Chemical Risk assessment
by Carla GUILCAPI DURAN
ASSIGNMENT 2

Chemical Risk Assessment

<table>
<thead>
<tr>
<th>Name</th>
<th>Z Number</th>
<th>Contribution</th>
</tr>
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<tbody>
<tr>
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<td>Z3421229</td>
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</table>
EXECUTIVE SUMMARY

The present risk assessment report has been undertaken for the University Research Committee to determine the degree of health risk that UNSW students and employees might be exposed due an accidental release to the atmosphere of 1 kg of Chromium VI from a test reactor in the form of aerosol 20m upward from the roof of the UNSW Dalton Building (place of future construction of the test reactor). In addition, it has been determined the level of acceptability of the possible risk.

The present risk assessment has been developed in compliance with the EnHealth (2012) Guidelines “Environmental Health Risk Assessment: Guidelines for Assessing Human Health from Environmental Hazards.

This report is based on the case study “Release of chromium VI from the Orica chemical plant Kooragang Island, Stockton” (Base Study) undertaken by the NSW Office of Environment and Heritage to assess the health risk of Stockton residents after their exposition to hexavalent Chromium (Cr VI) which was used as a rough estimation for the present risk assessment.

Five potential pathways of exposure at which students and employees from UNSW (adult people >16 years old) might be exposed were determined, which are: inhalation of suspended Cr VI in the air, oral ingestion of food contaminated with Cr VI, accidental ingestion of Cr VI by eating contaminated soil, inhalation of re-suspended dust contaminated by Cr VI and oral ingestion of Cr VI by contact with surfaces contaminated.

The assumptions used intended to address the worst case scenario of exposure. All of these assumptions lead to a great level of conservatism in the risk estimation. The final figures probably to significantly over forecast the non-threshold cancer risk estimations because the highest concentrations of the greatest mass of Cr VI released from a real incident (Base Study) were used in the estimations. Moreover, the Gaussian Plume Model used estimated the concentration of Cr VI in the air under the most unfavorable meteorological conditions and the cancer potency factors and estimation of risk were developed according to the hierarchy providing by enhelath guidelines which makes the process transparent and consistent (enhelath, 2012).

It is not expected that the students and employees from UNSW exposed to aerosol Cr VI experience short term effects due to the value of Hazard Index (HI), 4x10^{-5}, was less than 1.

The total increase lifetime cancer risk (ILCR total) estimated was 5x10^{-5}. Due to the fact that it exceeds the target risk level in Australia of 1x10^{-5}, the risk at which students and employees from UNSW would be exposed is under unacceptable level.

Nevertheless, according to the sensitivity analysis undertaken, it was found that under exposure time less than 18 minutes to the aerosol via direct inhalation, the risk level of the population may be acceptable and therefore, the possibility of installation of the project. Finally, a communication summary was developed to manage the potential risk.
Introduction and Issue Identification

The University Research Committee has hired Risk Group 15 to determine the degree of health risk that UNSW students and employees might be exposed due an accidental release to the atmosphere of 1 kg of Chromium VI from a test reactor in the form of aerosol 20m upward from the roof of the UNSW Dalton Building (future construction of the test reactor). In addition, it has been determined the level of acceptability of the possible risk.

The case study "Release of chromium VI from the Orica chemical plant Kooragang Island, Stockton" ('Base Study') undertaken by the NSW Office of Environment and Heritage to assess the health risk of Stockton residents after their exposition to hexavalent Chromium (Cr VI) was used as a rough estimation to assess the chemical risk of the Chromium VI test reactor project. In the mentioned study, 10 to 20 kg of Cr VI was released from one of Orica chemical plant's vent stacks, and estimated 1.5 kg of Cr VI was dispersed in form of aerosol on a section of Stockton on 8th August 2011.

The worst case scenario, assumptions and other data from the Base Study were used as a reference and after modification according to the site conditions of the Chromium VI test reactor project. This information is shown and explained in detail in each one of the respective sections and appendix, but is summarized in Table 1

The present report assess the exposure to Cr VI via five pathways, which are 1) exposure via inhalation of suspended Cr VI in the air, 2) exposure via inhalation of resuspended dust from surfaces contaminated with Cr VI, 3) exposure via ingestion of food contaminated with Cr VI, 4) exposure via ingestion of Cr VI by contact with surfaces contaminated and 5) exposure via ingestion of Cr VI by eating contaminated soil.

Table 1. Worst Case Scenario

<table>
<thead>
<tr>
<th>Release time of 1 kg of Cr VI from the vent: 20 minutes (Base Study).</th>
<th>Age of the population: &gt;16 years old; therefore all are considered adults.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wind speed: 3.5 m/s (lowest in the year): A low wind speed keeps the Cr VI in the boundary of UNSW</td>
<td>Daytime incoming solar radiation: Strong</td>
</tr>
<tr>
<td>Downwind distance of highest exposure: 0.2 km. The highest concentration of Cr VI was estimated at 0.5 km from the building where is located the test reactor by using the Gaussian plume model.</td>
<td>Values of vertical distance (z) and lateral distance from the plume centerline (y); zero. Estimation of the maximum concentration of Cr VI at ground level and prediction of violaton of guideline regulation is achieved when both parameters are zero in the Gaussian plume model.</td>
</tr>
<tr>
<td>Time of exposure by inhalation of the aerosol: 1 hour</td>
<td>Concentrations: highest concentrations of Cr VI in food (after modification), soil, dust, and, surfaces from the Base Study.</td>
</tr>
<tr>
<td>Population considered: Students and employees of UNSW</td>
<td>Exposure frequency to soil, dust and contaminated surfaces: 10 days. In 10 days is expected that Cr VI be converted to Cr III (ATSDR, 2012).</td>
</tr>
<tr>
<td>Time of the event: Lunch time due to the highest number of people found near the building where is located the test reactor.</td>
<td>Period of food intake: 2 hours</td>
</tr>
</tbody>
</table>

In the following section is presented the hazard assessment, the exposure assessment, the risk characterisation and finally a communication summary.
2. Hazard Assessment

2.1 Hazard Identification

Chromium exists in three oxidation states: chromium 0, chromium III and chromium VI. Chromium III is an essential trace element in humans and is relatively nontoxic. Chromium VI is considerably more toxic than chromium III, about 100 times toxic. It poses both cancer and non-cancer related adverse health effects.

2.1.1 Non-cancerous

Ingestion, whether by inhalation or ingestion of hexavalent chromium compound poses varied types of health hazards. For instance, doses of 50 to 80 mg already have a caustic effect and produce digestive disorders and doses as low as 300 mg have been observed to cause deaths (Haguenoer & Furon, 1982).

Also, a number of epidemiological studies conducted have shown the association between inhalation of chromium VI and non-carcinogenic endpoints such as upper respiratory irritation, lower respiratory effects and systemic effects. Exposure to chromium VI compounds like chromic acid, with concentration ranging from 0.12 to 5.6 mg/m³ causes nasal tissue damage, including perforated septum, ulcerated septum, chrome holes, nosebleed, and inflamed mucosa (Bloomfield & Blum, 1928).

