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Chemtrails And Barium Toxicity

From Dr. Marijah McCain
4-6-2

Toxicity Profiles
Toxicity Summary for BARIUM

NOTE - Although the toxicity values presented in these toxicity profiles were correct at the time they were produced, these values are subject to change. Users should always refer to the Toxicity Value Database for the currect toxicity values.

REFERENCES

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400.

EXECUTIVE SUMMARY

The soluble salts of barium, an alkaline earth metal, are toxic in mammalian systems. They are absorbed rapidly from the gastrointestinal tract and are deposited in the muscles, lungs, and bone. Barium is excreted primarily in the feces.

At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. Acute and subchronic oral doses of barium cause vomiting and diarrhea, followed by decreased heart rate and elevated blood pressure. Higher doses result in cardiac irregularities, weakness, tremors, anxiety, and dyspnea. A drop in serum potassium may account for some of the symptoms. Death can occur from cardiac and respiratory failure. Acute doses around 0.8 grams can be fatal to humans.

Subchronic and chronic oral or inhalation exposure primarily affects the cardiovascular system resulting in elevated blood pressure. A lowest-observed-adverse-effect level (LOAEL) of 0.51 mg barium/kg/day based on increased blood pressure was observed in chronic oral rat studies (Perry et al. 1983), whereas human studies identified a no-observed-adverse-effect level (NOAEL) of 0.21 mg barium/kg/day (Wones et al. 1990, Brenniman and Levy 1984). The human data were used by the EPA to calculate a chronic and subchronic oral reference dose (RfD) of 0.07 mg/kg/day (EPA 1995a,b). In the Wones et al. study, human volunteers were given barium up to 10 mg/L in drinking water for 10 weeks. No clinically significant effects were observed. An epidemiological study was conducted by Brenniman and Levy in which human populations ingesting 2 to 10 mg/L of barium in drinking water were compared to a population ingesting 0 to 0.2 mg/L. No significant individual differences were seen; however, a significantly higher mortality rate from all combined cardiovascular diseases was observed with

the higher barium level in the 65+ age group. The average barium concentration was 7.3 mg/L, which corresponds to a dose of 0.20 mg/kg/day. Confidence in the oral RfD is rated medium by the EPA.

Subchronic and chronic inhalation exposure of human populations to barium-containing dust can result in a benign pneumoconiosis called "baritosis." This condition is often accompanied by an elevated blood pressure but does not result in a change in pulmonary function. Exposure to an air concentration of 5.2 mg barium carbonate/m3 for 4 hours/day for 6 months has been reported to result in elevated blood pressure and decreased body weight gain in rats (Tarasenko et al. 1977). Reproduction and developmental effects were also observed. Increased fetal mortality was seen after untreated females were mated with males exposed to 5.2 mg/m3 of barium carbonate. Similar results were obtained with female rats treated with 13.4 mg barium carbonate/m3. The NOAEL for developmental effects was 1.15 mg/m3 (equivalent to 0.8 mg barium/m3). An inhalation reference concentration (RfC) of 0.005 mg/m3 for subchronic and 0.0005 mg/m3 for chronic exposure was calculated by the EPA based on the NOAEL for developmental effects (EPA 1995a). These effects have not been substantiated in humans or other animal systems.

Barium has not been evaluated by the EPA for evidence of human carcinogenic potential (EPA 1995b).

1. INTRODUCTION

Barium (CAS registry number 7440-39-3) is a divalent alkaline-earth metal found only in combination with other elements in nature. The most important of these combinations are the peroxide, chloride, sulfate, carbonate, nitrate, and chlorate. The pure metal oxidizes readily and reacts with water emitting hydrogen; it is chemically similar to calcium (Weast et al. 1987). The most likely source of barium in the atmosphere is from industrial emissions. Since it is usually present as a particulate form, it can be removed from the atmosphere by wet precipitation and deposition. Due to the element's tendency to form salts with limited solubility in soil and water, it is expected to have a residence time of hundreds of years and is not expected to be very mobile. Acidic conditions, however, will increase the solubility of some barium compounds facilitating their movement from the soil to the groundwater (EPA 1984). Trace amounts of barium were found in more than 99% of the surface waters and finished drinking water samples (average values of 43 g/L, and 28.6 g/L, respectively) across the United States (National Academy of Sciences 1977).

