SCREENING-LEVEL HAZARD CHARACTERIZATION

Petroleum Coke Category

SPONSORED CHEMICALS
Petroleum coke, green CASRN 64741-79-3
Petroleum coke, calcined CASRN 64743-05-1

The High Production Volume (HPV) Challenge Program[1]was conceived as a voluntary initiative aimed at developing and making publicly available screening-level health and environmental effects information on chemicals manufactured in or imported into the United States in quantities greater than one million pounds per year. In the Challenge Program, producers and importers of HPV chemicals voluntarily sponsored chemicals; sponsorship entailed the identification and initial assessment of the adequacy of existing toxicity data/information, conducting new testing if adequate data did not exist, and making both new and existing data and information available to the public. Each complete data submission contains data on 18 internationally agreed to "SIDS" (Screening Information Data Set[1,2]) endpoints that are screening-level indicators of potential hazards (toxicity) for humans or the environment.

The Environmental Protection Agency's Office of Pollution Prevention and Toxics (OPPT) is evaluating the data submitted in the HPV Challenge Program on approximately 1400 sponsored chemicals by developing hazard characterizations (HCs). These HCs consist of an evaluation of the quality and completeness of the data set provided in the Challenge Program submissions. They are not intended to be definitive statements regarding the possibility of unreasonable risk of injury to health or the environment.

The evaluation is performed according to established EPA guidance[2,3] and is based primarily on hazard data provided by sponsors; however, in preparing the hazard characterization, EPA considered its own comments and public comments on the original submission as well as the sponsor's responses to comments and revisions made to the submission. In order to determine whether any new hazard information was developed since the time of the HPV submission, a search of the following databases was made from one year prior to the date of the HPV Challenge submission to the present: (ChemID to locate available data sources including Medline/PubMed, Toxline, HSDB, IRIS, NTP, ATSDR, IARC, EXTOXNET, EPA SRS, etc.), STN/CAS online databases (Registry file for locators, ChemAbs for toxicology data, RTECS, Merck, etc.) and Science Direct. OPPT's focus on these specific sources is based on their being of high quality, highly relevant to hazard characterization, and publicly available.

OPPT does not develop HCs for those HPV chemicals which have already been assessed internationally through the HPV program of the Organization for Economic Cooperation and Development (OECD) and for which Screening Initial Data Set (SIDS) Initial Assessment Reports (SIAR) and SIDS Initial Assessment Profiles (SIAP) are available. These documents are presented in an international forum that involves review and endorsement by governmental

[1] U.S. EPA. High Production Volume (HPV) Challenge Program; http://www.epa.gov/chemrtk/index.htm.
[2] U.S. EPA. HPV Challenge Program – Information Sources; http://www.epa.gov/chemrtk/pubs/general/guidocs.htm.
[3] U.S. EPA. Risk Assessment Guidelines; http://cfpub.epa.gov/ncea/raf/rafguid.cfm.

authorities around the world. OPPT is an active participant in these meetings and accepts these documents as reliable screening-level hazard assessments.

These hazard characterizations are technical documents intended to inform subsequent decisions and actions by OPPT. Accordingly, the documents are not written with the goal of informing the general public. However, they do provide a vehicle for public access to a concise assessment of the raw technical data on HPV chemicals and provide information previously not readily available to the public.

Chemical Abstract Service Registry Number (CASRN)	**Sponsored Chemicals** **64741-79-3** **64743-05-1**
Chemical Abstract Index Name	**Sponsored Chemicals** **Coke (petroleum)** **Coke (petroleum), calcined**
Structural Formula	**See Appendix**

Summary

CASRN 64741-79-3 is a grayish-black, carbonaceous solid that is obtained from the heaviest portions of crude oil. CASRN 64743-05-1 is a product derived from CASRN 64741-79-3 under reducing conditions in kilns or hearths heated to over 1,200°C. These substances possess negligible vapor pressure and negligible water solubility. Volatilization is negligible. The rate of hydrolysis is negligible. The rate of atmospheric photooxidation is negligible. CASRN 64741-79-3 and CASRN 64743-05-1 both possess high persistence (P3) and low bioaccumulation potential (B1).

A guideline study is not available for acute inhalation toxicity; however, no mortality occurred following five days of repeated inhalation exposure to CASRN 64741-79-3 (0.058 mg/L) or CASRN 64743-05-1 (0.045 mg/L) in rats. No other data are available for CASRN 64743-05-1. Repeated exposure to CASRN 64741-79-3 dust during a 2-year inhalation toxicity study produced irreversible respiratory effects (chronic pulmonary inflammation and significantly increased absolute/relative lung weights) in rats and primates (both sexes) at all concentrations tested. Histological examination revealed macrophage accumulation (with test article deposits), focal fibrosis, bronchiolization, sclerosis and squamous alveolar metaplasia in rats at concentrations $\geq$ 0.01 mg/L; the NOAEC for systemic toxicity is not established. A combined reproductive/developmental toxicity screening test with CASRN 64741-79-3 dust showed no reproductive or developmental effects following inhalation exposure in rats; however, pulmonary inflammation (macrophage accumulation, lymphocyte hyperplasia and squamous metaplasia of respiratory epithelium) was observed in all exposed parental animals. The NOAEC for maternal toxicity is not established. The NOAEC for reproductive/developmental toxicity is 0.30 mg/L (highest concentration tested). CASRN 64741-79-3 was not mutagenic in bacteria or mammalian cells when tested *in vitro* and did not induce chromosomal aberrations in mice following inhalation exposure *in vivo*. Repeated dermal exposure to CASRN 64741-79-3

(as a 25% suspension in mineral oil) during a 2-year cancer bioassay produced acanthosis and hyperkeratosis in mice; however, no neoplastic changes were observed.