Chromium VI compounds also have dermal toxicity associated with them. Dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis. Primary irritant dermatitis is related to the direct cytotoxic properties of chromium, while allergic contact dermatitis is an inflammatory response mediated by the immune system (EPA, 1998).

2.1.2 Cancerous

Oral administration of chromium VI compounds has been demonstrated to cause cancer. Studies in rats NTP (NATIONAL TOXICOLOGY PROGRAM, 2008) and a study of miners in Ontario suggested that exposure to chromium may have been associated with stomach cancer.

Cr (VI) has been found to be carcinogenic from epidemiologic studies of chromium-exposed workers and from animal studies. Chromium VI compounds have produced the following cancer types in animal assays: lung tumors following inhalation of aerosols of sodium chromate and pyrolyzed Cr(VI)/Cr(III) oxide mixtures in rats, lung tumors following intratracheal administration of sodium dichromate in rats, intrapleural implant site tumors for various Cr(VI) compounds in rats, intrabronchial implantation site tumors for various Cr(VI) compounds in rats, intramuscular injection site tumors in rats and mice, and subcutaneous injection site sarcomas in rats (Glaser, Hochrainer, & Koppel, 1985).

2.2 Dose -Response

2.2.1 Health Risks (Non-cancerous)

By oral ingestion: There is no an acute oral MRL for hexavalent compounds but an oral MRL of 0.005 mg chromium(VI)/kg/day has been taken as guideline value for intermediate (15-364 days) exposure to hexavalent chromium compounds (Agency for Toxic Substances and Disease Registry, 2012).

By inhalation: A number of epidemiological studies conducted have shown the association between inhalation of chromium VI and non-carcinogenic endpoints such as upper respiratory irritation, lower
respiratory effects and systemic effects. However, we have chosen our guideline value based on Temporary Emergency Exposure Level (TEEL) are derived by the U.S. Department of Energy, which in our case for worst-case scenario is 0.096 mg/m³ or 9.62 μg/m³.

Dermal toxicity: Dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis. The threshold concentration of extractable Cr (VI) in solid material may be as low as 10 ppm to induce dermatitis (Stern, Bagdon, Hazen, & Marzulli, 1993). Thus, 10 ppm (μg/g) has been taken as TDI.

2.2.2 Health Risks (Cancerous)

Oral: Based guidelines derived by (OEHHA (2011) Public Health Goal for Hexavalent Chromium (Cr VI) in Drinking Water). Chronic oral cancer slope factor for chromium VI of 0.5 mg/kg day⁻¹ is taken.

Inhalation: For inhalation exposure related cancer risk WHO’s Risk Unit Factor of 4.2 × 10⁻⁶ (μg/m³)⁻¹ is taken as guideline value based on most relevant based on ranking of hierarchy of source of Toxicology information provided by enhealth guidelines. This is because WHO methodology is consistent with Australian science policy and thus has higher ranking in hierarchy of Toxicology sources recommended by enhealth guidelines.

3 Exposure Assessment

3.1 Site model and analysis of hazard locations

Exposure will only exist when the “worst-case-scenario” takes place in the campus. A conceptual site model is built based on enHealth guidelines (enHealth, 2012).

1. The boundary of the study is all the area of the UNSW. All the released Cr VI will be stored in the campus area. No Cr VI will be diffused outside the boundary. The released point is the roof of UNSW Dalton Building. Moreover, on the ground floor there is a café where is also sold food for people.

2. If the accident happens the released aerosol of Cr VI will flow into the air. The mass of Cr VI considered in the assessment is 1 kg of Cr VI. The whole releasing process is assumed to last 20 minutes.

3. Considering the café under the building, parts of Cr VI will be adhered to the surface of food. We assume that the worst time for the happening of accident is the lunch time. In that case, oral ingestion of Cr VI will take place when people would be eating food at lunch time.

4. Due to gravity, Cr VI particles will settle on the surface of buildings, grass, trees, benches and soil in the campus. Also the dust in the air will be contaminated by Cr VI.

5. In addition, after the settling, the chromium would be absorbed by soil. People who is outdoors would have the risk of ingestion of soil by close contacting with it.

![Figure 1 Site model of Cr VI exposure](image-url)
3.2 Identification of exposure population
According to the enHealth guideline, in a normal exposure assessment it is needed to analyze impacts on different age groups: children and adults. However, as requirement, it has only been considered students and employees from UNSW (>16 years old).

3.3 Identification of potential exposure pathways
The figure 3.1 illustrates the possible pathways that can cause the exposure to people in the campus area. Each of these potential ways are considered in order to calculate the amount of Cr VI that flow into human’s body.

![Potential pathways in campus](image)

As showed in the graph, the 5 most potential pathways to cause intake of Cr VI are as follow and the descriptions for each pathway are in:

1. Exposure via inhalation of suspended Cr VI in the air
2. Exposure via oral ingestion of food contaminated with Cr VI
3. Exposure via accidental ingestion of Cr VI by eating contaminated soil
4. Exposure via inhalation of re-suspended dust contaminated by Cr VI
5. Exposure via oral ingestion of Cr VI by contact with surfaces contaminated

The dermal pathway was not included because there is not published information of a threshold guideline value of Cr VI in the air that induce sensitization to skin or generate symptoms in sensitized people (Frangos, 2011). The dermal absorption of Cr VI is not likely to be sufficient through the intact skin but it can cause irritation or corrosive responses on it (Gad, 1989).

Description of each pathway
Air: In case of an accident, the majority of Cr VI would be in the air in the form of aerosol. The release time is assumed to be 20 minutes with the presence of the aerosol suspended in the air during 1 hour. The event is only assumed to happen once and anymore in the year.

Food: Considering that there is café restaurant under Dalton building, Cr VI can be adhere to the surface of food. It is assumed that the food would be bought and ingested in a maximum period of 2 hours, and people only eat the contaminated food for lunch the day of the incident. All the contaminated food will be disposed later by UNSW.

Soil: Contaminated soil can be ingested occasionally when people are in a close contact with soil. For example, students who like to sit on the grass and play soccer on the playground are likely to have risk to ingest soil into body through mouth.
Dust: The dust contaminated will exist after the incident in the form of Cr VI until its conversion to Cr III after 10 days.

Surface: After accident, people will touch the contaminated surface of buildings, plants, and infrastructures in the campus, and the Cr VI will be ingested into body by hand to mouth behaviors. The retention time of Cr VI is assumed at 10 days (ATSDR, 2012), after which Cr(III) will be converted to Cr(III) which is more stable than Cr VI (Stern, 2010).