2. METABOLISM AND DISPOSITION

2.1 ABSORPTION

The soluble forms of barium salts are rapidly absorbed into the blood from the intestinal tract. The rates of absorption of a number of barium salts have been measured in rats following oral exposure to small quantities (30 mg/kg body weight). The relative absorption rates were found to be: barium chloride barium sulfate barium carbonate. Large doses of barium sulfate do not increase the uptake of this salt because of its low solubility (McCauley and Washington 1983, EPA 1984).

Systemic toxic effects have been observed following both oral and inhalation exposure. No absorption kinetics are available following inhalation exposure, although it is obvious that absorption does occur (EPA 1984).

2.2 DISTRIBUTION

Barium absorbed into the bloodstream disappears in about 24 hours; however, it is deposited in the muscles, lungs, and bone. Very little is stored in the kidneys, liver, spleen, brain, heart, or hair. It remains in the muscles about 30 hours after which the concentration decreases slowly. The deposition of barium into bone is similar to calcium but occurs at a faster rate (Beliles 1994). The half life of barium in bone is estimated to be about 50 days (Machata 1988).

2.3 METABOLISM

About 54% of the barium dose is protein bound. Barium is known to activate the secretion of catecholamines from the adrenal medulla without prior calcium deprivation. It may displace calcium from the cell membranes, thereby increasing permeability and providing stimulation to muscles. Eventual paralysis of the central nervous system can occur (Beliles 1994).

2.4 EXCRETION

A tracer study in rats using 140Ba demonstrated that 7% and 20% of the barium dose was excreted in 24 hours in the urine and feces, respectively. In contrast, calcium is primarily excreted in the urine. The clearance of barium is enhanced with saline infusion (Beliles 1994). Following intravenous injection of barium into six healthy men, excretion was mainly fecal with the total relative fecal:urinary clearance for 14 days ranging from 6 to 15 (Newton et al. 1991).

3. NONCARCINOGENIC HEALTH EFFECTS

3.1 ORAL EXPOSURES

3.1.1 Acute Toxicity

3.1.1.1 Human

A number of accidental barium poisonings have occurred following the ingestion of barium salts. The estimated fatal dose of barium carbonate, a rodenticide, is about 5 grams for a 70 kg human (Arena 1979). The LD50 for barium chloride is estimated at about 1 gram for a 70 kg human (Machata 1988), and the LDLo (lowest published lethal dose) is reported to be about 0.8 grams (Lewis and Sweet 1984). The acute symptoms include excess salivation, vomiting, diarrhea, increased blood pressure, muscular tremors, weakness, paresis, anxiety, dyspnea, and cardiac irregularities. A severe loss of potassium can account for some of the symptoms. Convulsions and death from cardiac and respiratory failure can occur. Magnesium and sodium sulfate are antidotal if taken soon after ingestion since either salt will result in the formation of insoluble barium sulfate and prevent further absorption. Survival for more than 24 hours is usually followed by complete recovery (Arena 1979).

Complications occurred in a woman following a barium swallow investigation for severe dysphagia. Direct aspiration of a large amount of barium into the right main bronchus resulted in tachycardia, tachypnoea, fever, and an oxygen saturation of 82%; two weeks later the woman still had a moist cough with widespread rales but continued to recover (Penington 1993).

A family was accidentally poisoned with barium from eating their evening meal. The mother had fried fish breaded with a flour-like substance that turned out to be rat poison containing barium carbonate. All seven family members, aged 2 to 48 years, developed nausea, vomiting, diarrhea, and crampy abdominal pain within minutes of consuming the meal; the parents also developed ventricular tachycardia, flaccid paralysis of the extremities, shortness of breath (mother), and respiratory failure (father). Patients were treated symptomatically and all fully recovered (Johnson and VanTassell 1991).

3.1.1.2 Animal

Similar acute symptoms occur in animals; however, higher doses are usually involved. The LD50 for rats is listed as 630 mg/kg for barium carbonate, 118 mg/kg for barium chloride, and 921 mg/kg for barium acetate (Lewis and Sweet 1984).