Based on the category member CASRN 64741-79-3, the 96-h LC_{50} for fish and the 48-h EC_{50} for aquatic invertebrates are no effects at saturation. Based on the category member CASRN 64741-79-3, the 96-h EL_{50} for aquatic plants is greater than 1000 mg/L (WAF nominal loading rate). Based on the category member CASRN 64741-79-3, the 21-d terrestrial plants (corn, radish and soybean) NOEC and the 14-d earthworms NOEC are no effects at saturation.

No data gaps for were identified under the HPV Challenge Program.

The sponsor, the American Petroleum Institute (API) Petroleum HPV Testing Group, submitted a Test Plan and Robust Summaries to EPA for petroleum coke on March 31, 2000. EPA posted the submission on the ChemRTK HPV Challenge website on April 21, 2000 (http://www.epa.gov/chemrtk/pubs/summaries/ptrlcoke/c12563tc.htm). EPA comments on the original submission were posted to the website on August 14, 2000. Public comments were also received and posted to the website. The sponsor submitted updated/revised documents on December 28, 2007, which were posted to the ChemRTK website on June 30, 2008. The petroleum coke category consists of the following substances:

Coke (petroleum), "green coke"	CASRN 64741-79-3
Coke (petroleum), calcined	CASRN 64743-05-1

Category Justification

This category contains both green and calcined petroleum coke. The sponsor's rationale for this grouping is based on similarities in manufacture and processing. Their reasoning suggests that as byproducts of oil refining processes (at high temperature and pressure), these substances share similar physical-chemical characteristics that are expected to produce comparable toxicity. The sponsor proposed use of test data for green petroleum coke in a read across approach to estimate potential toxicities that may be associated with exposure to calcined petroleum coke. EPA agrees that it is appropriate for green and calcined petroleum coke to be grouped in one category and accepts the proposed read across approach for this hazard characterization.

1. Chemical Identity

1.1 Identification and Purity

Petroleum coke (both green and calcined) is a black solid produced by the high pressure thermal decomposition of heavy (high boiling) petroleum process streams and residues. The specific chemical composition of any given batch of petroleum coke is determined by the quality of feedstocks used in the coking process. Green coke is the initial product formed during the cracking and carbonization of feedstocks used to produce a substance with a high carbon-to-hydrogen ratio. Green coke may undergo additional thermal processing at very high temperatures to produce calcined coke. The additional processing required to form calcined coke removes most of the remaining volatile matter (< 0.5%), thereby increasing the percentage of elemental carbon and the relative abundance of metals. Compositional information on green coke is shown in Table 6 of the Appendix.

1.2 Physical-Chemical Properties

The physical-chemical properties of coke (petroleum) "green coke" and coke (petroleum), calcined are summarized in Table 1, while the environmental fate properties are provided in Table 2. In general, most physical-chemical and environmental fate properties are not applicable for these substances as they cannot be measured or estimated accurately.

Coke (petroleum) "green coke" and coke (petroleum), calcined are both grayish-black, solid (carbonaceous) materials that are produced during the thermal conversion process with crude oil. These substances possess negligible vapor pressure and negligible water solubility.

Table 1. Physical-Chemical Properties of Petroleum Coke[1,2]

Property	Coke (petroleum)	Coke (petroleum), calcined
CASRN	64741-79-3	64743-05-1
Molecular Weight	Complex mixture	Complex mixture
Physical State	Black-colored solid	Black-colored solid
Melting Point	Not applicable	Not applicable
Boiling Point	Not applicable	Not applicable
Vapor Pressure	<.000001 mm Hg (Negligible)	Negligible
Dissociation Constant (pK_a)	Not applicable	Not applicable
Henry's Law Constant	Negligible	Negligible
Water Solubility	< 0.0000001 g/L (Negligible)	Negligible
Log K_{ow}	Not applicable	Not applicable

[1] American Petroleum Institute Petroleum HPV Testing Group. 2007. Revised Robust Summary and Test Plan for Petroleum Coke. Available online at http://www.epa.gov/chemrtk/pubs/summaries/ptrlcoke/c12563tc.htm as of January 21, 2011.
[2] Predel, H. 2005. Petroleum Coke. Ullmann's Encyclopedia of Chemical Technology. Wiley Online Library.

2. General Information on Exposure

2.1 Production Volume and Use Pattern

The Petroleum Coke category chemicals had an aggregated production and/or import volume in the United States greater than two billion pounds in calendar year 2005.

- CASRN 64741-79-3: 1 billion pounds and greater;
- CASRN 64743-05-1: 1 billion pounds and greater;

CASRN 64743-05-1:
No industrial processing and uses or commercial and consumer uses were reported for this chemical.

CASRN 64741-79-3:
Non-confidential information in the IUR indicated that the industrial processing and uses for this chemical include petroleum refineries as fuels. Non-confidential commercial and consumer uses of this chemical include "other."