3.4 Estimation of threshold and non-threshold exposure concentration and intakes for each pathway
In this section, it has been estimated the threshold and non-threshold exposure concentration for the five pathways mentioned before. To estimate the exposure via inhalation of suspended Cr VI in the air, Gaussian plume model (Appendix B) was used to estimate the concentration of Cr VI in the air for several reasons: 1) it is one of the models approved in New South Wales for the modelling of air pollutants (NSW, 2005); 2) the impact of meteorology can be study by using this model; and 3) it is the most common model used due to its simplicity and 4) compliance of the air quality regulations and maximum concentration of the contaminant at ground level can be determined by using this model (Garcia, 2012). In addition, the most unfavorable meteorological conditions linked to the highest concentrations of Cr VI in the air at ground-level were used in the model (Appendix B). On the other hand, for the other four pathways, the concentrations of Cr VI in food, soil, dust and surface where taken from the Base Study (Frangos, 2011) after adjusting the values to the particularities of the present assessment. Due to limitation in space, the assumptions as well as other data for the calculations (Appendix C) to address the worst case scenario of exposure are provided in detail in Appendixes C to D. A summary of the threshold and non-threshold exposure and intake concentrations is provided in Table 2.

It is worthy to mention that for non-threshold exposure concentrations the average life time considered was 82 years (enHealth, 2012), whereas for threshold exposure concentrations, the average life time assumed was 1 year.

Different from the Base Study which only evaluates threshold exposure concentration via general inhalation, the present study additionally estimates the threshold exposure concentration for the other four exposure pathways.

Table 2. Summary of the threshold and non-threshold exposure and intake concentrations

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Inhalation of aerosol exposure</th>
<th>Ingestion of food exposure</th>
<th>Ingestion of soil exposure</th>
<th>Inhalation of dust exposure</th>
<th>Ingestion after contact with surface exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Threshold</td>
<td>$1 \times 10^4$ µg/m$^3$</td>
<td>$1 \times 10^8$ mg/kg/day</td>
<td>$7 \times 10^{11}$ mg/kg/day</td>
<td>$1 \times 10^{13}$ mg/kg/day</td>
<td>$1 \times 10^7$ mg/kg/day</td>
</tr>
<tr>
<td>Threshold</td>
<td>$9 \times 10^3$ µg/m$^3$</td>
<td>$1 \times 10^6$ mg/kg/day</td>
<td>$6 \times 10^9$ mg/kg/day</td>
<td>$8 \times 10^{11}$ mg/kg/day</td>
<td>$1 \times 10^5$ mg/kg/day</td>
</tr>
</tbody>
</table>

3.5 Uncertainty analysis for exposure assessment
Due to limitation of space, uncertainties of exposure assessment will be covered in risk characterisation section.

4. Risk Characterisation
Risk Estimation
Hazard Quotients and Total Increased lifetime cancer risk
To estimate hazard quotient (HI), acute exposure concentrations of five pathways were compared against Temporary Emergency Exposure Limit (TEEL) and Minimal Risk Level (MRL) to estimate the hazard quotients. To estimate the total Increased lifetime cancer risk (ILCR total), chronic exposure
concentrations were multiplied by a Cancer Slope Factor (CSF) or by a Unit Risk Factor (URF). Risk estimation does not show a high level of precision (Enhealth;2012), therefore the values are reported to one significant figure. Formulas according enhealth (2012) and calculations for threshold and non threshold risk estimation are shown in appendix D, and E, respectively. The final results are summarized in table 3.

Table 3. Threshold and non threshold risk estimation results

<table>
<thead>
<tr>
<th>Risk Estimation</th>
<th>Aerosol inhalation</th>
<th>Dust Resuspended</th>
<th>Food contaminated</th>
<th>Incidental surface contact</th>
<th>Ingestion of soil</th>
<th>Total</th>
<th>Acceptable?</th>
</tr>
</thead>
<tbody>
<tr>
<td>HI</td>
<td>1.6E-03</td>
<td>8.6E-13</td>
<td>2.6E-04</td>
<td>3.6E-03</td>
<td>1.6E-06</td>
<td>4.6E-03</td>
<td>Yes, HI&lt;1</td>
</tr>
<tr>
<td>ILCR</td>
<td>5.6E-06</td>
<td>4.6E-15</td>
<td>7.6E-06</td>
<td>6.6E-08</td>
<td>4.6E-11</td>
<td>5.6E-06</td>
<td>No, ILCR total &gt; 1x10^-6</td>
</tr>
</tbody>
</table>

Table 1 shows that the estimated HI for the proposed project was 4 x 10^-3 (less than 1) which implies compliance of the health guideline value and no need of a detailed risk assessment (enhealth, 2012). However, total increased lifetime cancer risk (ILCR total) of the proposed project was 5 x 10^-5 which exceeds the target risk level in Australia, which is 1 x 10^-5 (enhealth, 2012).

An ILCR total of 5 x 10^-5 means that 5 people out of 1000 exposed to the Cr VI aerosol have the probability of developing of cancer risk in a lifetime period of 82 years. Nevertheless, the allowed levels are 1 people out of 1000 exposed.

The Base Study reported a calculated lifetime risk (2 x 10^-6 to 4 x 10^-7) lower than the present risk assessment. It was showed to be within acceptable levels at NSW; however, according to the enhealth guideline, it was exceeded.

Uncertainties

The uncertainties accepted are as follows:

- **Model uncertainty:** All models have strengths and limitations and Gaussian Plume Model is not the exception. For example, the model does not account for surface roughness and terrain configuration which influence in the transport of the contaminant (Wong and Liu, 2013)
- **Population exposed:** The scope of the present report assess the cancer risk of adult people (>16 years old) at UNSW; however, in case of an incident, younger people may also be involved and exposed to maximum concentration of Cr VI at UNSW.
- **Pathways of exposure:** There may be more cancer mechanisms for Cr VI to be assessed.
- **Cancer Potency Factors:** The use of different cancer potency factors can also lead to variation in the figures of risk estimation.
- **Meteorology:** The weather conditions of the day that where the incident can occur are unpredictable.
- **Concentrations:** It is not possible to know, in case of an incident, the exact concentrations of Cr VI that the population would be exposed by each pathway.
- **Lack of scientific data:** The current risk estimation can differ due lack of acute guideline values to assess effects Cr VI inhalation and oral ingestion. Therefore intermediate MRL (ATSDR, 2013) and TEEIs were used.
- **Skin sensitisation:** There was not found threshold concentrations than can induce skin sensitisation by airborne chromium exposure. Therefore, this pathway of exposure was not
considered. However, some people may have a skin more sensitive than others putting them in a higher level of risk.

Above all, it is important to make emphasis that the worst case scenario and in fact, the worst case assumptions were used to estimate the cancer risk providing in this way an over conservatism in the final results.

**Sensitivity Analysis**

**Gaussian Plume Model:** Even though the model has limitations as all models, the Gaussian Plume Model is the most commonly used to determine the impact of meteorology in the concentration of contaminants for its simplicity, conservative estimation of risk and prediction of concentrations pretty well correlated with experience (Faucher et al., 1996). Moreover, it is approved by NSW for modeling of air contaminants. In addition, to solve the problems related with low consideration of surface roughness and terrain configuration, there has been undertaken studies to adopt this parameters highly influence in the transport of the contaminant. Moreover, future works are looking for solving issues related to variability of dispersion of the contaminant with buildings of different height and shape (Wong and Liu, 2013).

