 2.5 g/kg body weight of carbon black in feed for 2 years. All tissues were examined for gross pathology, only tissues with macroscopically diagnosed lesions were examined histologically. No increase in tumour incidence with exposure (Pense and Buddingh 1985).<br><br>**Dermal carcinogenicity**<br>CFW white and C3H brown male mice (unspecified numbers) were treated thrice-weekly with three carbon blacks (furnace, thermal, or channel black) at concentrations of 10% and 20% in cottonseed, mineral oil, or 1% carboxymethyl cellulose for 12–18 months. No local skin carcinogenicity was observed (Nau et al. 1958b).<br><br>[Additional (negative) dermal carcinogenicity study: Nau et al. 1976] |
| Reproductive/ developmental toxicity | No significant differences among control and treated groups were found for the number of implantation sites or pups per litter, survival rate, pup weight, or pup length in a study in Fischer 344 rats, in which groups of 10 females were exposed by inhalation to 100 µg/m$^3$ carbon black for 4 hours/day on days 11–20 of |

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| | pregnancy (Archibong et al. 2002).<br><br>Partial vacuolation of the seminiferous tubules was observed more frequently in groups of 15–16 male ICR mice exposed by intratracheal instillation to 0.1 mg of carbon black (Printex 90, Printex 25, and Flammruss 101) once a week for 10 weeks compared with controls. In addition, elevation of serum testosterone concentration and decreased daily sperm production was observed (Yoshida et al. 2008).<br><br>An *in vitro* study examining the effect of carbon black (Printex 90) on mouse Leydig TM3 cells for 24 hours at 1000 µg/mL showed significantly decreased cell viability. 48-hour incubation enhanced steroidogenic acute regulatory mRNA expression in the cells at 10 and 30 µg/mL. No effect on HO-1 expression (Komatsu et al. 2008).<br><br>No oral or dermal studies identified. |
| Genotoxicity and related endpoints: *in vivo* | *Mutagenicity*<br>**Negative:** male and female F344/N rats, inhalation of furnace black (Elftex 12) at 0, 2, 46, and 6.55 mg/m$^3$ for 16 hours/day, 5 days/week, for 1 year. No consistent K-*ras* or p53 mutation spectrum in pulmonary carcinomas (Swafford et al. 1995; IARC 1996).<br>**Positive:** Groups of four male F344 rats, inhalation of furnace black (Monarch 880) at 0, 1.1, 7.1, and 52.8 mg/m$^3$ for 6 hours/day, 5 days/week, for 13 weeks. *Hprt* mutation frequencies in alveolar type II cells were significantly increased at 7.1 mg/m$^3$ immediately after exposure. At 52.8 mg/m$^3$, mutant frequencies were 4.3-, 3.2-, and 2.7-fold higher than in control levels immediately, 3 months, or 8 months post-exposure, respectively. Also lung inflammation, epithelial hyperplasia, and fibrosis were observed. The addition of an antioxidant (catalase) inhibited the increase of mutation frequency (Driscoll et al. 1996; IARC 1996).<br>**Negative:** *Drosophila melanogaster* larvae fed diets containing 1% carbon black until pupation. Examined mosaics, and incidence of Y-chromosome loss, chromosomal aberrations, or dominant lethal and sex-linked lethal mutations (Kirwin et al. 1981).<br><br>*DNA damage (DNA adducts by $^{32}$P-postlabelling assay)*<br>**Negative:** female F344 rats (groups of three), inhalation of Printex 90 at 0, 1, 7, or 50 mg/m$^3$ or Sterling V at 50 mg/m$^3$ for 13 weeks (Borm et al. 2005).<br>**Negative:** groups of six male F344/N rats, inhalation of furnace black (Elftex 12) at 10 mg/m$^3$ for 7 hours/day, 5 days/week, for 12 weeks (Wolff et al. 1990).<br>**Negative:** female Wistar Crl(WI)BR rats, inhalation of Printex 90 furnace black at 7.5 mg/m$^3$ for 4 months followed by 12 mg/m$^3$ for 20 months, 18 hours/day, 5 days/week (Gallagher et |