2.2 Environmental Exposure and Fate

If released to soils, coke (petroleum) and coke (petroleum), calcined will become incorporated into the soil, as they have no mobility. They are essentially inert; therefore, biodegradation,

atmospheric photooxidation, and hydrolysis will be negligible. Volatilization is negligible. These substances are not bioaccumulative. Coke (petroleum) and coke (petroleum), calcined both possess high persistence (P3) and low bioaccumulation potential (B1).

Table 2. Environmental Fate Properties of Petroleum Coke[1,2]

Property	Coke (petroleum)	Coke (petroleum), calcined
CASRN	64741-79-3	64743-05-1
Photodegradation Half-life	Stable	Stable
Hydrolysis Half-life	Stable	Stable
Biodegradation	Stable	Stable
Bioaccumulation Factor	Not applicable	Not applicable
Log K_{oc}	Not applicable	Not applicable
Fugacity (Level III Model) Air (%) Water (%) Soil (%) Sediment (%)	Not applicable	Not applicable
Persistence	P3 (High)	P3 (High)
Bioaccumulation	B1 (Low)	B1 (Low)

[1] American Petroleum Institute Petroleum HPV Testing Group. 2007. Revised Robust Summary and Test Plan for Petroleum Coke. Available online at http://www.epa.gov/chemrtk/pubs/summaries/ptrlcoke/c12563tc.htm as of January 21, 2011.

[2] Traditional environmental fate properties cannot be measured or accurately estimated for these substances; however, it is assumed that these substances will be stable in the environment and non-bioaccumulative due to their high molecular weight.

Conclusion: Coke (petroleum) "green coke" is a grayish-black carbonaceous solid that is obtained from the heaviest portions of crude oil. Petroleum (coke), calcined is a product derived from coke (petroleum) under reducing conditions in kilns or hearths heated to over 1,200°C. These substances possess negligible vapor pressure and negligible water solubility. Volatilization is negligible. The rate of hydrolysis is negligible. The rate of atmospheric photooxidation is negligible. Coke (petroleum) and coke (petroleum), calcined possess high persistence (P3) and low bioaccumulation potential (B1).

3. Human Health Hazard

A summary of health effects data submitted for SIDS endpoints is provided in Table 3. The table also indicates where data for the supporting chemical are read-across (RA) to the sponsored chemical.

Acute Inhalation Toxicity

A guideline acute inhalation toxicity study (OECD 403) is not available for green petroleum coke; however, no mortalities occurred in the 5-day or 2-year repeated-dose inhalation studies described below.

Green petroleum coke (CASRN 64741-79-3)
Calcined petroleum coke (CASRN 64743-05-1)

(1) Male Fischer 344 rats (40/group) were administered green petroleum coke dust (100% purity) at 58.2 mg/m^3 or calcined petroleum coke dust (99.5% purity) at 45.0 mg/m^3 (~ 0.058 or 0.045 mg/L, respectively) via (nose-only) inhalation 6 hours/day for 5 consecutive days. Positive and negative controls received silicon dioxide and titanium dioxide, respectively. The mass median aerodynamic diameters for green and calcined petroleum coke particles were 2.71 and 2.69 μm, respectively. Ten animals from each group were sacrificed at 7, 28 and 63 days post-exposure. No mortalities occurred. An increased incidence of chromodacryorrhea (red tears) was apparent in all treatment groups except titanium dioxide. At terminal sacrifice, biochemical and cytological examinations were made of bronchoalveolar lavage fluid. Histological examination of lung tissue was confined to animals sacrificed at 63 days post-exposure. Examination of bronchoalveolar lavage fluid obtained at 7 and 28 days post-exposure revealed no indication of pulmonary toxicity in exposed or control rats; however, evidence of pulmonary inflammation (increased n-acetylglucosamidase, neutrophils, lymphocytes, total protein and total cell count) was evident in both silicon dioxide and petroleum coke exposed rats at 63 days post-exposure. Macroscopic examination showed red discoloration of the lungs and parabronchial lymph nodes in petroleum coke-exposed animals. The rank order of increasing severity was: titanium dioxide < calcined petroleum coke < green petroleum coke < silicon dioxide. No signs of pulmonary fibrosis were observed in this study.

LC_{50} (Green petroleum coke) > ~ 0.058 mg/L
LC_{50} (Calcined petroleum coke) > ~ 0.045 mg/L

Repeated-Dose Toxicity

Green petroleum coke (CASRN 64741-79-3)

(1) Sprague-Dawley rats (150/sex/group) were administered (Delayed process) green petroleum coke dust (average mass median aerodynamic diameter = 3.1 ± 1.9 μm) via whole-body inhalation of the aerosol at 0, 10.2 or 30.7 mg/m^3 (~ 0.010 or 0.031 mg/L, respectively) for 6 hours/day, 5 days/week for 2 years (Klonne et al., 1987). Clinical chemistry (alanine aminotransferase, alkaline phosphatase, aspartate aminotransferase, blood urea nitrogen, calcium, phosphorus, total bilirubin, total protein, and glucose) and hematologic evaluations (mean corpuscular volume, hematocrit, hemoglobin, erythrocyte, reticulocyte, leukocyte and platelet counts) were conducted after 3, 6, 12, 18 and 24 months of exposure using ten randomly selected rats per group. Interim sacrifices were made at 5 and 30 days (10/sex/group), at 3, 6 and 12 months (20/sex/group) and at 18 months (10/sex/group) post-exposure. All surviving animals were sacrificed at 24 months. All animals sacrificed in extremis or found dead were also evaluated. Fasting body and organ weights (heart, lung plus trachea, liver, gonads, adrenals, thyroid/parathyroids, kidneys, spleen and brain) were recorded at each scheduled necropsy. Thirty-one designated tissues (not specified) from control and high exposure groups (10 rats/sex) were examined microscopically after 3,6,12 and 18 months; all remaining animals from control and high exposure groups were similarly evaluated after 24 months of exposure. Only the lung plus trachea (at 12, 18 and 24 months) and nasal turbinates (at 24 months) were examined microscopically in the lowest exposure group.