**Pathways of exposure:** Although only five exposure pathways were assessed, those represent the most critical ones linked to the generation of the worst case scenario.

**Quantitative Sensitivity Analysis**

The change in the ILCR total was evaluated under 5 scenarios. The results are summarized in Table 2.

**Population exposed:** If under the same worst case scenario a population between 3 to 16 years old were exposed, the ILCR total would increase and thus, the risk level would be not acceptable.

**Cancer Potency Factors:** If other cancer potency factor were used, such as URF of $1.5 \times 10^{-1}$ µg/m³ (OEHHA, 2011), the ILCR total would still be under unacceptable level.

**Increased of wind speed:** One of the meteorology parameters that has highly influenced in Gaussian Plume Model is wind speed (Carlos García-Díaz and Gozalvez-Zafrilla, 2012), so if the highest wind speed in Sydney (25.3 km/h) is considered (BOU (2013), the ILCR total would be reduced having compliance with the target risk.

**Increase in distance:** If the distance increases to 600 m that is the maximum distance from the test reactor to the boundary of the UNSW at Botany St, the ILCR total would still be under unacceptable level.

**Reduction of Exposure Time:** If the exposure time via direct inhalation of the aerosol is reduced to less than 18 minutes, the ILCR total would be under acceptable level.

**Table 4. Sensitivity Analysis**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ILCR total value</th>
<th>Variation from the current value</th>
<th>Probability of developing cancer after a lifetime period of 82 years after the exposure to the CrVI aerosol</th>
<th>Is the Target risk level acceptable?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present Risk Assessment Report</td>
<td>5E-06</td>
<td>-</td>
<td>5 people out of 1,000,000</td>
<td>No</td>
</tr>
<tr>
<td>Children (3-16 years old)</td>
<td>1E-06</td>
<td>(+10000%)</td>
<td>1 people out of 100,000</td>
<td>No</td>
</tr>
<tr>
<td>URF</td>
<td>2E-06</td>
<td>(+1000%)</td>
<td>2 people out of 100,000</td>
<td>No</td>
</tr>
<tr>
<td>Highest wind speed (25.3 km/h)</td>
<td>1E-06</td>
<td>(-4000%)</td>
<td>1 people out of 1,000,000</td>
<td>Yes</td>
</tr>
<tr>
<td>Distance (600 m)</td>
<td>2E-06</td>
<td>(-6000%)</td>
<td>2 people out of 1,000,000</td>
<td>No</td>
</tr>
<tr>
<td>Exposure time (&lt;18 min)</td>
<td>1E-06</td>
<td>(-8000%)</td>
<td>1 people out of 1,000,000</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Table 4 shows that the ILCR total still exceeds the target risk level when there is exposition of younger population (3-16 years old), change in cancer potency factor (URF) and increase in distance. Nevertheless, by using the high wind speed, there is compliance with the target risk value, but the worst case scenario would not be evaluated because the boundaries of UNSW end at 600m. Finally,
when the exposure time via direct inhalation of the aerosol is less than 18 minutes, the ILCR total is acceptable (health risk is reduced) which leads to an alternative to manage the risk that is addressed in the communication summary section.

Summary of Risk Information

Five potential pathways of exposure at which students and employees from UNSW (adult people >16 years old) may be exposed were estimated in the exposure assessment section. From those pathways, the HQ of inhalation of suspended Cr VI in the air (1x10^-3), and oral ingestion of Cr VI by contact with surfaces contaminated (1x10^-5) had the highest HQs with exposure doses of 9 x 10^-3 µg/m^3 and 1×10^-5 mg/kg/day, respectively. However, it is not expected that the population exposed experience short term effects due to the value of HI (4x10^-5) was less than 1. Only if people were short term exposed at doses greater than 60 µg/m^3, acute effects such as nasal irritation may be observed (Blumfeld & Blum; 1928). For HI estimation, a TEEL of 9.62 x 10^-3 µg/m^3 and MRL of 5x10^-3 mg/kg day^-1 were used due to the absence of acute inhalation and oral guideline data, respectively. On the other hand, Cr VI can cause cancer via oral and inhalational pathways. In the ILCR total estimation, a CFS of 0.5 mg/kg day^-1 was selected due to its well determination based on NTP bioassay animal study that assessed carcinogenic effects by Cr VI via ingestion. Moreover, a URF of 4.2 x 10^-5 µg/m^3 was used based on hierarchy of toxicological information provided by enHealth (2012) and because it was determined from human studies. The ILCR total estimated (5x10^-6) exceeds the target risk level in Australia. Only if the exposure time is less than 18 minutes, the risk level may be acceptable according to the sensitivity analysis that was undertaken.

5. Communications Summary

The School of Industrial Engineering in UNSW is going to investigate a new industrial process which involved a construction of a test reactor on the roof of the UNSW Dalton Building and Cr VI will be used in this project. Before this project begin, a health risk assessment has been undertaken by the University Research Committee.

According to the health risk assessment, if the worst-case-scenario occurs, 1kg of Cr VI will be released into air. There are 5 potential pathways of exposure through which ones people may be at risk: air, food, soil, dust and contacting with contaminated surface. Cr VI will convert to Cr III after 10 days. There is not short term effects according to the risk assessment; however, there is an unacceptable lifetime cancer risk for students and employees in this campus. Therefore, if an accidental release does occur, there is only 18 minutes to have a lower health risk, so the next information should be communicated advices to stakeholders, students and media:

- To be more safety, students and employees should stay inside the building at least for the first one hour after the incident.
- Stop eating any food when you are in the café under the Dalton Building. Never buy any food from the café before the authority of university announce that it is safe enough. Also, it is better for people to have their food indoor during the exposure period.
- Do not sit on the grass land in the contaminated area, the outdoor activities in campus should be cancelled temporarily during 10 days after the incident.
- Remember to wash hands every time after touching the contaminated surface in campus.
- Taking a protective mask if people have to go outside in campus to avoid inhaling the dust suspended in the air.
- For the sensitive people who has allergic response to Cr VI, it is better for them to stop any activities in campus and stay outside it.
Conclusion

In conclusion, the present health risk assessment elaborated in compliance with enHealth guidelines shows that in case of an incidental release of 1kg of CrVI in the form of aerosol from the future test reactor, no acute effects would be observed (HI<1). However, the estimated total increase lifetime cancer risk ($5 \times 10^{-4}$) exceeded the target risk level in Australia, which show that students and employees from UNSW would be exposed under an unacceptable level of risk. Nevertheless, with an exposure time less than 18 minutes to the aerosol via direct inhalation, the risk level would be acceptable level and therefore there is the possibility of installation of the project. In case of incident, the authorities can rely on the communication summary developed to manage the potential risk.
Appendix A: Dose Response

Non-Cancer
INGESTION (ORAL):
There is no an acute oral MRL for hexavalent compounds but an oral MRL of 0.005 mg chromium(VI)/kg/day has been derived for intermediate (15-364 days) exposure to hexavalent chromium compounds (Agency for Toxic Substances and Disease Registry, 2012).

