SIDS INITIAL ASSESSMENT PROFILE

CAS No.	1333-82-0; 7775-11-3; 10588-01-9; 7789-09-5; 7778-50-9
Chemical Name	Chromium trioxide; sodium chromate; sodium dichromate; ammonium dichromate; potassium dichromate
Structural Formula	CrO_3; $NaCrO_4$; $Na_2Cr_2O_7$; $NH_4 Cr_2O_7$; $K_2 Cr_2O_7$

RECOMMENDATIONS

These chemicals are candidates for further work.

SUMMARY CONCLUSIONS OF THE SIAR

These five chromium (VI) substances have been assessed as a group, since after release to the environment the chromium species produced are the same from each substance, and so the fate and effects in the environment can be considered together. Similarly for human health, the species produced will behave similarly in biological tissues and so the effects can be treated as a group. (There is also an additional concern about the acidity of solutions of chromium trioxide.)

Human Health

The toxicological database for chromium (VI) (Cr(VI)) is generally extensive. Sodium chromate, dichromates of sodium, potassium and ammonium, and chromium (VI) trioxide, the substances covered in this review are all highly water-soluble hexavalent compounds.

Chromium (VI) trioxide in solution produces chromic acid, concentrated solutions that are highly acidic. Hence, of the five Cr(VI) compounds covered by the assessment, there are site-of-contact issues related to low pH that are a consideration for chromium (VI) trioxide but not for the other four.

Beyond this, the five Cr(VI) compounds will all readily dissolve in aqueous environments in the body, to release chromate (CrO_4^{2-}) or dichromate ($Cr_2O_7^{2-}$) ions. These two ions will co-exist, in equilibrium, regardless of the particular Cr(VI) compound involved. The chromate/dichromate ions produced from all five compounds will behave similarly in biological tissues and hence, other than the additional property of acidity and its potential influence on toxicity for chromium (VI) trioxide, the five can be treated as a common group. Furthermore, toxicological observations made with other chromium (VI) compounds that can similarly readily dissociate to produce chromate/dichromate ions in solution can be legitimately made use of in predicting the toxicity of these five compounds.

There is a reasonably good database available on the toxicokinetics of the chromium (VI) compounds under review, although there are relatively few human data. The available data indicate that generally the chromium (VI) compounds covered by this document are likely to behave in a similar manner in respect of toxicokinetics, and that the kinetic behaviour of these substances would be similar in those species studied, including humans.

Following inhalation exposure, animal studies have shown that 20-30% of the administered Cr(VI) is absorbed via the respiratory tract. Highly water-soluble Cr(VI) is poorly absorbed via the gastrointestinal tract (only 2-9% of the

dose was absorbed in human studies) due to reduction to the relatively poorly absorbed Cr(III). Only limited dermal absorption takes place through intact skin, with 1-4% Cr(VI) from an aqueous solution crossing the skin in guinea pig studies.

According to results of animal testing, chromium derived from these compounds can remain in the lungs for several weeks after inhalation exposure and also becomes bound to haemoglobin in erythrocytes for the lifespan of the cells. Cr(VI) becomes reduced to Cr(III) after entering the body due to the influence of reducing agents, for example glutathione. Distribution is widespread even after a single dose and includes transfer of absorbed Cr(VI) across the placenta. Excretion occurs in urine and faeces. Repeated exposure leads to accumulation of chromium in several tissues, particularly the spleen because of uptake of senescent erythrocytes.

Case reports show that inhalation by workers of aqueous solutions of Cr(VI) mists have resulted in irritation and inflammation of the respiratory tract, with symptoms and signs including dyspnoea and cyanosis; associated airborne levels were not reported. Accidental or deliberate oral ingestion has resulted in signs and symptoms some of which are indicative of corrosive damage and deaths have been reported in numerous cases in adults. Among the survivors, clinical manifestations of liver and kidney damage were present. There have also been cases of kidney damage and death following dermal exposure to Cr(VI). In most of these cases the skin was broken or damaged by the acidity or high temperature of the solution, facilitating Cr(VI) absorption across the skin.

The qualitative picture of acute toxicity seen in humans is supported by observations from studies in experimental animals.