3.1.2 Subchronic Toxicity

3.1.2.1 Human

An experiment testing the subchronic toxicity of barium chloride on human volunteers was conducted by Wones et al. (1990). The diets of 11 male subjects were controlled. They were given 1.5 L/day of distilled and charcoal-filtered drinking water that contained 0 mg/L barium for weeks 1 and 2, 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. No clinically significant effects were observed in blood pressures, serum chemistry, urinalysis, or electrocardiograms. The 10 mg/L (0.21 mg/kg/day) dose was identified as a NOAEL.

3.1.2.2 Animal

Groups of 30 male and 30 female Charles River rats were exposed to barium chloride at 0, 10, 50, or 250 ppm in drinking water for 90 days (Tardiff et al. 1980). The highest average dose in this study was calculated to be 45.7 mg/kg/day for female rats. No significant clinical signs of toxicity were observed. Blood pressure was not measured.

McCauley et al. (1985) conducted drinking water studies in which six male Sprague-Dawley rats/group were given water containing 0, 10, 100, or 250 mg/L barium for 36 weeks, or 1, 10, 100, or 1000 mg/L barium for 16 weeks. Female rats were given 0 or 250 mg/L for 46 weeks. Animals receiving the 1000 mg/L dose developed ultrastructural changes in the kidney glomeruli. No other effects were reported.

Tardiff et al. (1980) exposed groups of 30 male and 30 female Charles River rats to 0, 10, 50, or 250 ppm barium (given as barium chloride) in drinking water for 90 days. A slight reduction in adrenal weights was seen in female rats with the 250 ppm (45.7 mg/kg/day) dose at 13 weeks, and no other adverse effects were observed in male rats with the 50 ppm (8.1 mg/kg/day) and the 250 ppm (38.1 mg/kg/day) doses at 8 weeks. No clear dose effect or dose duration effect was seen with the adrenal weight decrease; therefore,

the clinical significance is uncertain.

3.1.3 Chronic Toxicity

3.1.3.1 Human

An epidemiology study conducted by Brenniman and Levy (1984) compared a human population ingesting barium levels of 2 to 10 mg/L in their drinking water to a population ingesting 0 to 0.2 mg/L. Although significantly higher mortality rates from all cardiovascular diseases were observed with the higher barium level in the 65 and over age group, there were no significant individual differences in blood pressures, strokes, or heart and renal diseases within the two groups. The average barium concentration for the mortality study was 7.3 mg/L, which corresponds to a dose of 0.20 mg/kg/day assuming drinking water consumption of 2 L/day for a 70 kg human.

3.1.3.2 Animal

A series of experiments were performed in which groups of 52 male and female Long-Evans rats and 42 male and female Swiss mice were exposed to 5 mg barium/L (given as barium acetate) in drinking water for their lifetime (Schroeder and Mitchener 1975a,b). The barium doses were about 0.25 and 0.825 mg/kg/day for rats and mice, respectively. No adverse clinical effects were observed; however, blood pressure was not measured. A slight but significant reduction in longevity of treated male mice was noted when measured as the mean age at death of the last surviving 10% of animals. The overall average life span of the group, however, was about the same as the control group (EPA 1984, 1989).

Perry et al. (1983) exposed 12 to 13 female weanling rats/group to 0, 1, 10, or 100 ppm barium (given as barium chloride) for up to 16 months. Average doses were calculated to be 0, 0.051, 0.51, and 5.1 mg/kg/day (EPA 1985). A clinically significant increase in average blood pressure was observed in the highest dose group; a slight but statistically significant increase was seen in the 10 ppm (0.51 mg/kg/day) dose group. The controlled diet, which restricted the intake of trace metals, calcium, and potassium, may have contributed to the effect.

3.1.4 Developmental and Reproductive Toxicity

Information on developmental and reproductive toxicity in humans or

animals following oral exposure was unavailable.

3.1.5 Reference Dose

3.1.5.1 Subchronic

ORAL RfDs: 0.07 mg/kg/day (EPA 1995a)

UNCERTAINTY FACTOR: 3

NOAEL: 0.21 mg/kg/day

PRINCIPAL STUDIES: The same studies and comments apply to both the subchronic and chronic RfD derivations. See Sect. 3.1.5.2.

