ACTIVATED CARBON

Some regulatory bodies treat the carbon compounds with the CAS No 's 7440-44-0 and 1333-86-4 as one and the same. However the CAS No 7440-44-0 seems to relate to a more granular form of carbon that is treated to make it more absorbent, whilst the CAS No 1333-86-4 seems to relate to a more powdered version largely used as pigments.

SYNONYMS

AG 3 (Adsorbent), AG 5, AG 5 (Adsorbent), AK (Adsorbent), AR 3, ART 2, AU 3, Acticarbone, Activated carbon, Activated charcoal, Adsorbit, Amoco PX 21, Anthrasorb, Aqua nuchar, BAU, BG 6080, Black 140, Black pearls, CF 8, CF 8 (Carbon), CLF II, CMB 200, CMB 50, CUZ 3, CWN 2, Calcotone Black, Canesorb, Carbolac , Carbon , Carbon-12 , Carbopol Extra , Carbopol M , Carbopol Z 4, Carbopol Z Extra, Carbosieve, Carbosorbit R, Caswell No. 161, Coke powder, Cecarbon, Colgon BPL , Colgon PCB 12X30 , Colgon PCB-D , Conductex, Darco, Filtrasorb, Columbia LCK , Filtrasorb 200 , Filtrasorb 400 , Grosafe, Hydrodarco , Irgalite 1104 , Jado , K 257 , MA 100 (Carbon) , Norit , Nuchar, OU-B, Pelikan C 11/1431a , SKG, SKT, SKT (adsorbent) , SU 2000 , Suchar 681, Supersorbon IV , Supersorbon S 1 , U 02 , Watercarb, Witcarb 940, XE 340, XF 4175L or Carbon, colloidal.

CHEMICAL STRUCTURE

The basic structural unit of activated carbon is closely approximated by the structure of pure graphite. The graphite crystal is composed of layers of fused hexagons held by weak van de Waals forces. The layers are held by carbon–carbon bonds [DESOTECH 2009].

CHEMICAL FORMULA

C

IDENTIFIER DETAILS

CAS Number	:	7440-44-0
CoE Number	:	-
FEMA	:	-
EINECS Number	:	231-153-3
E Number	:	-

EPA Pesticide Chemical Code 016001

SPECIFICATIONS

Melting Point: N/A

Boiling point: N/A

STATUS IN FOOD AND DRUG LAWS

CoE limits:

Beverages (mg/kg)	Food (mg/kg)	Exceptions (mg/kg)
-	-	-

Acceptable Daily Intake:

ADI (mg/kg)	ADI Set by	Date Set	Comments
Not limited	JECFA	1987	-

FDA Status: [CFR21]

Section Number	Comments
173.25	Ion exchange resins

HUMAN EXPOSURE

Natural Occurrence: A non-metallic element with atomic symbol C, atomic number 6, and atomic weight 12.011. It may occur as several different allotropes including diamond; charcoal; and graphite; and as soot from incompletely burned fuel [ToxNet, 2009].

Reported Uses: Typically used as a filter in purification systems for air water gas, chemicals, used to refine vodka and whisky. It is also used to treat acute poisonings [AACT 1999].

TOXICITY DATA

This ingredient has been registered under REACH. Under REACH, registrants have an obligation to provide information on substances they manufacture or import. This information includes data on hazardous properties (covering various toxicological endpoints), guidance on safe use and classification and labelling. The European Chemicals Agency (ECHA) makes this information publicly available on its website: http://echa.europa.eu/.

Activated carbon is a micro porous inert carbon with a large internal surface (up to 1500 m^2/g). On this surface organic molecules from liquids or gases can adsorb. Adsorption is the natural phenomenon in which molecules from the gas or liquid phase are attached to the surface of the solid. Carbon materials are activated by a series of processes which include: *(1)* removal of all water (dehydration), *(2)* conversion of the organic matter to elemental carbon, driving off the noncarbon portion (carbonisation) and *(3)* burning off tars and pore enlargement (activation) [DESOTEC, 2009].