Aerosols were toxic when inhaled by rats. LC_{50} values of 99 mg/m^3 (potassium dichromate) (35 mg Cr(VI)/m^3), 200 mg/m^3 (sodium and potassium dichromate) (70 mg Cr(VI)/m^3), 200 mg/m^3 (ammonium dichromate) (83 mg Cr(VI)/m^3) and 104 mg/m^3 (sodium chromate) (33 mg Cr(VI)/m^3) have been reported for male rats with a 4-hour exposure period. Similarly, an LC_{50} value of 217 mg/m^3 (113 mg Cr(VI)/m^3) for chromium (VI) trioxide has been reported for rats with a 4-hour exposure period. It is predicted that severe damage to tissues of the respiratory tract would occur at low concentrations due to the corrosive nature of this substance.

Available oral LD_{50} values for chromium (VI) trioxide were 52-113 mg/kg (27-59 mg Cr(VI)/kg) in rats and 135-175 mg/kg (70-91 mg Cr(VI)/kg) in mice. Aqueous chromium (VI) trioxide produced bleeding and ulceration of the stomach due to its corrosive properties. Oral LD_{50} values of 74 mg/kg (26 mg Cr(VI)/kg) (potassium dichromate), 59 mg/mg (23 mg Cr(VI)/kg) sodium dichromate), 55 mg/kg (23 mg Cr(VI)/kg) (ammonium dichromate) and 87 mg/kg (28 mg Cr(VI)/kg) (sodium chromate) have been reported for male rats. Female rats were more sensitive with LD_{50} values of 48 mg/kg (17 mg Cr(VI)/kg), 46 mg/kg (16 mg Cr(VI)/kg), 48 mg/kg (20 mg Cr(VI)/kg) and 40 mg/kg (13 mg Cr(VI)/kg) respectively. Toxic effects reported at necropsy included pulmonary congestion and corrosion of mucosa in the gastrointestinal tract.

Highly water-soluble Cr(VI) compounds were also toxic following skin application. In a standard dermal LD_{50} study in rabbit, the following values were determined: sodium dichromate 960 mg/kg (380 mg Cr(VI)/kg); potassium dichromate 1150 mg/kg (410 mg Cr(VI)/kg); ammonium dichromate 1860 mg/kg (770 mg Cr(VI)/kg) and sodium chromate 1330 mg/kg (430 mg Cr(VI)/kg). In another study, percutaneous doses of 207 mg/kg sodium chromate (66 mg Cr(VI)/kg) and 170 mg/kg sodium dichromate (66 mg Cr(VI)/kg) produced death in guinea pigs. A dermal LD_{50} value of 57 mg/kg (30 mg Cr(VI)/kg) has been reported for chromium (VI) trioxide.

In conclusion, highly water-soluble Cr(VI) compounds are very toxic by inhalation and toxic by ingestion. The respiratory tract and the kidney are damaged by these compounds following inhalation and oral exposure respectively. Although acutely harmful or toxic by the dermal route, more severe responses may be observed due to greater uptake via the skin if there is any prior or simultaneous damage to the skin. Depending upon the pH of the Cr(VI) solution, corrosive effects can occur on contact.

Single application of highly water-soluble chromium (VI) in solution to undamaged human skin resulted in only a mild irritant response around the hair follicles. Aqueous chromium (VI) trioxide is a corrosive substance due to its low pH. In addition, when high temperature solutions of Cr(VI) are splashed onto the skin, serious burns occur. Animal data are consistent with the observations made in humans. It is not possible to determine a clear

concentration-response relationship for human skin irritation from the single-exposure animal or occupational data available. Highly water-soluble chromium (VI) compounds can cause very severe skin effects under certain conditions. In workers repeatedly exposed to highly water-soluble chromium (VI), where there is some slight initial damage to the skin, ulcers can develop which constitute a serious and persistent effect. Again, animal data are consistent with the observations made in humans. It is not possible to determine a clear concentration-response relationship for repeated-exposure human skin effects from the occupational data available and quantitative data could be misleading given the potential for severe effects resulting from repeated contamination of slightly damaged skin. Overall, highly water-soluble chromium (VI) compounds should be regarded as corrosive.

Significant damage to the eye can occur upon accidental exposure to highly water-soluble chromium (VI) compounds. Severe and persistent effects occur when there is contact with the low pH aqueous chromium (VI) trioxide or Cr(VI) solutions at high temperature. Repeated, but not single administration of highly water-soluble chromium (VI) caused severe irritation in the rabbit eye. It is not possible to determine a clear concentration-response relationship from the data available.