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| | al. 1994; IARC 1996).<br>**Positive:** male and female F344/N rats, inhalation of furnace black (Elftex 12) at 6.2 mg/m$^3$ for 16 hours/day, 5 days/week, for 12 weeks. Significant increases in DNA adducts in alveolar type II cells (25 and 5 adducts/10$^9$ bases in exposed and filtered-air control rats, respectively) (Bond et al. 1990; IARC 1996).<br><br>*DNA damage* (strand breaks by Comet assay)<br>**Positive:** in lung cell suspension from male C57BL/6J mice (groups of five), intratracheal instillation of carbon black (Printex 90) at 0.2 mg/mouse for 3 and 24 hours (Totsuka et al. 2009).<br><br>*Oxidative DNA damage*<br>**Positive:** female F344 rats (groups of 10 females), inhalation of Printex 90 at 0, 1, 7, or 50 mg/m$^3$ or Sterling V at 50 mg/m$^3$ for 6 hours/day, 5 days/week, for 13 weeks. For 14 nm, carbon black increased 8-OHdG levels immediately after the 13-week exposure at 50 mg/m$^3$ and at 7 mg/m$^3$ after the 44-week recovery period. 70-nm carbon black had no effect on 8-OHdG formation even with lung particle overload occurring (Gallagher et al. 2003). |
| Genotoxicity and related endpoints: *in vitro* | **Bacterial tests:**<br><br>*Mutagenicity*<br>**Negative:** *Salmonella typhimurium* TA98, TA100, TA1535, TA1537, and TA1538 strains. 7.5 mg/plate with and without S9. Note that cells were suspended in DMSO for unspecified duration (Kirwin et al. 1981).<br>**Positive:** *Salmonella typhimurium* TA98, TA100, TA98NR, and TA98/1, 8DNP strains. Extracts of 20 commercial carbon blacks. Fifteen of the 20 extracts were said to be mutagenic to strains TA98 and/or TA100, while the other five were inactive. Note that benzene was used in the extraction process. Mutagenicity attributed to presence of PAHs (Agurell and Lofroth 1983).<br><br>**Mammalian cell tests:**<br><br>*Chromosome damage (micronucleus assay)*<br>**Positive:** RAW264.7 cells, incubations of carbon black (Huber 90) at 1, 3, and 10 µg/mL for 48 hours (Poma et al. 2006).<br>**Positive:** Foetal Syrian hamster lung epithelial cells, 0.1–2.0 µg/mL Furnace Black (Printex 90) for 72 hours without S9. Dose-related increase in the frequency of micronuclei, although maximal responses were only approximately 50% greater than the control frequency of 4.5% (Riebe-Imre et al. 1994; IARC 1996).<br>**Positive:** A549 cells, increased percentage of micronucleated cells (up to 3.3%) with doses of carbon black (Printex 90) up to 2 µg/mL (percentage of micronucleated cells plateau beyond this dose). In addition, 200 µg/mL carbon black for 6 hours caused growth inhibition of 60% (Totsuka et al. 2009). |

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| | *Chromosome damage (sister chromatid exchange)*<br>**Negative:** CHO cells, 0.32–1000 µg/mL of furnace black (N339) for 2 hours in presence and absence of S9. Note that cells were suspended in DMSO for unspecified duration (Kirwin et al. 1981; IARC 1996).<br><br>*DNA damage (DNA adduct formation)*<br>**Negative:** in human alveolar epithelial cell line (A549 cells), 24-hour incubations of Printex 90, Sterling V, N330, and Lampblack at ≤300 µg/cm$^2$ (Borm et al. 2005).<br><br>*DNA damage (Comet assay)*<br>**Positive:** primary mouse embryo fibroblasts, 24-hour incubations of carbon black at 5 and 10 µg/mL (Yang et al. 2009).<br>**Positive:** A549 and THP-1 cells, without S9, 48-hour incubations of furnace black (Vulcan M) at 0.016–1.6 µg/mL (Don Porto Carero et al. 2001).<br>**Positive:** single-stranded breaks in A549 cells, without S9, 3-hour incubations of carbon black (Printex 90) at 100µg/mL. Also induced Ser15-p53 phosphorylation and NFκB expression (Mroz et al. 2007).<br>**Positive:** A549 cells, 3-hour exposure to carbon black (Printex 90) resulted in single-stranded breaks along with alterations of cell cycle kinetics. Huber 990 did not cause DNA strand breaks (Mroz et al. 2008).<br>**Positive:** FE1 Muta™Mouse lung epithelial cells, 3-hour incubation of carbon black (Printex 90) at 2.08, 6.25, 18.75, and 75 µg/mL (Jacobsen et al. 2008).<br>**Positive:** in FE1 Muta™Mouse lung epithelial cell line, 75 µg/mL carbon black (Printex 90), exposure for 8 repeated 72-hour incubations. DNA strand breaks and oxidized purines (Jacobsen et al. 2007).<br><br>*Mutagenicity*<br>**Negative:** in mouse lymphoma cells (L5178Ytk$^{+/-}$), 10–40 mg/mL without S9 and 5 × 15 mg/mL with S9, exposure for >4 hours. Cell survival was <1% at highest concentration (Kirwin et al. 1981; IARC 1996).<br>**Weak Positive:** in lacZ and cII transgenes of murine epithelial cell line (Jacobsen et al. 2007).<br><br>*Other:*<br>Cell transformation assay<br>**Positive:** foetal Syrian hamster lung epithelial cells, 100–300 µg/mL Furnace black (Printex 90) for 72 hours without S9. Peak cell transformation responses occurred at 200 µg/mL in differentiated cells (fourfold increase over controls) and at 300 µg/mL in undifferentiated cells (eightfold increase over controls) (Riebe-Imre et al. 1994; IARC 1996).<br>**Negative:** C3H/10T1/2 mouse fibroblasts, 2–16 mg/mL. No |