This was based on studies in rats where Hematological effects like microcytic, hypochromic anemia were observed in male rats after exposure for 22 days in the (NATIONAL TOXICOLOGY PROGRAM, 2008) were identified as the most sensitive effect of intermediate-duration oral exposure to chromium(VI). In this study, male F344/N rats (6–7 weeks old) were exposed to sodium dichromate dihydrate in drinking water in a 2-year toxicology and carcinogenicity study, with hematological assessments conducted at 22 days, 3 months, 6 months, and 1 year. To determine the point of departure for derivation of the intermediate-duration oral MRL, available continuous-variable models in the EPA Benchmark Dose were fit to the data for Hct, Hgb, MCV, and MCH in male rats (NTP 2008a). Because several hematological parameters are used to define the clinical picture of anemia, the BMDL10 values for hemoglobin, MCV, and MCH (none of the models provided an adequate fit for hematocrit) were averaged resulting in a BMDL10 of 0.52 mg chromium(VI)/kg/day. The intermediate-duration MRL of 0.005 mg chromium(VI)/kg/day was derived by dividing the average BMDL10 by a composite uncertainty factor of 100 (10 for extrapolation from animals to humans and 10 for human variability).

Inhalation
Absence of acute inhalation data from Level 1 sources
It has been adopted a guideline value based on Temporary Emergency Exposure Level (TEEL) which are derived by the U.S. Department of Energy Subcommittee on Consequence Assessment and Protective Actions (SCAPA) according to a specific, standard methodology. The TEEL methodology uses available levels of concern and manipulates current data using a peer-reviewed, approved procedure in order to establish the TEELs.
A chemical may have up to three TEEL values, each of which corresponds to a specific tier of health effects. The three TEEL tiers are defined as follows:

- **TEEL-3** is the airborne concentration (expressed as ppm [parts per million] or mg/m³ [milligrams per cubic meter]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening adverse health effects or death.
- **TEEL-2** is the airborne concentration (expressed as ppm or mg/m³) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting, adverse health effects or an impaired ability to escape.
- **TEEL-1** is the airborne concentration (expressed as ppm or mg/m³) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. However, these effects are not disabling and are transient and reversible upon cessation of exposure.
### TEELs based on Protective Action Criteria (PAC) Rev 27

<table>
<thead>
<tr>
<th>Chemical</th>
<th>CAS No.</th>
<th>TEEL-1 (mg/m³)</th>
<th>TEEL-2 (mg/m³)</th>
<th>TEEL-3 (mg/m³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chromates</td>
<td>13907-45-4</td>
<td>0.096</td>
<td>0.1</td>
<td>30</td>
</tr>
<tr>
<td>Chronic trioxide; (Chromium(VI) oxide (1:3))</td>
<td>1333-82-0</td>
<td>0.096</td>
<td>0.1</td>
<td>30</td>
</tr>
<tr>
<td>Potassium dichromate</td>
<td>7778-50-9</td>
<td>0.14</td>
<td>1.5</td>
<td>44</td>
</tr>
<tr>
<td>Sodium dichromate dihydrate</td>
<td>7788-12-0</td>
<td>0.14</td>
<td>1.5</td>
<td>45</td>
</tr>
</tbody>
</table>

### Dermal toxicity

Dermatitis results from exposure to Cr (VI) can occur either by direct dermal contact or by ingestion. Toxicity by ingestion is already covered in acute toxicity. Although, Cr(VI), is a potenter sensitizer and inducer of allergic contact dermatitis, no standard or guideline currently exists for protection against these effects. There appears to be a generalized allergenic potential among the various compounds of Cr(VI). Nevertheless, threshold concentration of extractable Cr(VI) in solid material may be as low as 10 ppm to induce dermatitis (Stern, Bagdon, Hazen, & Marzulli, 1993). Thus, 10 ppm (µg/g) may be taken as TDI.

### Cancer

**Oral:**

Chronic oral cancer slope factor for chromium VI of 0.5 mg/kg day⁻¹ has been derived by (OEHHA (2011) Public Health Goal for Hexavalent Chromium (Cr VI) in Drinking Water) which is based on NTP (NATIONAL TOXICOLOGY PROGRAM, 2008).

In this study, drinking water containing 14.3, 57.3, 172, or 516 mg of sodium dichromate dihydrate per liter of water was administered to groups of 50 male and female rats and female mice for 2 years. Similar groups of male mice were given 14.3, 28.6, 85.7, or 257.4 mg of sodium dichromate dihydrate per liter of water. Control animals received the same tap water with no chemical added. At the end of the study, tissues from more than 40 sites were examined for every animal. Male and female rats exposed to sodium dichromate dihydrate had carcinomas of the mouth, but none occurred in the control rats. Male and female mice receiving sodium dichromate dihydrate had greatly increased rates of cancer of the small intestine.

**Inhalation:**

For inhalation exposure related cancer risk WHO’s Risk Unit Factor is considered most relevant based on ranking of hierarchy of source of Toxicology information provided by enhealth guidelines, which is 4.2 x 10⁻³ (µg/m³)⁻¹. This is because WHO methodology is consistent with Australian science policy and thus has higher ranking in hierarchy of Toxicology sources recommended by enhealth guidelines.

A WHO Working Group in 1994 reviewed four sets of data for chromate production workers to estimate the lung cancer risk posed by the presence of Cr(VI) in the atmosphere (WHO, 2000). The “best estimate” of the risk resulting from a lifetime exposure at a concentration of 1 µg m⁻³ was 4.2 x 10⁻².

A risk assessment can also be made on the basis of the study carried out by Langard et al. on ferrochromium plant workers in Norway. The chromium concentration to which the workers were exposed is unknown, but measurements taken in 1975 showed a geometric mean value of about 530 µg/m³. Assuming that the content of chromium(VI) in the sample was 19% and previous...
concentrations were at least as high as in 1975, the ambient concentration would have been about 100 μg/m3. On the assumption that occupational exposure lasted for about 22 years, the average lifetime exposure can be determined as 6.9 μg/m³ (X = 100 μg/m³ × 8/24 × 240/365 × 22/70). When workers in the same plant who were not exposed to chromium were used as a control population, the relative risk of lung cancer in chromium exposed workers was calculated to be 8.5. The lifetime unit risk is therefore $4.3 \times 10^{-2}$.