There were no treatment-related effects on body/organ weights, serum biochemistry, cytogenetic evaluations, ophthalmologic examinations or mortality; however, macroscopic examination revealed pigment accumulation (presumably test material) and gray/black discoloration of the lungs and thoracic lymph nodes in exposed animals. Significant, dose-related increases in absolute and relative lung (plus trachea) weights and chronic pulmonary inflammation (significant elevations in the number of segmented neutrophils and leukocytes and a decreased number of lymphocytes) was also observed following exposure at 0.010 and 0.03 mg/L. Histological changes observed in treated rats include macrophage accumulation, bronchiolization (adenomatous hyperplasia), focal fibrosis, sclerosis and squamous alveolar metaplasia (keratin cysts). Observed lung effects were non-reversible and increased in severity with increasing concentration and duration of exposure.
LOAEC ~ 0.010 mg/L (based on pulmonary inflammation and histopathology)
NOAEC = Not established

Green petroleum coke (CASRN 64741-79-3)
(2) Mature Cynomolgus (Macaca fascicularis) monkeys (4/sex/group) were administered (Delayed process) green petroleum coke dust (average mass median aerodynamic diameter = 3.1 ± 1.9 μm) via whole body inhalation of the aerosol at 0, 10.2 or 30.7 mg/m^3 (~ 0.010 or 0.031 mg/L, respectively) 6 hours/day, 5 days/week for 2 years (Klonne et al., 1987). No mortalities occurred. Ophthalmologic, clinical chemistry (alanine aminotransferase, alkaline phosphatase, aspartate aminotransferase, blood urea nitrogen, calcium, phosphorus, total bilirubin, total protein, and glucose) and hematologic evaluations (mean corpuscular volume, hematocrit, hemoglobin, erythrocyte, reticulocyte, leukocyte and platelet counts) were conducted at 1, 3, 6, 12, 18 and 24 months. At scheduled sacrifice (24 months), fasting body and organ weights (heart, lung plus trachea, liver, gonads, adrenals, thyroid/parathyroids, kidneys, spleen and brain) were recorded and thirty-one tissues (unspecified) from control and high exposure groups were examined microscopically. Only the lung (plus trachea) and nasal turbinates were examined in the lowest exposure group. There were no treatment-related effects on body/organ weights, serum chemistry, hematology, cytogenetic evaluations, ophthalmologic examinations or mortality; however, significant, dose-related increases in absolute and relative lung (plus trachea) weights were observed in both sexes at 0.010 and 0.03 mg/L. Histological examination showed macrophage accumulation (with test material deposits) and discoloration within the alveoli, thoracic lymph nodes and in paratracheal lymphoid tissue of all exposed animals. Observed lung effects were non-reversible and increased in severity with increasing concentration and duration of exposure. These findings are consistent with the development of pulmonary inflammation; however, no other evidence of inflammatory or metaplastic changes was reported.
LOAEC ~ 0.010 mg/L (based on pulmonary effects)
NOAEC = Not established

Reproductive/Developmental Toxicity

Green petroleum coke (CASRN 64741-79-3)
In a combined reproductive/developmental toxicity screening test, Sprague-Dawley rats (12/sex/group) were exposed via nose-only inhalation to micronized green petroleum coke (average mass median aerodynamic diameter = 2.29 μm) at 0, 30, 100 or 300 mg/m^3 (~ 0.030, 0.10 or 0.30 mg/L, respectively) for up to 52 days (Klonne et al., 1987). A two week range finding study was conducted initially to select exposure levels for the definitive study. In the

main study, rats were exposed for 6 hours/day for two weeks prior to mating. Males were then exposed for 28 days during the mating and post-mating period. Females continued to be exposed until evidence of mating, or for 14 consecutive days. Pregnant females were treated throughout gestation until scheduled sacrifice on postnatal day 4. Viability, clinical observations, body weights, feed consumption, survival, organ weights and macroscopic and microscopic findings were evaluated in parental rats. Standard reproductive (mating indices, pregnancy rates, male fertility indices, gestation length, number of implantation sites and corpora lutea, pre- and post-implantation loss, pups per litter, live born and stillborn pups and incidence of dams with no viable pups) and developmental indices (pup viability, weight, sex ratio and survival) were evaluated. Exposure-related parental effects included pigment deposition and associated discoloration of the lungs, mediastinal lymph nodes and nasal olfactory epithelium of male and female rats. Pigment deposits were also observed in the nasal turbinates and pharynx of male rats. Hyperplasia of paracortical T lymphocytes (in the mediastinal lymph nodes) and squamous metaplasia of respiratory epithelium (in the larynx) were also observed. All exposed animals showed evidence of pulmonary inflammation and discoloration. Significant dose-related increases in lung weights were observed in males (37%) and females (58%). No effects on reproductive or developmental parameters were reported in this study.
NOAEC (reproductive toxicity) > ~ 0.30 mg/L (highest concentration tested)
LOAEC (maternal toxicity) ~ 0.030 mg/L (based on pulmonary effects and histopathology)
NOAEC (developmental toxicity) > ~ 0.30 mg/L (highest concentration tested)
Genetic Toxicity – Gene Mutation