The structure developed is a function of the carbonisation and activation temperatures. In terms of pore structure, the adsorbent pores can be divided into three basic classes: *(1)* macropores (> 1000Å), *(2)* transitional or mesopores and *(3)* micropores (<10Å). The macropores do not add appreciably to the surface area of the carbon, but provide a passageway to the

particle interior and the micropores. The micropores are developed primarily during carbon activation and result in the large surface area for adsorption to oc cur [DESOTEC, 2009].

Activated carbon contains: *(a)* bulk atoms that are neutral, *(b)* surface atoms that are the real 'adsorption' atoms and *(c)* corner atoms that are very reactive and even react with metals [DESOTEC, 2009].

In Vivo Toxicity Status

Organism	Test Type	Route	Reported Dosage
Dog	LD	Intraperitoneal	> 5gm/kg (5000mg/kg)
Dog	LD	Oral	> 5gm/kg (5000mg/kg)
Dog	LD	Subcutaneous	> 5gm/kg (5000mg/kg)
Mouse	LD	Intraperitoneal	> 5gm/kg (5000mg/kg)
Mouse	LD	Oral	> 5gm/kg (5000mg/kg)
Mouse	LD	Subcutaneous	> 5gm/kg (5000mg/kg)
Mouse	LD_{50}	Intravenous	440mg/kg (440mg/kg)
Rat	LD	Intraperitoneal	> 5gm/kg (5000mg/kg)
Rat	LD	Oral	> 5gm/kg (5000mg/kg)
Rat	LD	Subcutaneous	> 5gm/kg (5000mg/kg)

[ToxNet, 2009]

Groups of 20 one-day old chicks received a diet containing 0% or 2% charcoal for 34 days. No adverse effects other than those due to physical adsorption on to the charcoal of the essential nutrients vitamin A and K were seen. These effects could be reversed by additional administration of these factors [JECFA 1970].

Groups of 10-30, mostly male, occasionally female mice of either CFW (white) or C3H (brown) strain were fed for periods of 12-18 months on diets containing 0% or 10% activated vegetable carbon. Diets had either a water or oil emulsion base. Controls received 15% flour using similar bases in their diet. No significant differences from controls were reported. Similar groups of male mice received 10% of benzene -extracted carbon black for 53-76 wee ks. A number of tumours appeared irregularly and unrelated to duration of treatment or dose, probably due to contamin ation with carcinogenic extract [JECFA 1970].

Charcoal (0.27g) suspended in cottonseed oil was injected subcutaneously into C3H Mice. After 16 months no gross or histopathological changes were observed related to the injection. Cottonseed oil, used to extract activated charcoal for either 30 or 90 days, was injected subcutaneously into mice. After 20 months no significant gross or histopathological changes were seen. Benzene extract (0.05-5 mg) suspended in cottonseed oil was injected subcutaneously into mice. After 13 months the only abnormal finding was a significant incidence of glomerulonephritis in treated animals as compared with controls [JECFA 1970].

Carcinogenicity / Mutagenicity

A three part study by Anisinov *et al.,* (1998, 1999a, 1999b) investigated the role of dietary activated carbon fibre Aqualen on tumourigeneic endpoints in rodents. The authors found that Aqualen (0.1-1g/kg daily dose) inhibited the development of N-methyl-N'-nitro-N-nitrosoguanidine-induced stomach cancer (12 month exposure , 16 month study duration) and of 1,2-dimethyl hydrazine-induced intestinal cancer (6 month exposure) in female LIO rats. In female SHR mice dietary Aqualen (0.1g/kh daily dose) had no effect on overall tumour incidence but did significantly increase their life-span by 4 months in mice with all tumours and by 5 months in mice with malignant tumours. The authors further showed that Aqualen itself was non-carcinogenic in mice [Anisinov *et al.,* (1998), Anisinov *et al.,* (1999a), Anisinov *et al.,* (1999b)]