3.1.5.2 Chronic

ORAL RfDc: 0.07 mg/kg/day (EPA 1995b)

UNCERTAINTY FACTOR: 3

MODIFYING FACTOR: 1

NOAEL: 0.21 mg/kg/day

CONFIDENCE:

Study: Medium

Data Base: Medium

RfD: Medium

VERIFICATION DATE: 06/21/90

PRINCIPAL STUDIES: Wones et al. (1990); Brenniman and Levy (1984).

COMMENTS: The RfD values are based on a weight-of-evidence approach using subchronic to chronic human drinking water studies. The uncertainty factor accounts for protecting sensitive individuals and is reduced from the usual factor of 10 because the selected studies examined the population judged most at risk.

3.2 INHALATION EXPOSURES

3.2.1 Acute Toxicity

3.2.1.1 Human

Barium carbonate dust has been reported to be a bronchial irritant. Barium oxide dust is considered a dermal and nasal irritant (Beliles 1994). The effect of barium dusts on welders was investigated under simulated working conditions over a one-week time period (Zschiesche et al. 1992). Barium fume concentrations were 4.4 and 2.0 mg/m3 during welding with stick electrodes and flux cored wires, respectively. No adverse health effects on the welders were attributable to barium exposure, but there was a slight decrease in plasma potassium levels at the end of the work shift.

3.2.1.2 Animal

Information on the acute inhalation toxicity of barium in animals was not available.

3.2.2 Subchronic Toxicity

3.2.2.1 Human

Industrial workers exposed to barium dust, usually in the form of barium sulfate or carbonate, often develop a benign pneumoconiosis referred to as "baritosis." Because of the radiopacity of barium compounds, this condition can be specifically diagnosed radiologically. After removal from the sources of exposure, baritosis is reversible in most cases. Baritosis results in a significantly higher incidence of hypertension, but no changes are usually seen in pulmonary function (Stokinger 1981, EPA 1995b).

3.2.2.2 Animal

Male rats were exposed to 1.15 and 5.2 mg/m3 of barium carbonate dust for 4 hours/day for 6 months. The high dose animals developed increased arterial pressure; decreased body weight gain; decreased blood levels of hemoglobin, sugar, protein, cholinesterase and thrombocytes; increased

blood levels of leukocytes, phosphorous and alkaline phosphatase; increased urine calcium; and perivascular and peribronchial sclerosis in the lungs. (EPA 1984, Tarasenko et al. 1977).

3.2.3 Chronic Toxicity

3.2.3.1 Human

Baritosis and bronchial irritation have been reported in workers chronically exposed to barium containing dust (Beliles 1994).

3.2.3.2 Animal

Information on the chronic inhalation toxicity of barium in animals was not available.

3.2.4 Developmental and Reproductive Toxicity

Tarasenko et al. (1977) performed a series of experiments in rats designed to test for possible reproductive and developmental effects. Increased fetal mortality was observed following the mating of males exposed to barium carbonate (5.2 mg/m3 air) with untreated females. Decreased sperm motility was observed in males treated with 22.6 mg/m3. The mating of females exposed to 13.4 mg/m3 for 4 months also resulted in increased fetal mortality and a general under development of the newborn pups. Ovarian follicle atresia was seen in female rats exposed to 3.1 mg/m3. No significant adverse effects were noted with the 1.15 mg/m3 concentration (EPA 1984).

3.2.5 Reference Concentration/Dose

3.2.5.1 Subchronic

INHALATION RfCs: 0.005 mg/m3; 0.001 mg/kg/day (EPA 1995a)

UNCERTAINTY FACTOR: 100

NOEL: 0.8 mg Ba/m3 given 4 hr/day (EPA 1995a)

PRINCIPAL STUDY: The same study and comments apply to the subchronic

and chronic RfC. The study is described in Sect. 3.2.4.

3.2.5.2 Chronic

INHALATION RfCc: 0.0005 mg/m3; 0.0001 mg/kg/day (EPA 1995a)

UNCERTAINTY FACTOR: 1000

NOEL: 0.8 mg Ba/m3 given 4 hr/day (EPA 1995a)

PRINCIPAL STUDY: Tarasenko et al. 1977

COMMENTS: The dose of 1.15 mg $BaCO_3$/m3 was given as the NOEL in the principal study, which is equivalent to 0.8 mg barium/m3 used as the basis for the RfC calculations. An inhalation risk assessment for barium is under review by an EPA work group (EPA, 1995b).