In vitro
Green petroleum coke (CASRN 64741-79-3)
(1) *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 were exposed to micronized green petroleum coke (Delayed process) dissolved in dimethylsulfoxide (DMSO) at 123.5, 370.4, 111.1, 333.3 and 10,000 µg/plate in the presence and absence of metabolic activation. No evidence of cytotoxicity was observed; however, precipitation occurred at the highest concentration tested (10,000 µg/plate). Results for positive and negative (solvent) controls were not reported in the robust summary.
Green petroleum coke was not mutagenic in this assay.

(2) *S. typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 were exposed to micronized green petroleum coke (Fluid process) dissolved in dimethylsulfoxide (DMSO) at 123.5, 370.4, 111.1, 333.3 and 10,000 µg/plate in the presence and absence of metabolic activation. No evidence of cytotoxicity was observed; however, precipitation occurred at the three highest concentrations tested. Heavy bacterial contamination also occurred at the highest concentration (~10,000 µg/plate). Results for positive and negative (solvent) controls were not reported in the robust summary.
Green petroleum coke was not mutagenic in this assay.

(3) L5417Y mouse lymphoma cells were exposed to (Delayed process) green petroleum coke dissolved in DMSO at concentrations up to 2000 µg/plate in the presence and absence of metabolic activation. Positive and negative controls were tested concurrently and responded appropriately. No precipitation or cytotoxicity was observed. Green petroleum coke did not induce forward mutations at the thymidine kinase locus in L5417Y mouse lymphoma cells.
Green petroleum coke was not mutagenic in this assay.

Genetic Toxicity – Chromosomal Aberrations

In vivo

Green petroleum coke (CASRN 64741-79-3)

(1) In the chronic inhalation study described above, cytogenetic evaluations were performed on bone marrow from Sprague-Dawley rats (10/sex/group) after five days, 1, 3 and 6 months, 1 year and 22 months of inhalation exposure to (Delayed process) green petroleum coke dust at 0, 10.2 or 30.7 mg/m^3. Due to high mortality in control and treated groups, only five to eight rats per group were evaluated after 22 months on test. No significant differences in chromosome aberrations were observed in treated rats when compared to controls.

Green petroleum coke did not induce chromosomal aberrations in this assay.

(2) In a 28-day inhalation repeated-dose toxicity study, cytogenetic evaluations were performed on bone marrow from Sprague-Dawley rats (8 males/group) that were exposed to (Delayed process) green petroleum coke (powder) at 0, 10 or 40 µg/L (nominal concentrations) 6 hours/day for 5 (high-dose group) or 20 consecutive days (low-dose group). A mitosis inhibitor (colchicine) was administered 24 hours post-exposure and bone marrow smears were made from the femur. No significant differences in chromosome aberrations were observed in treated versus control animals [TSCATS (OTS00001654)].

Green petroleum coke did not induce chromosomal aberrations in this assay.

Additional Information

Carcinogenicity

Green petroleum coke (CASRN 64741-79-3)

C3H mice (25/sex/group) were exposed to 100 µL green petroleum coke (as a 25% suspension in mineral oil) via topical application to shaved dorsal skin 3 times per week throughout their lifespan (two years). The positive control group was similarly exposed to benzo-a-pyrene via topical application twice per week. The negative control group was shaved, but remained untreated. Histological assessments were conducted on all mice. A wide range of tissues and organs (not specified) were examined. The incidence of acanthosis and hyperkeratosis increased with dermal exposure to green petroleum coke; however, no neoplastic changes were observed at the application site in petroleum coke-exposed animals. Positive controls developed squamous epithelial cell neoplasms at treated sites.

Green petroleum coke was not carcinogenic to mice in this study.

Conclusion: A guideline study is not available for acute inhalation toxicity; however, no mortality occurred following five days of repeated inhalation exposure to CASRN 64741-79-3 (0.058 mg/L) or CASRN 64743-05-1 (0.045 mg/L) in rats. No other data are available for CASRN 64743-05-1. Repeated exposure to CASRN 64741-79-3 dust during a 2-year inhalation toxicity study produced irreversible respiratory effects (chronic pulmonary inflammation and significantly increased absolute/relative lung weights) in rats and primates (both sexes) at all concentrations tested. Histological examination revealed macrophage accumulation (with test article deposits), focal fibrosis, bronchiolization, sclerosis and squamous alveolar metaplasia in rats at concentrations $\geq$ 0.01 mg/L; the NOAEC for systemic toxicity is not established. A

combined reproductive/developmental toxicity screening test with CASRN 64741-79-3 dust showed no reproductive or developmental effects following inhalation exposure in rats; however, pulmonary inflammation (macrophage accumulation, lymphocyte hyperplasia and squamous metaplasia of respiratory epithelium) was observed in all exposed parental animals. The NOAEC for maternal toxicity is not established. The NOAEC for reproductive/developmental toxicity is 0.30 mg/L (highest concentration tested). CASRN 64741-79-3 was not mutagenic in bacteria or mammalian cells when tested *in vitro* and did not induce chromosomal aberrations in mice following inhalation exposure *in vivo*. Repeated dermal exposure to CASRN 64741-79-3 (as a 25% suspension in mineral oil) during a 2-year cancer bioassay produced acanthosis and hyperkeratosis in mice; however, no neoplastic changes were observed.