<table>
<thead>
<tr>
<th>Agency</th>
<th>Value (μg/m³)⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>(WHO, 2000)</td>
<td>$4.2 \times 10^{-2}$</td>
</tr>
<tr>
<td>(US EPA IRIS, 1998)</td>
<td>$1 \times 10^{-2}$</td>
</tr>
<tr>
<td>(Agency for Toxic Substances and Disease Registry, 2012)</td>
<td>$2.5 \times 10^{-2}$</td>
</tr>
</tbody>
</table>
APPENDIX B: Gaussian Model in Calculation of Aerosol Concentration

According to Garcia (2012), in order to determine compliance of the air quality regulations and maximum concentration of the contaminant at ground level, the vertical distance (z) and lateral distance from the plume centerline (y) should be zero in the Gaussian plume model (figure B1). Therefore, these considerations were used in the estimation of the concentration of Cr VI in the air and the most unfavorable meteorological conditions shown in the table below.

![Figure B1. Nomenclature of Gaussian Plume Model](image)

The formula used in the Gaussian Plume Model is shown as follows:

\[
C(x,y,z) = \frac{Q}{2\pi u \sigma_y \sigma_z} \left\{ e^{-\frac{(z-h)^2}{2\sigma_z^2}} + e^{-\frac{(z+h)^2}{2\sigma_z^2}} \right\} \left\{ e^{-\frac{(y)^2}{2\sigma_y^2}} \right\}
\]

Among this formula:

\[
\sigma_y = 465.11628 \times x \times (\tan \theta) \\
\theta = 0.017453293 \times (c - d \times \ln(x))
\]

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source Emission Rate</td>
<td>Q (g/s)</td>
<td>0.8 Assumed that 1 kg of Cr VI is release in 20 minutes</td>
</tr>
<tr>
<td>Downwind distance</td>
<td>x (km)</td>
<td>0.2 Distance at which the concentration of Cr VI is the highest</td>
</tr>
<tr>
<td>Lateral distance from the plume centerline</td>
<td>y (km)</td>
<td>0 y=0 and z =0 to achieve maximum concentration at ground level and to predict any violation of the guideline values (Garcia&amp;Nafrilla, 2012)</td>
</tr>
<tr>
<td>Vertical distance in</td>
<td>z (km)</td>
<td>0</td>
</tr>
<tr>
<td>Wind velocity</td>
<td>u (m/s)</td>
<td>3.5 Minimum wind speed in Sydney (May) during a year (Australian Government, 2013).</td>
</tr>
<tr>
<td>Effective stack height</td>
<td>h (m)</td>
<td>30 Plume rise is 20 m (given) + assumed that Chemical Building height is 10 m</td>
</tr>
<tr>
<td>Pasquill Stability Class</td>
<td>B</td>
<td>Pasquill Stability Class is B (Pasquill, 1974) when surface wind speed is between 3-5 m/s and the daytime incoming solar radiation is strong (lunch time)</td>
</tr>
</tbody>
</table>
\[ \sigma_z = ax^b \]
\[ \Theta = 0.017453293(c - d \ln(x)) \]

<table>
<thead>
<tr>
<th>Pasquill Stability Category</th>
<th>c</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>24.1679</td>
<td>2.5334</td>
</tr>
<tr>
<td>B</td>
<td>18.3330</td>
<td>1.8096</td>
</tr>
<tr>
<td>C</td>
<td>12.9000</td>
<td>1.0857</td>
</tr>
<tr>
<td>D</td>
<td>8.3330</td>
<td>0.72382</td>
</tr>
<tr>
<td>E</td>
<td>6.2500</td>
<td>0.54287</td>
</tr>
<tr>
<td>F</td>
<td>4.1667</td>
<td>0.36191</td>
</tr>
</tbody>
</table>

Figure B2. Parameters in pasquill stability class B

Thus the pasquill stability class is B, so in the formulas:

\[ a=90.673, \ b=0.93198, \ c=18.333, \ d=1.8096 \]

\[ \theta = 0.017453293(18.333 - 1.8096 \times \ln(0.2)) = 0.2 \text{ radians} \]

\[ \sigma_y = 465.11628 \times 0.2 \times (\tan 0.2) = 15.6 \text{ m} \]

\[ \sigma_p = 90.673 \times 0.2^{0.93198} = 20.2 \text{ m} \]

\[ C_{(300,0,0)} = \frac{0.8 g/s}{2 \times 3.14 \times 3.5 m/s \times 15.6 m \times 20.2 m} \left( \exp \left( -\frac{(-30m)^2}{2 \times (20.2m)^2} \right) - \exp \left( -\frac{(-0)^2}{2 \times (15.6m)^2} \right) \right) \]

\[ = (1.16 \times 10^{-4} g/m^3) \times (0.33 + 0.33) \times 1 = 0.00008 g/m^3 = 80 \mu g/m^3 \]
APPENDIX C: Dose Calculation of Each Pathway

Inhalation of aerosol exposure

Derived from Environmental Health Risk Assessment Guidelines (enHealth, 2012)

\[ EC = \frac{C_{air} \times ET \times EF \times ED}{CF \times AT} \]

EC (\( \mu g/m^3 \)) = exposure concentration

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Adult</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration in air</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( C_{air} ) (( \mu g/m^3 ))</td>
<td>80</td>
<td>Gaussian Plume Model</td>
</tr>
<tr>
<td>Exposure time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ET (hrs/day)</td>
<td>1</td>
<td>Assumed 1 hour of exposure</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF (days/yr)</td>
<td>1</td>
<td>Release no expected to occur again</td>
</tr>
<tr>
<td>Exposure duration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED (yrs)</td>
<td>1</td>
<td>Assumed 1 year</td>
</tr>
<tr>
<td>Conversion Factor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CF (hrs/yr)</td>
<td>8760</td>
<td>365x24</td>
</tr>
<tr>
<td>Averaging time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>( AT_1 ) (yrs)</td>
<td>82</td>
<td>Non-carcinogenic effects (enHealth, 2012)</td>
</tr>
<tr>
<td>( AT_2 ) (yrs)</td>
<td>1</td>
<td>carcinogenic effects</td>
</tr>
</tbody>
</table>

Non-carcinogenic effects:

\[ EC = \frac{80 \mu g/m^3 \times 1 \text{ hr/day} \times 1 \text{ day/yr} \times 1 \text{ yr}}{8760 \text{ hrs/yr} \times 82 \text{ yrs}} = 0.00011 \mu g/m^3 \]

Carcinogenic effects:

\[ EC = \frac{80 \mu g/m^3 \times 1 \text{ hr/day} \times 1 \text{ day/yr} \times 1 \text{ yr}}{8760 \text{ hrs/yr} \times 1 \text{ yrs}} = 0.009 \mu g/m^3 \]

17
Ingestion of food exposure
Derived from Environmental Health Risk Assessment Guidelines (enHealth, 2012)

\[ I_{\text{food}} = \frac{C_{\text{food}} \times AoF \times IGR \times EF \times ED}{365 \times AT \times BW} \]

\( I_{\text{food}} (\mu g/kg/day) = \) ingestion dose of food exposure

IGR (kg/day) = ingestion rate; 0.5 kg/day; the ingested rate of food for an adult is 1.4 kg/day (enHealth, 2012); however, it was considered for the present case, the 1/3 of that rate due to the fact that it is assumed as part of the worst case scenario intake of food only at lunch time.