Overall, seven studies were considered to be informative for lung cancer of carbon black, of which three were among carbon black production workers. The Working Group of IARC considered the studies of carbon black production workers in the United Kingdom, Germany and the USA to be the most informative for assessing cancer risk. The two studies from the United Kingdom and Germany indicated an excess risk compared with external references. Confounding by smoking could not be excluded, although it was considered unlikely to have explained the entire excess risk. However, in both cohorts, internal analyses by level of exposure to carbon black gave equivocal but mainly null results. The study of carbon black workers in the USA suggested no excess mortality, but did not assess risk by level of exposure. In studies that assessed risks for lung cancer among user industries, the most informative study of German rubber workers showed some indication of excess risk that disappeared when asbestos and talc were adjusted for in the analysis. Of the remaining studies, two others showed non-significant excesses (US formaldehyde cohort and the Canadian community-based case–control study) and one showed no excess risk for lung cancer linked to the handling of carbon black (Italian dockworkers). For cancers of the urinary bladder, kidney, stomach and oesophagus, isolated results indicate excess risks, but these are not sufficient to support an evaluation of human carcinogenicity. There was no evidence of an effect of carbon black for other cancer sites [IARC, 2006].

Dermal Toxicity

A total of 2.9 g carbon black (suspended in mineral oil) or 3.2 g (suspended in cottonseed oil) were brushed three times a week on to the back of mice for 1 year. Animals were killed after 1 year or 16-17 months. No significant gross or histopathological changes were seen compared with controls (Nau et al., 1963). Suspensions of activated vegetable carbon in water or oil (20%) were painted in another experiment three times per week on to the back of groups of 10-20 CFW or C3H mice. After 12-17 months no abnormalities were seen in test as compared with control groups. Similar results were obtained when benzene-extracted carbon black was used [JECFA, 1970].

Inhalation Toxicity

Test groups of 60 guinea-pigs, 30 rats and 131 mice, with 22 guinea-pigs, 15 rats and 20 mice as controls were exposed to activated vegetable carbon dust for 7 hours per day, 5 days per week, for 1 year. No significant effects were noted on mortality. At autopsy the lungs showed evidence of multifocal dust deposition. Histopatholo gy showed dust deposits in alveoli with focal atelectasis and adjacent alveolar overexpansion as well as interstitial pneumonitis. Mice showed the least changes but rats had areas of lipid pneumonia. These findings were consistent with inert dust reaction [JECFA 1970].

A short report by Uragoda (1989) showed that, in 66 activated carbon workers (working on average 7.2 years) in Sri Lanka, there was no increase in radiological evidence of pneumoconiosis compared to a control group.

Wehr *et al.,* (1975) inve stigated the incidence of respiratory effects on 397 workers at an activated carbon plant. Radiographic findings of p-type, rounded opacities without fibrosis or coalescence indicated presence of pneumoconiosis. Pneumoconiosis was present in 9.6% of men an d was related to cumulative dust exposure. The authors also stated that 'lesser degrees' of radiographic abnormalities were suggestive of pneumoconiosis and had incidences of 11% in men and 2% in women . Lung spirometry showed that cumulative dust exposure was not found to have an effect on pulmonary function. Biopsies of two individuals revealed 'extensive carbon deposition but minimal associated fibrosis' [Wehr *et al.,* 1975].

A workshop consensus report on 'The relevance of rat lung response to particle o verload for human risk assessment ' gave guidance on how to perform risk assessment of poorly soluble particles (PSPs) (i.e. carbon black, coal dust etc). However in the introduction literature was reviewed and suggested that PSPs 'elicit tumours in rats wh en deposition overwhelms the clearance mechanisms of the lung '. However the authors also state that this tumour induction is not observed in mice and hamsters, and what data there is available in humans is 'consistently negative' [ILSI, 2000].

There are m any studies on the deposition and retention kinetics of inhaled carbon particles following intratracheal instillation or inhalation in rodents. In general, all rodent species investigated show evidence of rapid clearance of inhaled carbon particles when ex posure concentrations did not result in impaired clearance resulting in accumulation of particles in the lung (i.e. lung overload). The experimental studies of ultrafine particles of carbon black have shown that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance, which occurs at lower mass doses of ultrafine particles than with larger particles. Overloading has been observed in rats, mice and hamsters exposed to carbon black. Hamsters appear to exhibit the most efficient clearance of carbon black particles compared with rats and mice. Adverse lung responses to inhaled carbon black (pulmonary inflammation and epithelial injury) increase significantly with increasing retained lung dose of carbon black particles. Fine and ult rafine carbon black particles can translocate beyond the lungs to other organs [IARC 2006].