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| | transformed focus observed (Kirwin et al. 1981).<br><br>Oxidative stress and inflammation<br>**Positive:** primary mouse embryo fibroblasts, incubations of carbon black at 5, 10, 20, 50, and 100 µg/mL for 24 hours, increased ROS formation and oxidative stress (Yang 2009).<br>**Positive:** SV40T2 (rat type II pulmonary epithelial cells transformed by SV40) and rat alveolar macrophages, incubations of 10 µg/mL carbon blacks (Printex 90 and Printex 25) caused increase in oxidative stress and HO-1 protein levels. 10 µg/mL of Flammuruss 101 also caused increase in HO-1 protein levels in alveolar macrophages (AM) (Koike and Kobayashi 2006).<br>**Positive:** rat AMs, incubations of 2.5–20 µg/mL carbon black (Regal 250R) caused an increase in ROS (Aam and Fonnum 2007).<br>**Positive:** A549 and NCl-H292 cells, incubations of carbon black at 100 µg/mL, increased inflammatory response (Newland and Richter 2008).<br>**Positive:** A549 cells, incubations of 63, 125, and 250 µg/mL carbon black (Printex 90) for 24 hours. No response with 31 µg/mL. 125 and 250 µg/mL carbon black (Huber 990) for 4 hours resulted in ROS production (Monteiller et al. 2007).<br>**Positive:** RAW 264.7 cells, incubations of 50 µg/mL carbon black, only increase in MMP-12 mRNA (not other inflammatory factors), no response with 5 µg/mL (Bachoual et al. 2007).<br>**Positive:** 16HBE14o- cells, incubations of carbon blacks at 12.5, 25, 50, and 100 µg/mL for 24 hours, caused an increase in ROS production and a pro-inflammatory response (5 µg/cm$^2$). No observable effects at 6.25 µg/mL dose (Hussain et al. 2009).<br>**Positive:** BEAS-2B cells, incubations of carbon black (Printex 90) at 10 µg/cm$^2$ caused increase in CXCL8, -10, and -11 gene expression (pro-inflammatory) (Ovrevik et al. 2009).<br>**Positive:** NHBE and A549 cells, 1-hour exposure to carbon black (Printex 90) at 50 µg/mL resulted in increased ROS formation (greater increase with addition of DPPC to prevent aggregate formation) (Herzog et al. 2009).<br>**Positive:** 16HBE14o- cells, 24-hour exposure to carbon black at 5 or 10 µg/cm$^2$ resulted in a pro-inflammatory response. No response observed at 1 and 2.5 µg/cm$^2$ (Val et al. 2009).<br>**Positive:** in FE1 Muta™Mouse lung epithelial cell line, 75 µg/mL carbon black (Printex 90), exposure for eight repeated 72-hour incubations (Jacobsen et al. 2007).<br>**Negative:** RAW264.7 and rabbit whole blood, incubations of carbon black for varying lengths of time (24 hours to 50 days), no effect on macrophage phenotype or platelet aggregation (Niwa and Iwai 2007).<br>**Positive:** Human monocytes, macrophages, bronchial epithelial cells, A549 cells, and Calu-3 cells, incubations of carbon black (Printex 90) at 32 µg/mL for 3 hours, caused an increase in CYP1B1 gene expression (Eder et al. 2009).<br>**Positive:** bronchial epithelial cells (16HBE14o-), incubations of |