Table 3. Summary Table of the Screening Information Data Set as Submitted under the U.S. HPV Challenge Program - Human Health Data

Endpoints	Green Petroleum Coke (CASRN 64741-79-3)	Calcined Petroleum Coke (CASRN 64743-05-1)
Acute Inhalation Toxicity LC_{50} (mg/L)	**> ~ 0.058**	**> ~ 0. 045**
Repeated-Dose Toxicity NOAEC/LOAEC Inhalation (mg/L/day)	**NOAEC = Not established LOAEC ~ 0.010** (based on chronic pulmonary inflammation and associated histopathology observed in a 2-year inhalation study)	No Data NOAEC = Not established LOAEC ~ 0.010 (RA)
Reproductive/Developmental Toxicity NOAEC/LOAEC Inhalation (mg/L/day)	**NOAEL = Not established**	No Data
Maternal Toxicity	**LOAEC ~ 0.030**	LOAEC ~ 0.030 (RA)
Reproductive Toxicity	**NOAEC > ~ 0.30**	NOAEC > ~ 0.30 (RA)
Developmental Toxicity	**NOAEC > ~ 0.30**	NOAEC > ~ 0.30 (RA)
Genetic Toxicity – Gene Mutation *In vitro*	**Negative**	No Data Negative (RA)
Genetic Toxicity – Chromosomal Aberrations *In vivo*	**Negative**	No Data Negative (RA)
Additional Information Carcinogenicity	**Negative**	No Data Negative (RA)

Measured data in bold text; (RA) = Read Across

4. Hazard to the Environment

A summary of aquatic toxicity data submitted for SIDS endpoints is provided in Table 4. The table also indicates where data for tested category members are read-across (RA) to untested members of the category.

EPA suggested that the sponsor conduct a chronic toxicity test in aquatic invertebrates with CASRN 64741-79-3 instead of acute toxicity for fish, aquatic invertebrates and aquatic plants because of a concern that leaching of hydrocarbons and metals from test substances into water may be too slow to result in effects during the acute toxicity period. The sponsor conducted acute toxicity tests using a water accommodated fraction (WAF) of the coke sample, which EPA believes that this approach can resolve the original EPA's concern.

Petroleum coke is sometimes used in a manner that can result in exposure to selected terrestrial species; therefore, the sponsor submitted the terrestrial plants test and earthworm test in addition to the acute toxicity tests for aquatic organisms.

Acute Toxicity to Fish

Green petroleum coke (CASRN 64741-79-3)
Fathead minnows *(Pimephales promelas*) were exposed to CASRN 64741-79-3 as water accommodated fractions (WAFs) under semi-static conditions for 96 hours in the closed system. The loading rates were 0 and 1000 mg/L (limit test). Milled and sieved CASRN 64741 to approximately 2 mm grain was used to prepare the WAF solutions. No mortality occurred and no clinical signs of toxicity were noted. Attempts to measure the constituents of the test substance (i.e. unalkylated polycyclic aromatic hydrocarbons (unalkylated PAHs), metals and sulfur) in aged and fresh WAFs showed that concentrations were below detection limits.
96-h LC_{50} = No effects at saturation.

Acute Toxicity to Aquatic Invertebrates

Green petroleum coke (CASRN 64741-79-3)
Daphnia (*Daphnia magna)* were exposed to CASRN 67471-79-3 as WAFs under semi-static conditions for 48 hours in the closed system. The loading rates were 0 and 1000 mg/L (limit test). Milled and sieved CASRN 64741to approximately 2 mm grain was used to prepare the WAF solutions. No immobility occurred and no clinical signs of toxicity were noted. Attempts to measure the constituents of the test substance (i.e. unalkylated PAHs, metals and sulfur) in aged and fresh WAFs showed that concentrations were below detection limits.
48-h EC_{50} = No effects at saturation.

Toxicity to Aquatic Plants

Green petroleum coke (CASRN 64741-79-3)
Freshwater algae *(Selenastrum capricornutum)* were exposed to CASRN 64741-79-3 as WAFs under static conditions for 96 hours in the closed system. The loading rates were 0 and 1000 mg/L (limit test). Milled and sieved CASRN 64741to approximately 2 mm grain was used to prepare the WAF solutions. Some statistically significant ($p < 0.05$) inhibition of growth (the

area under the growth curve; biomass) and growth rate were observed in the 1000 mg/L WAF at 72 hours (26 and 12%, respectively) and at 96 hours (28 and 7.1%, respectively), although no such effect was observed in prior range finding test. Attempts to measure the constituents of the test substance (i.e. unalkylated PAHs, metals and sulfur) in aged and fresh WAFs showed that concentrations were below detection limits.

96-h EL_{50} (biomass) > 1000 mg/L (WAF nominal loading rate)
96-h EL_{50} (growth rate) > 1000 mg/L (WAF nominal loading rate)

Toxicity to Terrestrial Plants

Green petroleum coke (CASRN 64741-79-3)

Corn (*Zea mays*), radish (*Raphanaus sativus*) and soybean (*Glycine max*) were exposed to soil-incorporated CASRN 64741-79-3 at 0 and 1000 mg/kg (limit test) for 21 days. CASRN 64741-79-3 milled to mean particle size of 3.3 µm was used to prepare soil-incorporated CASRN 64741-79-3. No statistically significant differences in all three species were found for seedling emergence, seedling survival, seedling height, and shoot dry weight between the dosed and control groups. Attempts to measure the constituents of the test substance (i.e. unalkylated PAHs, metals) in soil showed unalkylated PAHs were below detection limits and metals were not greater than soil background levels.