Symptoms of sensory irritation of the respiratory tract are known to occur among chrome plating workers exposed to a mist of aqueous chromium (VI) trioxide. Since this is corrosive, such symptoms are to be expected. No quantitative data on such irritation are available from studies of workers. No studies reporting symptoms of sensory irritation are available for the other chromium (VI) compounds. Overall, it is not possible to determine a reliable concentration-response relationship for respiratory tract irritation using the available data.

Skin sensitisation resulting from contact with Cr(VI) is relatively common in humans working with the compounds. This has been demonstrated in patch testing of contact dermatitis patients and in investigations of various occupational groups. In addition, skin sensitisation potential has been clearly demonstrated in standard and modified guinea pig maximisation tests and in the mouse ear swelling test.

Current understanding of the mechanism involved in the sensitisation indicates that Cr(III) is the ultimate hapten. Skin contact with Cr(VI) leads to penetration of Cr(VI) into the skin where it is reduced to Cr(III). There is some evidence for cross-reactivity between Cr(III) and Cr(VI); Cr(VI)-sensitised subjects may also react to Cr(III). Overall, it is not possible to reliably determine a threshold for either induction or challenge in an exposed population using the available data.

The available case reports and evidence from well-conducted bronchial challenge tests, show that inhalation of chromium (VI) compounds can cause occupational asthma. As with skin, Cr(VI)-sensitised subjects may react to Cr(III). It is not possible to determine a no-effect level or exposure-response relationship for induction or elicitation of occupational asthma.

With respect to repeated exposure, a large number of studies are available relating to exposure of workers to highly water-soluble chromium (VI), specifically sodium or potassium chromate/dichromate and chromium (VI) trioxide. The main effects reported are irritant and corrosive responses in relation to inhalation and dermal exposure. These include inflammation in the lower respiratory tract, and nasal septum perforation in the upper respiratory tract. It is not possible to relate these effects to reliable measures of Cr(VI) exposure. Although in principle a threshold dose should be identifiable, in practice the location of such a threshold is not possible from the data available. Some evidence of kidney damage has also been found among chromate production and chromium plating workers. No exposure-response data or no-effect levels are available. However, it appears that the exposure levels at which kidney toxicity occurs overlap with the atmospheric concentrations at which respiratory tract effects have been reported.

Only limited animal repeated dose toxicity information is available. In general, the effects seen are consistent with those found in humans. Although in principle a threshold dose should be identifiable, in practice the location of such a threshold is not possible from the data available. Inhalation of sodium chromate dust for 8 months caused deaths in mice exposed to 0.3-3.7 mg/m^3 (0.1-1.2 mg Cr(VI)/m^3). Rats appeared to be less sensitive (no deaths occurring after 16 months). Concentrations down to 0.07 mg/m^3 (0.025 mg Cr(VI)/m^3) sodium dichromate (aerosol) produced increased alveolar macrophage and spleen lymphocyte activities following a 90-day exposure in the rat. Much of this enhancement was lost at 0.57 mg/m^3 sodium dichromate (0.2 mg Cr(VI)/m^3); this dose inhibited alveolar

macrophage phagocytosis. Repeated chromic acid mist (chromium (VI) trioxide) exposure produced irritant and corrosive effects in the respiratory tract at 3.5 mg/m^3 (1.8 mg Cr(VI)/m^3) and above in an 8-month study. Overall, little useful dose-response information is available.

In the rat, testicular degeneration was observed at a dose level (40 mg/kg/day (14 mg Cr(VI)/kg/day)) which caused a large decrease in body weight gain following gavage administration of sodium dichromate for 90 days. A NOAEL of 20 mg/kg/day (7 mg Cr(VI)/kg/day) was determined for effects on the testis, the only organ examined. Other studies found no significant toxicity following administration of potassium dichromate by the dietary route for 9 weeks. The highest dose levels in these studies were 24 mg/kg/day (8 mg Cr(VI)/kg/day) in the rat and 92 mg/kg/day (32 mg Cr(VI)/kg/day in the mouse.

No repeated dermal studies are available, although these substances are recognised as being corrosive on repeated dermal exposure.

Few studies of genotoxic potential in humans are available. No evidence of genotoxic activity has been found in adequately-conducted studies in circulating lymphocytes from chromium-exposed workers. In contrast, there is a vast array of genotoxicity data *in vitro* and less extensive testing in animals available. The evidence clearly indicates that highly water-soluble chromium (VI) compounds can produce significant mutagenic activity *in vitro* and *in vivo*. The chromium (VI) compounds under consideration are therefore regarded as *in vivo* somatic cell mutagens. In addition, toxicokinetic and dominant lethal data suggest that water-soluble chromium (VI) has the potential to be an *in vivo* germ cell mutagen.