The highest concentration on surface of food: \( C_{\text{food}} = 0.06 \text{mg/kg} \); For this parameter, the Base Study used the highest detected concentration in a plant sample test which is 0.8 mg of Cr VI per kg of green leafy vegetables exposed to Cr VI (dry weight) during a day. However, for the worst scenario in our case, it is considered that the food would be bought and ingested in a maximum period of 2 hours reducing the deposition of Cr VI in the food. Therefore, by applying a partitioning coefficient, it results a Cr VI concentration of 0.06 mg/kg of food ingested.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Adult</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration on food</td>
<td>( C_{\text{food}} ) (mg/kg)</td>
<td>0.06</td>
</tr>
<tr>
<td>Oral absorption factor</td>
<td>AoF   (unitless)</td>
<td>1</td>
</tr>
<tr>
<td>Ingestion rate</td>
<td>IGR (kg/day)</td>
<td>0.5</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td>EF (days/yr)</td>
<td>1</td>
</tr>
<tr>
<td>Exposure duration</td>
<td>ED (yrs)</td>
<td>1</td>
</tr>
<tr>
<td>Body weight</td>
<td>BW (kg)</td>
<td>70</td>
</tr>
<tr>
<td>Averaging time</td>
<td>AT₁ (yrs)</td>
<td>82</td>
</tr>
<tr>
<td></td>
<td>AT₂ (yrs)</td>
<td>1</td>
</tr>
</tbody>
</table>

Non-carcinogenic effects:

\[ I_{\text{food}} = \frac{0.06 \text{mg/kg} \times 1 \times 0.5 \text{kg/day} \times 1 \text{day/year} \times 1 \text{yr}}{365 \text{days/yr} \times 82 \text{yrs} \times 70 \text{kg}} = 1 \times 10^{-8} \text{mg/kg/day} \]

Carcinogenic effects:

\[ I_{\text{food}} = \frac{0.06 \text{mg/kg} \times 1 \times 0.5 \text{kg/day} \times 1 \text{day/year} \times 1 \text{yr}}{365 \text{days/yr} \times 1 \text{yr} \times 70\text{kg}} = 1 \times 10^{-6} \text{mg/kg/day} \]
Ingestion of soil exposure

Derived from Environmental Health Risk Assessment Guidelines (enHealth, 2012)

\[
I_{\text{soil}} = \frac{C_{\text{soil}} \times AoF \times IGR \times EF \times ED \times CF}{365 \times AT \times BW}
\]

\(I_{\text{soil}}\) (mg/kg/day) = ingestion dose of soil exposure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Adult</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration in soil</td>
<td>C_{\text{soil}} (mg/kg)</td>
<td>0.3</td>
</tr>
<tr>
<td>Oral absorption factor</td>
<td>AoF (unitless)</td>
<td>1</td>
</tr>
<tr>
<td>Ingestion rate</td>
<td>IGR (mg/day)</td>
<td>50</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td>EF (days/yr)</td>
<td>10</td>
</tr>
<tr>
<td>Exposure duration</td>
<td>ED (yrs)</td>
<td>1</td>
</tr>
<tr>
<td>Conversion Factor</td>
<td>CF (unitless)</td>
<td>(10^{-6})</td>
</tr>
<tr>
<td>Body weight</td>
<td>BW (kg)</td>
<td>70</td>
</tr>
<tr>
<td>Averaging time</td>
<td>AT₁ (yrs)</td>
<td>82</td>
</tr>
<tr>
<td></td>
<td>AT₂ (yrs)</td>
<td>1</td>
</tr>
</tbody>
</table>

Non-carcinogenic effects:

\[
I_{\text{soil}} = \frac{0.3\, \text{mg/kg} \times 1 \times 50\, \text{mg/day} \times 10\, \text{days/yr} \times 1\, \text{yr} \times 10^{-6}}{365\, \text{days/yr} \times 82\, \text{yrs} \times 70\, \text{kg}} = 7 \times 10^{-11} \, \text{mg/kg/day}
\]

Carcinogenic effects:

\[
I_{\text{soil}} = \frac{0.3\, \text{mg/kg} \times 1 \times 50\, \text{mg/day} \times 10\, \text{days/yr} \times 1\, \text{yr} \times 10^{-6}}{365\, \text{days/yr} \times 1\, \text{yr} \times 70\, \text{kg}} = 6 \times 10^{-9} \, \text{mg/kg/day}
\]
Inhalation of dust exposure

Derived from Environmental Health Risk Assessment Guidelines (enHealth, 2012)

\[ I_{\text{dust}} = \frac{C_{\text{dust}} \times IR \times ET \times EF \times ED}{CF \times AT \times BW \times PEF} \]

\[ I_{\text{dust}} \text{ (mg/kg/day)} = \text{inhaled dose of dust exposure} \]

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Adult</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration in dust</td>
<td>( C_{\text{dust}} ) (mg/m(^3))</td>
<td>0.011</td>
</tr>
<tr>
<td>Inhalation rate</td>
<td>( IR ) (m(^3)/day)</td>
<td>15</td>
</tr>
<tr>
<td>Exposure time</td>
<td>( ET ) (hrs/day)</td>
<td>3</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td>( EF ) (days/yr)</td>
<td>10</td>
</tr>
<tr>
<td>Exposure duration</td>
<td>( ED ) (yrs)</td>
<td>1</td>
</tr>
<tr>
<td>Conversion Factor</td>
<td>( CF ) (hrs/yr)</td>
<td>8780</td>
</tr>
<tr>
<td>Body weight</td>
<td>( BW ) (kg)</td>
<td>70</td>
</tr>
<tr>
<td>Particle emission factor</td>
<td>( PEF ) (m)</td>
<td>( 10^6)</td>
</tr>
<tr>
<td>Averaging time</td>
<td>( AT_1 ) (yrs)</td>
<td>82</td>
</tr>
<tr>
<td></td>
<td>( AT_2 ) (yrs)</td>
<td>1</td>
</tr>
</tbody>
</table>

Non-carcinogenic effects:

\[ I_{\text{dust}} = \frac{0.011 \text{mg/m}^3 \times 15 \text{m}^3/\text{day} \times 3 \text{hrs/day} \times 10 \text{days/yr} \times 1 \text{yr}}{8760 \text{hrs/yr} \times 82 \text{yrs} \times 70 \text{kg} \times 10^6 \text{m}} = 1 \times 10^{-13} \text{ mg/kg/day} \]

Carcinogenic effects:

\[ I_{\text{dust}} = \frac{0.011 \text{mg/m}^3 \times 15 \text{m}^3/\text{day} \times 3 \text{hrs/day} \times 10 \text{days/yr} \times 1 \text{yr}}{8760 \text{hrs/yr} \times 1 \text{yr} \times 70 \text{kg} \times 10^6 \text{m}} = 8 \times 10^{-12} \text{ mg/kg/day} \]
Ingestion after contact with surface exposure