21-d LC_{50} = No effects at saturation.
21-d NOEC = No effects at saturation.

Toxicity to Soil Dwelling Organisms

Green petroleum coke (CASRN 64741-79-3)

Earthworms (*E. fetida*) were exposed to soil-incorporated CASRN 64741-79-3 at 0 and 1000 mg/kg for 14 days. CASRN 64741-79-3 milled to mean particle size of 3.3 µm was used to prepare soil-incorporated CASRN 64741-79-3. No mortality, aversion to the soil or soil burrowing behavior was observed. There were no statistical differences in earthworm body weight or change in body weight when measured at the end of the test. Attempts to measure the constituents of the test substance (i.e. unalkylated PAHs, metals) in soil showed unalkylated PAHs were below detection limits and metals were not greater than soil background levels.

14-d LC_{50} = No effects at saturation.
14-d NOEC = No effects at saturation.

Conclusion: Based on the category member CASRN 64741-79-3, the 96-h LC_{50} for fish and the 48-h EC_{50} for aquatic invertebrates are no effects at saturation. Based on the category member CASRN 64741-79-3, the 96-h EL_{50} for aquatic plants is greater than 1000 mg/L (WAF nominal loading rate). Based on the category member CASRN 64741-79-3, the 21-d terrestrial plants (corn, radish and soybean) NOEC and the 14-d earthworms NOEC are no effects at saturation.

Table 4. Summary Table of the Screening Information Data Set as Submitted under the U.S. HPV Challenge Program - Aquatic Toxicity Data		
Endpoints	**Green Petroleum Coke (64741-79-3)**	**Calcined Petroleum Coke (64743-05-1)**
Fish **96-h LC_{50} (mg/L)**	**NES**	No Data NES (RA)
Aquatic Invertebrates **48-h EC_{50} (mg/L)**	**NES**	No Data NES (RA)
Aquatic Plants **96-h EL_{50} (mg/L; WAF nominal loading rate)** **(growth rate)** **(biomass)**	 **> 1000** **> 1000**	No Data > 1000 > 1000 (RA)

Bold=experimental data (i.e. derived from testing); NES = No effects at saturation (water solubility limit); (RA) = Read Across

5. References

Klonne, D. R., Burns, J.M., Halder C.A., Holdsworth C.E., Ulrich C.E. Two-Year Inhalation Toxicity Study of Petroleum Coke in Rats and Monkeys. *Am. J. Indust. Med.* 11:375-389 (1987).

APPENDIX

The following pages show:

- Table 5 with a list of representative structures
- Table 6 with compositional information on green coke

Table 5. Structural Information on the Petroleum Coke Category		
Sponsored Chemicals		
Chemical Name	**CASRN**	**Structure[1]**
Coke (petroleum)	64741-79-3	A solid material resulting from high temperature treatment of petroleum fractions. It consists of carbonaceous material and contains some hydrocarbons having a high carbon-to-hydrogen ratio.
Coke (petroleum), calcined	64743-05-1	A complex combination of carbonaceous material including extremely high molecular weight hydrocarbons obtained as a solid material from the calcining of petroleum coke at temperatures in excess of 1,000°C (1,832°F). The hydrocarbons present in calcined coke have a very high carbon-to-hydrogen ratio.
[1]Meaningful molecular structures cannot be drawn for these highly carbonaceous, high molecular weight materials.		

Table 6. Compositional Data for Green Coke (taken from the Petroleum Coke Category Analysis and Hazard Characterization document: http://www.epa.gov/chemrtk/pubs/summaries/ptrlcoke/c12563rr2.pdf)

	Delayed Process Green Coke - 2003 Sample [1]				API Sample # 4-1-140 [2]	Micronized Delayed Process Green Coke – 1981 sample [3]	
Sample	pellet (initial)[4]	pellet (final)[5]	micro-nized (initial)	micro-nized (final)	Delayed Process Coke	1981 Analysis	1984 Analysis
Bi (bismuth)	<19.3		<29.6				
Ca (calcium)	178	81.7	121.6	158.7			
Cd (cadmium)	<9.6		<14.8				
Co (cobalt)	<9.6	1.9	<14.8	1.7			
Cr (chromium)	<9.6	3.9	<14.8	4.6			
Cu (copper)	<11.6	1.8	<17.8	2.3			
Fe (iron)	310	215.9	247	276.1			
Hg (mercury)					<1	<1	<0.01
K (potassium)	<28.9	10.9	<44.4	20.5			
Li (lithium)	<9.6	<1.2	<14.8	<1.16			
Mg (magnesium)	77.4	50.3	60.9	65.5			
Mn (manganese)	<19.3	5.3	<29.6	7.3			
Mo (molybdenum)	<19.3	16.7	<29.6	16.0			
Na (sodium)	133	87.8	114.6	99.0			
Ni (nickel)	367.1	319.6	351.7	304.6	95	78	85
P (phosphorus)	<19.3	19.8	30.3	25.0			
Pb (lead)	<19.3	4.88	<29.61	7.4			
Pd (palladium)		<6.9		<6.9			
Pt (platinum)		3.8		4.5			
S (sulfur)	73920		58060				
Sb (antimony)	<48.2		<74.0				
Se (selenium)	<19.3		<29.6		4.5	<0.2	<0.5
Si (silicon)	743.2	86.75		204			
Sn (tin)	<28.9	<2.3		<2.3			