Chrome plating workers exposed to chromium (VI) trioxide in aqueous solution have shown a clear excess in mortality from lung cancer. Therefore chromium (VI) trioxide should be regarded as a human carcinogen by the inhalation route. The excess in lung cancer mortality cannot be related to particular atmospheric Cr(VI) levels in any reliable manner. These chrome plating workers were exposed specifically to a mist of Cr(VI) in aqueous acidic solution, emanating from the surface of the plating bath. The acidic nature of the entity might be a significant contributory factor in the type and onset of lesions and uptake of Cr(VI), precluding direct extrapolation of the human carcinogenic activity of the trioxide to the ammonium, sodium or potassium chromates or dichromates.

With respect to the other chromium (VI) compounds under review, epidemiology data from chromate production, chromium pigment manufacture and other chromium-exposed groups showing clear increases in lung cancers cannot be specifically related to exposure to any of the chromium (VI) compounds under consideration here. However, it is highly probable that Cr(VI) ions in solution were the ultimate carcinogenic entity in these situations. Hence these epidemiological studies raise concerns for the carcinogenic potential of the other four chromium (VI) compounds covered in this review.

Animal carcinogenicity studies have been conducted on only two of the compounds covered in this review. In these studies, sodium dichromate was carcinogenic in rats, causing lung tumour production, when given by repeated long-term inhalation or intratracheal instillation. In rats and mice, inhalation or intrabronchial implantation studies using chromium (VI) trioxide produced 1-2 test group animals with lung tumours where such were mainly absent among corresponding controls. Thus, in animal studies there is some evidence of respiratory tract carcinogenic activity for sodium dichromate and chromium (VI) trioxide. Similar studies in rats using other chromium (VI) compounds (not covered by this review), able to produce Cr(VI) in solution, produced carcinogenicity in the lung. Hence there is good reason from animal studies to be concerned about the carcinogenic potential of all five Cr(VI) compounds covered by this review, in terms of the inhalation route and the respiratory tract as a site of action. Data for the oral and dermal routes and carcinogenicity studies on the other compounds under consideration are not available. Chromium (VI) compounds might be expected to have potential to cause cancer on repeated oral or dermal exposure. In the case of the oral route, any systemic carcinogenic potential could be limited by poor absorption from, and reduction to Cr(III) within the gastrointestinal tract although site of contact activity would remain an issue. Similar considerations apply to the skin.

Overall, therefore, all five chromium (VI) compounds covered by this review are considered to have proven or suspect carcinogenic potential by the inhalation route. From the available information, and taking into account the genotoxic potential of these substances, it is not possible to identify any dose-response relationship or thresholds for

this effect.

Human data relating to effects on reproduction are limited to poorly reported studies of female workers from which no conclusions can be drawn. There are two animal studies available which focus on fertility. Adverse effects were produced in mice receiving potassium dichromate for 12 weeks in drinking water at 333 mg/kg/day (120 mg Cr(VI)/kg/day) and 400 mg/kg/day (140 mg Cr(VI)/kg/day) and above in males and females respectively. A NOAEL of 166 mg/kg/day (60 mg Cr(VI)/kg/day) was identified in males but no NOAEL was found for females as 400 mg/kg/day was the lowest dose level tested. An increase in resorptions following treatment of males and a decrease in implantations in treated females were among the findings in this study. In another study, pregestational oral administration of potassium dichromate in drinking water to female mice produced adverse effects on fertility (reduced number of corpora lutea and increased pre-implantation loss) at 500 ppm (119 mg/kg/day (40 mg Cr(VI)/kg/day)) and above. NOAEL values of 119 mg/kg/day (40 mg Cr(VI)/kg/day) and 63 mg/kg/day (20 mg Cr(VI)/kg/day) can be identified from this study for maternal toxicity and fertility effects respectively. In a third study, also in the mouse, at 86 mg/kg/day (30 mg Cr(VI)/kg/day), the highest dose level tested, there were no effects of treatment on fertility parameters.