3.3 OTHER ROUTES OF EXPOSURE

3.3.1 Acute Toxicity

3.3.1.1 Humans

Barium oxide dust is considered to be a dermal and nasal irritant (Beliles 1994).

3.3.1.2 Animal

A number of experiments have used intravenous and subcutaneous injections to measure lethal levels of soluble barium compounds. LD50 values for barium chloride, nitrate, and acetate were determined in two strains of mice by intravenous injection (Syed and Hosain 1972). The affected animals either died within one hour or survived the treatment. The LD50 values obtained were 8.12, 8.49, and 11.32 mg barium/kg for the chloride, nitrate, and acetate, respectively, in Swiss-Webster mice, and 19.20, 20.10, and 23.31 mg barium/kg for the chloride, nitrate, and acetate, respectively, in ICR mice. Although the relative toxicity of the barium salts remained the same, there was an unexplained two-fold difference in the LD50 values between the two mice strains.

The LDLo has been determined by subcutaneous injection in mice to be 10 mg/kg for the barium nitrate and chloride salts. The LDLo in rabbits was 55 mg/kg for the chloride and 96 mg/kg for the acetate salts. The LDLo values vary widely with the route and test animal. For example, with barium chloride the LDLo value for oral administration to rabbits is 170 mg/kg, whereas the value for subcutaneous injection is 55 mg/kg. Subcutaneous injection in mice results in a value of 10 mg/kg, which is higher than the LD50 value for intravenous injection in the Swiss-Webster strain (Lewis and Sweet 1984).

3.3.2 Subchronic Toxicity

Information on the subchronic toxicity of barium in humans and animals was not available.

3.3.3 Chronic Toxicity

Information on the chronic toxicity of barium in humans and animals was not available.

3.3.4 Developmental and Reproductive Toxicity

Information on the developmental and reproductive toxicity of barium in humans and animals was not available.

3.4 TARGET ORGANS/CRITICAL EFFECTS

3.4.1 Oral Exposures

3.4.1.1 Primary Target Organs

1. Cardiovascular system: Subchronic to chronic symptoms include increased blood pressure and increased incidence of cardiovascular disease in humans. An acute overdose can result in cardiac irregularities. Convulsions and death from cardiac and respiratory failure can occur.

2. Nervous system: Acute to subchronic symptoms include weakness, tremors, anxiety, and dyspnea. An acute overdose can result in convulsions and death from cardiac and respiratory failure.

3.4.1.2 Other targets

Gastrointestinal system: Acute to subchronic symptoms include excess salivation, vomiting, and diarrhea in humans.

3.4.2 Inhalation Exposures

3.4.2.1 Primary target(s)

1. Cardiovascular system: Symptoms include increased blood pressure in humans.

2. Reproduction and development: Subchronic exposure of rats resulted in decreased sperm motility and ovarian follicle atresia. Increased fetal mortality and underdevelopment of newborn pups were also reported.

3.4.2.2 Other target(s)

Lungs: Subchronic to chronic exposure in humans results in a pneumoconiosis known as "baritosis" that usually does not adversely affect pulmonary function.

4. CARCINOGENICITY

4.1 ORAL EXPOSURES

4.1.1 Human

Information on the carcinogenicity of barium in humans was not available.

4.1.2 Animal

No significant differences in tumor incidence were found in either rats or mice in the lifetime exposure experiments of Schroeder and Mitchener (1975a,b), as described in Sect. 3.1.3.2.

4.2 INHALATION EXPOSURES

Information on the carcinogenicity of barium in humans and animals was not available.

4.3 OTHER ROUTES OF EXPOSURE

Information on the carcinogenicity of barium in humans and animals was not available.

4.4 EPA WEIGHT-OF-EVIDENCE

Barium has not been evaluated by the EPA for evidence of human carcinogenic potential (EPA 1995b).

4.5 CARCINOGENICITY SLOPE FACTORS

Data are insufficient to calculate a slope factor for barium.

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Last Updated 10/07/97

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