Sample	Delayed Process Green Coke - 2003 Sample [1]				API Sample # 4-1-140 [2]	Micronized Delayed Process Green Coke – 1981 sample [3]	
	pellet (initial)[4]	pellet (final)[5]	micro-nized (initial)	micro-nized (final)	Delayed Process Coke	1981 Analysis	1984 Analysis
Average Mass Median Aerodynamic Particle Size, μm	2000*	2000*	2.3/3.3*		≤ 5**	3.1	3.1
Elemental Analysis, % wt							
Carbon					89.93	89.97	89.58
Hydrogen					3.71	5.04	3.89
Oxygen					1.3	1.62	2.14
Sulphur	7.4		5.8		3.36	3.27	3.42
Nitrogen					1.1	1.1	1.2
Other Analysis, % wt							
SiO2					0.04	<0.04	<0.02
Ash					0.21	0.19	0.28
Trace Metals, ppm							
Al (aluminum)	321	205.1	300.2	250.7			
As (arsenic)	<19.3	<2.3	<29.6	<2.3	<0.001	0.3	0.7
B (boron)	<19.3		<29.6				
Ba (barium)	<19.3	7.74	<29.6	6.9			
Be (beryllium)	<9.6		<14.8				

Sample	Delayed Process Green Coke - 2003 Sample [1]				API Sample # 4-1-140 [2]	Micronized Delayed Process Green Coke – 1981 sample [3]	
	pellet (initial)[4]	pellet (final)[5]	micro-nized (initial)	micro-nized (final)	Delayed Process Coke	1981 Analysis	1984 Analysis
Ti (titanium)	12.9	11.7	<14.8	14.4			
V (vanadium)	1938	1559	1805	1580	145	140	130
Zn (zinc)	12.0	8.9	<14.8	11.2			
Benzene Extract, % wt					1.79	2.08	2.64
PAHs, ppm							
Naphthalene	3.6	3.6	11	11			
1-methyl naphthalene	2.7	3.1	10	12			
2-methyl naphthalene	11	12	26	26			
Acenaphthene	ND	0.18	ND	0.51			
Acenaphthylene	ND	0.12	ND	0.5			
Fluorene	0.34	0.37	1.5	1.5	11	ND	ND
Phenanthrene	0.69	0.64	7.8	8.2	ND	ND	ND
Anthracene	ND	0.29	3.3	3.6			
Pyrene	1.3	1.2	8.6	10	ND	165	158
Fluroanthene	ND	0.1	1.4	1.6			
Benzofluorenes					ND	ND	ND
Benzo(a)anthracene	0.58	0.59	7.1	8	544		
Benzp(a,b)anthrcene						280	287
Chrysene	0.88	1.1	9.4	10	126	210	255
Benzo(a)pyrene	1.8	1.7	11	13	440	175	190
Benzo(e)pyrene					110	85	134
Beno(b)fluoranthene	0.52	0.62	3.8	3.9	ND	ND	ND
Benzo(k)fluoranthene	ND	ND	ND	1.5			

Sample	Delayed Process Green Coke - 2003 Sample [1]				API Sample # 4-1-140 [2]	Micronized Delayed Process Green Coke – 1981 sample [3]	
	pellet (initial)[4]	pellet (final)[5]	micro-nized (initial)	micro-nized (final)	Delayed Process Coke	1981 Analysis	1984 Analysis
Perylene					ND		
Methyl benzo(a)pyrene					ND	ND	
Benzo(g,h,i)perylene	1.1	1.4	8.7	12	439	120	167
Dibenzo(a,h)anthracene	0.49	0.51	4.1	4.3	ND	NQ	ND
Benzo(g,h,i)fluoranthene					ND	ND	ND
Indeno(1,2,3-cd)pyrene	0.34	0.45	3.5	3.3			
Dimethylbenz(a)anthracene							ND
Methylbenzo(g,h,i)perylene							377
Coronene					ND	ND	ND

Toxicology study(s) in which samples were used:

[1] OECD 203 Fish acute toxicity test; OECD 202 Invertebrate acute toxicity test; OECD 201 Algal growth inhibition test; OECD 208 Seedling emergence and growth of terrestrial plants; OECD 207 Earthworm acute toxicity test; OECD 421 Reproduction/developmental toxicity screening test

[2] Mouse dermal carcinogenicity study; *Salmonella* assay; mouse lymphoma cell assay

[3] Rat chronic inhalation study; Monkey chronic inhalation study; *Salmonella* assay; Rat *in vivo* cytogenicity assay

[4] initial refers to analyses conducted prior to initiation of the toxicology studies

[5] final refers to analyses conducted following completion of the toxicology studies

ND = not detected

NQ = detected, but not quantifiable

Blank cells = analysis not performed

* values are average mean particle size

** size not measured; value estimated from scanning electron micrographs

References: Aveka, Inc., 2003; CONCAWE, 1993; Chevron Products Company, 2003, 2005; Lancaster Laboratories, Inc., 2003, 2005.