Fetotoxicity, including post-implantation losses, has been observed in the mouse following administration of potassium dichromate in drinking water during gestation (days 0-19). Significant developmental effects occurred at the lowest dose level tested, 60 mg/kg/day (20 mg Cr(VI)/kg/day) in the absence of maternal toxicity. Therefore no developmental NOAEL was determined. Qualitatively similar results were obtained in another study in which 350 mg/kg potassium dichromate (125 mg Cr(VI)/kg) was administered for a shorter period, on days 6-14 of gestation. In a pregestational study in female mice, fetotoxic effects were seen starting from the lowest dose level tested, 250 ppm (63 mg/kg/day (20 mg Cr(VI)/kg/day)) potassium dichromate. Significant levels of total chromium were found in treated animals at sacrifice. No NOAEL could be identified for the developmental effects which included post-implantation losses. These fetal effects may possibly be explained by the presence of chromium in the dams after the end of treatment. Overall, highly water-soluble chromium (VI) compounds should be considered to be developmental toxicants in the mouse. These findings can be regarded as relevant to humans.

It is noted that some of the adverse effects on reproduction observed in animal studies may be related to the germ cell mutagenicity of these chromium (VI) compounds (see Mutagenicity section). No reproductive toxicity studies are available using the inhalation or dermal routes of exposure.

Environment

The database on the effects of chromium (VI) ion compounds to aquatic organisms is extensive. Acute toxicity tests are available for algae (range of EC_{50} values 0.13 to 4.6 mg/l), invertebrates ($L(E)C_{50}$ values 0.03 to 35 mg/l), fish (LC_{50} values 18 to 213 mg/l) and amphibians (LC_{50} values 43 to 100 mg/l), all expressed as concentrations of chromium (VI). The acute toxicity of chromium (VI) depends on a number of factors, including pH, water hardness, salinity and temperature. In general the toxicity increases with decreasing pH, water hardness or salinity and with increasing temperature, although there are also studies which appear to show little change in toxicity with changing water properties. Invertebrates appear to be generally more sensitive in acute tests.

There are also a large number of long term studies on aquatic organisms, though far less than the number of acute studies. These show less variation of toxicity with water properties, although the smaller number limits the comparisons that can be made. Valid long-term NOEC values have been identified for 28 species (some derived by combining the results of multiple determinations). The species include representatives of algae, macrophytes, crustaceans, coelenterates, insects, molluscs, fish and amphibians, and the range of NOEC values is 0.0047 mg Cr(VI)/l (for reproduction in the cladoceran *Ceriodaphnia dubia*) to 3.5 mg Cr(VI)/l (for fish). A PNEC of 0.47 µg Cr(VI)/l can be derived using an assessment factor of 10. However, due to the large amount of data, a statistical approach has also been used in the EU. The data were assumed to come from a log normal distribution and the lower 5% value from the distribution at the 50% confidence level (HC_5) was determined as 10.2 µg Cr(VI)/l. To take account of limitations in the database (only one representative of molluscs and insects, and no corroborating mesocosm or field studies) an assessment factor of 3 was applied, giving a PNEC of 3.4 µg Cr(VI)/l for the surface water compartment.

Using the equilibrium partitioning method and different partition coefficients for acidic and neutral-alkaline environments gives sediment PNECs of 1.5 mg Cr(VI)/l for 'acidic' conditions and 0.15 mg Cr(VI)/l for 'alkaline'.

For the terrestrial compartment, long-term toxicity data are available for three trophic levels (plants, earthworms and soil processes/micro-organisms), with plants generally being the most sensitive species group (although a clear NOEC has not been determined for earthworms, the EC_{50} values are generally higher than those found in the plant experiments). The lowest NOEC from a plant growth test is 0.35 mg Cr(VI)/kg dry weight. Applying an assessment factor of 10 to this gives a PNEC in soil of 35 μg Cr(VI)/kg dry weight (31 μg/kg on a wet weight basis).

Soil studies tend to show a rapid reduction of chromium (VI) to chromium (III), and so toxicity data for chromium (III) may be more relevant for effects in the terrestrial environment. For earthworms, a NOEC of 32 mg Cr(III)/kg dry weight has been determined, while for plants the NOEC is around 100 mg Cr(III)/kg dry weight. For soil processes, a large number of values (30) has been used in a statistical extrapolation to give a HC_5 value of 5.9 mg Cr(III)/kg. A PNEC of 3.2 mg Cr(III)/kg dry weight (2.8 mg/kg wet weight) was derived from the earthworm NOEC using an assessment factor of 10. It should be noted that the soil tests were carried out with a highly soluble (and hence bioavailable) form of chromium (III). In the environment, chromium (VI) is likely to be reduced to forms of chromium (III) of limited solubility and bioavailability, and it is unlikely that the concentration of "dissolved" and hence available chromium (III) will reach the levels where effects might be expected. Similarly, there are many natural soils where the levels of total chromium (present as poorly bioavailable chromium (III) complexes) are above the PNECs derived here. The PNECs are therefore not appropriate for such situations.