A number of toxic effects of carbon black have been reported in various rodent species. The toxic effects reported are dose-dependent and include inflammation, lung epithelial cell injury and lung lesions that are more severe and prolonged in rats than in mice and hamsters. Exposure to carbon black particles modulates the immune system. In-vitro studies show evidence that carbon black particles can generate reactive oxygen species in cell-free systems, increase the production of tumour necrosis factor- α and activate serum factors such as complement [IARC 2006].

The deposition pattern of carbon black particles depends on the particle size (aerodynamic or thermodynamic) and on the anatomical and physiological characteristics of the host. The deposition fraction of carbon black influences the dose to a given region of the respiratory tract. Several studies describe the retention of carbon black in the respiratory tract of exposed workers, as well as the health effects of these exposures. For example, lung tissues from workers in carbon black factories contain deposits of carbon black. Lung diseases or conditions may influence the deposition and retention of particles such as carbon black. For instance, asthmatics had a higher total deposition of ultrafine carbon particles in the respiratory tract compared with healthy individuals. The amount of carbon particles deposited can also increase with increasing minute ventilation, for instance in individuals taking exercise or during heavy physical labour. High retained mass lung burdens and decreased lung clearance have been observed in coal miners [IARC 2006].

Other Relevant Studies

Activated carbon has also been used as a delivery agent for anti-cancer therapeutics [Nakase *et al.,* 2004, Hagiwara *et al.,* 1987] and for the removal of pharmacological agents in overdoses [Raper *et al.,* 1982]. However intraperitoneal instillation of activated carbon in rats was shown to cause increased adhesion formation and reduced the fibrinolytic activity of mesothelial cells [Jansen *et al.,* 2000].

Another use of carbon is in the formation of nano materials, carbon single walled nanotubes are typically constructed from graphite sheets rolled in to tubes in the order of 1nm in diameter and 100 nm in length. Tsuji *et al.,* (2006) summarised a series of studies on nanoparticles which indicate that, if inhaled into the lungs, nanotubes (depending on their content) are capable of eliciting an inflammatory, granulomatous, and fibrogenic response, and that the mass-based permissible exposure level (PEL) for respirable graphite dust may be inadequately protective for exposure to single walled nanano tubes (SWNTs). If a 30-g mouse were exposed to airborne nanotubes at a concentration of 5 mg/m3, the PEL for respirable graphite dust, and 40% of the respired nanotubes deposited in the pulmonary region, the lungs would accumulate a mass of nanotubes equivalent to the low dose within 4 working days and a mass equivalent to the high dose within 17 working days. Moreover, because SWNTs were more toxic than quartz based on histopathology, assuming similar relative toxicity in humans, a PEL below that for quartz dust (0.05 mg/m3) is suggested until further characterization of nanotube toxicity, assessment of the fraction of airborne nanotubes in the respirable size range, and determination of whether a mass based standard is even applicable given

evidence for a surface area based standard (Maynard and Zimmer, 2002 as cited in Tsuji *et al.,* (2006)). Additional questions raised by the authors include the relevance for human health assessments to include under what conditions are the nanotubes respirable; how quickly are inhaled nanotubes cleared from the lungs and what is their fate; what are the long-term tissue responses to persisting nanotubes; and how do shape (e.g., varying tube lengths) and metal impurities such as Ni and Fe influence toxicity [Tsuji *et al.,* 2006].

Behavioural Data

No data identified

***In Vitro* Toxicity Status**

Carcinogenicity / Mutagenicity

The genotoxicity of carbon black has been evaluated in various in-vivo and in-vitro systems. In rats exposed to carbon black by inhalation for 13 weeks, the Hprt mutant frequency and the level of pro-mutagenic 8-oxo-deoxyguanosine adducts were elevated in lung e pithelial cells. In vitro, catalase inhibited the increase in Hprt mutation frequency in lung epithelial cells induced by lavage fluid from carbon black-instilled rats, which implies a role for cell-derived oxidants in this response. In two of five studies , carbon black induced an increase in DNA adducts in the peripheral lung tissue of rats after inhalation exposure. Mutations in K-Ras and p53 do not seem to be induced by exposure to carbon black. In separate in-vitro studies with hamster cells, carbon bla ck caused micronucleus induction and cell transformation, but no sister chromatid exchange. The results of most bacterial mutagenicity studies of carbon black were negative [IARC 2006].

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