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| | 20 µg/cm$^2$ carbon black caused morphological and biochemical features of apoptosis, ROS, and hydrogen peroxide production (Hussain et al. 2010).<br>**Positive:** adult male Wistar rat cardiomyocytes and cardiofibroblasts (primary culture), 0, 50, 100, 200, or 400 µg/mL of carbon black (Printex 90) for 2, 4, 8, or 24 hours. Particle concentration-dependent increase in IL-6 release observed in cardiomyocyte mono and co-cultures (Totlandsdal et al. 2008). |
| **Humans** | |
| Epidemiological Studies | *Genotoxicity*<br>Positive association in peripheral blood lymphocytes, 28 workers in tire industry exposed to carbon black over a period of 2–8 years, incidence 5.07% in exposed compared with 2.27% in unexposed controls, little information on exposure groups or co-exposures (Babu et al. 1989, abstract only).<br>[No additional genotoxicity studies identified.]<br><br>*Respiratory effects*<br>Two phases of a study of respiratory symptoms and lung function in 2342 and 1994 male workers in 19 and 16 European carbon black production factories, respectively; estimated mean current exposure to inhalable dust was 0.77 mg/m$^3$ and 0.57 mg/m$^3$, respectively, with a mean duration of employment of 175 and 178 months (i.e., 14.6 and 14.8 years). In both phases, there were positive associations between current exposure level and cough, cough with sputum production, and chronic bronchitis, and a borderline significant association with sputum production; slight and borderline significant associations were also observed for cumulative exposure. In lung function testing, positive associations were observed between current and cumulative exposure and forced mid-expiratory flow and the forced expiratory volume/forced vital capacity ratio. Positive associations, although only reaching borderline significance, were observed for forced expiratory volume in the first phase for current exposure and in both phases for cumulative exposure. All data assessed using multiple linear regression, adjusting for the effects of factory, age, height, and smoking (Gardiner et al. 2001).<br><br>[Additional respiratory studies: Valic et al. 1975; Oleru et al. 1983; Robertson et al. 1988; Gardiner et al. 1993; Kupper et al. 1996; Robertson and Inman 1996; van Tongeren et al. 2002; Harber et al. 2003]<br><br>*Cancer*<br>Retrospective cohort; UK; 1422 men employed for at least 1 year between 1947–1974 in one of the five major carbon black production factories; exposure based on industrial hygiene measurements taken in 1976. Positive association with lung cancer deaths, SMR: 1.5; 95% CI, 1.0–2.2 (all factories); the |

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| | factory with the greatest risk of mortality from lung cancer had lower-than-average carbon black exposure levels; no association with urinary cancer deaths; no data on smoking; IARC describes study as most informative (Hodgson and Jones 1985; IARC 1996). Follow-up study of cohort to 1996; used limited work histories to calculate estimates of individual exposure; increased mortality seen for all neoplasms (SMR: 142; 95% CI, 119–168) and lung cancer (SMR: 173; 95% CI, 132–222), not elevated at all factories when examined separately; again no link found between cumulative exposure to carbon black and risk of lung cancer (Sorahan et al. 2001).<br><br>Nested case-control study; cumulative exposure index based on type of job and duration of employment; Texas, Oklahoma, and Louisiana; male workers age ≥ 15 years employed in 1980 at any of the seven carbon black producers, 24 cases and 48 controls; unrepresentative number of cases. No increased risk of cancer or specifically skin cancer (Robertson and Ingalls 1989; IARC 1996).<br><br>Retrospective cohort; Texas, Oklahoma, and Louisiana; no details of exposure measure given; male employees of four carbon black producers for at least 1 year between 1935 and 1974; no association with cancer mortality; no information on smoking, IARC felt study had methodological limitations (Robertson and Ingalls 1980; IARC 1996). Follow-up study, cohort members from two companies followed for another 20 years, short communication (Robertson and Inman 1996).<br><br>Population-based case-control study; Montreal; male residents age 35–70 diagnosed with cancer between 1979 and 1985; cumulative exposure categorized as substantial or "any" (only 5% of study population ever exposed). Positive associations for cancer of the esophagus (11 cases, OR: 2.2; 95% CI, 1.1–4.4), kidney (14 cases, OR: 1.9; 95% CI, 1.1–3.3) and lung (52 cases, OR: 1.6; 95% CI 1.1–2.3); no associations for cancer of the stomach, colon, rectum, pancreas, prostate, bladder, skin melanoma, or non-Hodgkin's lymphoma (Siemiatycki 1991). In a follow-up study, incidence of oat-cell tumours of the lung was greatest for highly exposed workers; exposure categorized as either substantial or non-substantial; OR: 5.1; 95% CI, 1.7–14.9 using cancer controls and OR: 4.8; 95% CI, 1.4–17.0 using population controls; controlled many factors including smoking and exposures to other carcinogens (IARC 1996; Parent et al. 1996).<br><br>Two case-control studies conducted in Montreal (including the re-analysis of the data from Parent et al. 1996); 857 and 1236 lung cancer cases with matched controls; exposure determined based on job history. No association with risk of lung cancer, regardless of level of exposure; smoking a potential confounding |