Exposure

The first manufacturing step involves the production of sodium chromate from chromite ore. The production of sodium chromate in the EU was 103,000 tonnes in 1997. The vast majority of this sodium chromate is converted into sodium dichromate (110,000 tonnes in 1997), and the other three substances are made from sodium dichromate.

The major uses for the chromium (VI) compounds are in the manufacture of other chromium-containing chemicals (e.g. pigments and dyes, chromium sulphate for leather tanning), in metal treatment (mainly chrome plating, but also conversion coatings and brightening) and in wood preservation.

All five substances are solids (coloured crystals). Water solubilities are in the range 115-1670 g/l. Vapour pressures and octanol-water partition coefficients are not relevant for this type of substance.

Emissions to air and to water are possible from production of the five substances. Releases from their use are expected to be to water. Chromium (VI) ions are recognised as toxic to aquatic organisms, and so methods are available to remove them from water before release; however, it is not clear how widely these methods are applied.

The main route of occupational exposure is inhalation from the use of Cr (VI) compounds. Manufacture is largely in enclosed systems although there is potential for exposure during breeches in the system and bagging of product. Although dermal exposure may occur, due to the corrosive nature of Cr (VI) compounds, it is considered that measures taken to prevent substantial skin contact, mean that under intended exposure conditions there would be no prospect of systemic effects arising through dermal exposure. Significant oral exposure is not anticipated in the workplace. The potential for consumer exposure due to the presence of residual chromium VI compounds in copper chromium arsenate (CCA) wood preservative treated timber is very low - the exposure may be higher if CCA treated wood is still wet after impregnation. In the EU it is reported that there is no consumer exposure to Cr(VI) compounds from leather goods, pigments, dyestuffs, stainless steel goods, products derived from vitamin K or montan waxes. Potential exposure by contact, inhalation or ingestion of Cr(VI) from environmental sources (air, water, food) is very low.

Once in the environment, the major dissolved species are $HCrO_4^-$ and CrO_4^{2-} - dichromate species are only important at high concentrations (> 0.4 g Cr/l). Chromium (VI) is a strong oxidising agent, and reacts with a range of reducing agents in the environment to give chromium (III) species. The reverse reaction is possible but requires a

strong oxidising agent and is unlikely under general environmental conditions. It is expected that the majority of the chromium released to the environment will be converted to chromium (III), although this may not be rapid for all releases.

Chromium (VI) species are more mobile in soils and sediments than chromium (III) species. The sorption of chromium (VI) species decreases with increasing pH, while the sorption of chromium (III) species increases with increasing pH. Chromium (VI) does not appear to bioconcentrate significantly in aquatic organisms, but reduction to chromium (III) once in the organism can lead to higher levels of total chromium in organisms.

NATURE OF FURTHER WORK RECOMMENDED

Sufficient information exists to address the hazard classification for all SIDS endpoints, and for a number of other non-SIDS endpoints. However the substances are candidates for further work as follows:

- National or regional exposure information gathering, and if indicated, a risk assessment may need to be considered for the water and soil compartments (based on an existing regional risk assessment for Europe, where a need to limit risks has been identified for a number of uses).

- No toxicity data are available for sediment organisms. These data could be generated as a post-SIDS activity (as the European risk assessment has already identified a risk to surface water, this will not be pursued in the EU).

- A need to limit the risks for all occupational exposure scenarios and from exposures via the environment in view of the mutagenic and carcinogenic properties. For workers, there is also a need to limit the risks for acute toxicity as a result of short-term exposure, skin and eye irritation, respiratory tract sensory irritation, skin sensitisation, occupational asthma and reproductive toxicity (fertility and development).

- No risk assessment for consumer exposure to wood wet from CCA impregnation was performed by the sponsor (UK) as there are controls to prevent the sale of wood which has not been fully dried. If specific controls are not available, then there may be need for exposure data gathering, and if indicated, risk assessment for this exposure scenario.