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| | factor was not considered when matching cases and controls (Ramanakumar et al. 2008).<br><br>Retrospective cohort; Germany; 8933 men in rubber industry for at least 1 year between 1950 and 1981; workers classified as exposed or unexposed. Positive association for laryngeal cancer, hazard rate ratio: 5.3; 95% CI, 1.3–21.4 among workers exposed to carbon black after a 10-year lag; no evidence of association with lung or stomach cancer deaths; limited study, appeared to be confounded by exposure to nitrosamines, asbestos, and talc (IARC 1996; Straif et al. 2000).<br><br>Retrospective cohort; Genova, Italy; 2101 longshoremen employed at three dockyard companies between 1933 and 1980; categorized as having lifetime exposure to low, medium, or high levels of carbon black. Positive association for bladder cancer, standardized incidence ratio: 204; 95% CI, 112–343 for highly exposed workers. Note that after 1958 exposure levels reduced due to changes in carbon black handling. (Puntoni et al. 2001, 2004).<br><br>Case-control study; Sweden; 254 men in general population diagnosed with urothelial cancer from 1985–1987, and 287 controls; classified as ever or never exposed. No association with urothelial cancer; adjusted for smoking, other occupational exposures; some workers had been exposed to printing inks and other substances (Steineck et al. 1990; IARC 1996).<br><br>Cohort study; USA; mortality patterns among 5011 employees (mean length of employment 6.7 years) at 18 carbon black facilities from 1930s to 2003 as compared to state-specific mortality rates; considered exposed if ever assigned a job with potential exposure to carbon black. No association with mortality for all-cause, all-cancer, lung cancer, bladder cancer, or from non-malignant respiratory diseases; individual exposure to carbon black and smoking not available; observed a decrease in all-cause and all-cancer mortality with exposure to carbon black, which may be due to healthy worker effect (Dell et al. 2006).<br><br>Cohort study; Germany; mortality of male workers employed at a carbon black manufacturing plant between 1960 and 1998 for at least 1 year; exposure based on work history; positive association with all-cause mortality and mortality from lung cancer using national and state rates although no dose-response relationship; results may be affected by healthy worker effect (i.e. risk underestimated), smoking data incomplete (Wellmann et al. 2006).<br><br>Cohort study; Germany; lung cancer deaths of male workers in carbon black production plant within 15 years after employment; little information on exposure. Although lung cancer standard |

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| | mortality ratio increased with time from leaving employment, there was no significant association and no dose-response relationship observed between carbon black exposure and elevated lung cancer deaths (Morfeld and McCunney 2007).<br><br>Case-control study; Germany; lung cancer mortality among 1528 male carbon black production workers employed for at least 1 year between 1960 and 1998 and matched controls; exposure based on type of carbon black and cumulative working years. No association with lung cancer mortality observed, but there was a positive association for quartz; lamp workers could have also been exposed to shales (silica) and PAHs (Buchte et al. 2006).<br><br>Cohort study; UK; 1147 male workers from five carbon black manufacturing factories employed between 1947 and 1974 for at least 12 months; limited available work histories used to estimate individual cumulative exposure. Positive association with lung cancer mortality but only at two plants and for those who worked within last 15 years, which may be due to differences in carbon black particle size used at different plants; no association with overall mortality (Sorahan and Harrington 2007).<br><br>Retrospective cohort study; Germany; 1528 carbon black workers (50 lung cancer deaths) followed between 1976 and 1998; increased hazard ratio for lung cancer mortality per 10 years for the lamp black sub-section when applying a lag of 20 years; no association with cumulative carbon black exposure; may have also been exposed to shales and high levels of PAHs (Morfeld et al. 2006).<br><br>Nested case-control study; Arkon, Ohio; men employed in rubber manufacture industry in 1964 or earlier; categorized as low, medium, or high exposure based on concentration and frequency of exposure. 65 cases of squamous cell carcinoma and 254 matched controls; appears to have no association but 95% CI not given; no exposure–response relationship or any trend by duration of exposure (Bourguet et al. 1987; IARC 1996).<br><br>[Additional cancer studies: Ingalls 1950; Ingalls and Risquez-Iribarren 1961; Blair et al. 1990; Parent et al. 2000] |

[a] Definitions: 8-OHdG, 8-hydroxydeoxyguanosine; CKSC, cystic keratinizing squamous cell; HR, heart rate; $LD_{50}$, median lethal dose; LOEL, lowest-observed-effect level; MMP, matrix metalloproteinase; NOEL, no-observed-effect level; ROS, reactive oxygen species.