The formula was from the base study in form of US EPA (EPA, 2013) because the enhealth guideline do not provide formula in this aspect

\[
I_{\text{contact}} = \frac{C_{\text{surface}} \times SAI \times SAF \times CF \times TE \times fdo \times fgi \times EF \times ED}{365 \times AT \times BW}
\]

\[
I_{\text{surface}} \text{ (\(\mu g/\text{kg/day}\))} = \text{ingestion of surface contact exposure}
\]

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Adult</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration on surface</td>
<td>(0.001) mg/cm(^2)</td>
<td>Base study (Frangos, 2011)</td>
</tr>
<tr>
<td>Surface area on hand</td>
<td>600</td>
<td>Base study (Frangos, 2011)</td>
</tr>
<tr>
<td>Fraction of Surface area</td>
<td>SAF (unitless) 10%</td>
<td>Base study (Frangos, 2011)</td>
</tr>
<tr>
<td>Hand to mouth behavior</td>
<td>CF (events/day) 4</td>
<td>Base study (Frangos, 2011)</td>
</tr>
<tr>
<td>Transfer efficacy from surface to skin</td>
<td>TE (unitless) 0.1</td>
<td>Base study (Frangos, 2011)</td>
</tr>
<tr>
<td>Fraction transferred from dermal to oral</td>
<td>fdo (unitless) 1</td>
<td>Assumed that all the Cr VI on skin can be transferred to the mouth</td>
</tr>
<tr>
<td>Fractional gastrointestinal absorption</td>
<td>fgi (unitless) 1</td>
<td>Oral absorption assumed 100%</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td>EF (days/yr) 10</td>
<td>Assumed to be converted to Chromium III after 10 days of exposure</td>
</tr>
<tr>
<td>Exposure duration</td>
<td>ED (yrs) 1</td>
<td>Assumed 1 year</td>
</tr>
<tr>
<td>Body weight</td>
<td>BW (kg) 70</td>
<td>Adult lifetime average weight (enHealth, 2012)</td>
</tr>
<tr>
<td>Averaging time</td>
<td>AT(_1) (yrs) 82</td>
<td>Non-carcinogenic effects (enHealth, 2012)</td>
</tr>
<tr>
<td></td>
<td>AT(_2) (yrs) 1</td>
<td>Carcinogenic effects</td>
</tr>
</tbody>
</table>

Non-carcinogenic effects:

\[
I_{\text{surface}} = \frac{0.0011 \text{mg/cm}^2 \times 600 \text{cm}^2 \times 10\% \times 4 \text{events/day} \times 0.1 \times 1 \times 1 \times 10 \text{days/yr} \times 1 \text{yr}}{365 \text{days/yr} \times 62 \text{yrs} \times 70 \text{kg}}
\]

\[
= 1 \times 10^{-7} \text{mg/kg/day}
\]

Carcinogenic effects:

\[
I_{\text{surface}} = \frac{0.0011 \text{mg/cm}^2 \times 600 \text{cm}^2 \times 10\% \times 4 \text{events/day} \times 0.1 \times 1 \times 1 \times 10 \text{days/yr} \times 1 \text{yr}}{365 \text{days/yr} \times 1 \text{yr} \times 70 \text{kg}}
\]

\[
= 1 \times 10^{-5} \text{mg/kg/day}
\]
APPENDIX D: Threshold Risk Estimation

Threshold risk estimation

For each one of the five pathways, the HQ was calculated and then all the HQs were summed to obtain the HI.

For threshold risk estimation, the Temporary Emergency Exposure Limit (TEEL) and Minimal Risk Level (MRL) were used as threshold values. TEEL (9.62 μg/m³) established by US Department of Energy was used to evaluate the immediate risk of exposure to Cr VI in the air and to Cr VI in the dust resuspended. MRL (5 x 10⁻³ mg/kg/day) established by US Center for Disease Control and Prevention was used to assess the immediate risk from oral exposure to Cr VI via ingestion of food, ingestion of soil contaminated with Cr VI, and ingestion of Cr VI due to contact with contaminated surfaces. The formulas and calculations for threshold risk estimation are shown in appendix D and summarized in Figure D1.

![Figure D1. Hazard Quotient for Each Pathway and Hazard Index](image.png)

The enhealth (2012) formula used to calculate the threshold risk estimation is shown below.

\[
\text{Hazard quotient (HQ)} = \frac{\text{Exposure concentration (ug/m}^3\text{)}}{\text{Threshold TRV (ug/m}^3\text{)}}
\]

Hazard quotient of exposure to Cr VI in the air

\[
HQ \text{ of aerosol inhalation} = \frac{9 \times 10^{-3} \text{ (ug/m}^3\text{)}}{9.62 \text{ (ug/m}^3\text{)}} = 1 \times 10^{-3}
\]

Hazard quotient of Cr VI in the Dust Resuspended

\[
HQ \text{ of dust Resuspended} = \frac{8 \times 10^{-12} \text{ (ug/m}^3\text{)}}{9.62 \text{ (ug/m}^3\text{)}} = 8 \times 10^{-13}
\]
Hazard quotient of Ingestion of Food with Cr VI

\[ HQ_{\text{food ingestion}} = \frac{1 \times 10^{-6} \text{ (mg/kg/day)}}{5 \times 10^{-3} \text{ (mg/kg/day)}} = 2 \times 10^{-4} \]

Hazard quotient of Ingestion of Cr VI due to contact with contaminated surfaces

\[ HQ_{\text{surface contact}} = \frac{1 \times 10^{-5} \text{ (mg/kg/day)}}{5 \times 10^{-3} \text{ (mg/kg/day)}} = 2 \times 10^{-3} \]

Hazard quotient of ingestion of soil contaminated with Cr VI

\[ HQ_{\text{ingestion soil}} = \frac{6 \times 10^{-9} \text{ (mg/kg/day)}}{5 \times 10^{-3} \text{ (mg/kg/day)}} = 1 \times 10^{-6} \]

Calculation of Hazard Index

\[
\text{Hazard Index (HI)} = \sum \text{HQ of aerosol inhalation} + \text{HQ of dust Resuspended} \\
+ \text{HQ of food ingestion} + \text{HQ of surface contact} + \text{HQ of ingestion soil}
\]

\[ \text{Hazard Index (HI)} = 4 \times 10^{-3} < 1 \]

The hazard index estimated for the proposed project was \( 3 \times 10^{-3} \) which being less than 1 implies no need of a detailed risk assessment.
APPENDIX E: Non-Threshold Risk Estimation

Non-Threshold risk estimation

For no-threshold risk estimation, it was used a cancer slope factor (CSF) and a Unit Risk Factor (URF). The CSF (0.5 (mg/kg/day)^{-1}) determined by US EPA was used to estimate the risk of cancer for oral exposure to Cr VI, and the URF (4.2 x 10^2 (mg/m^3)^{-1}) determined by WHO was used to estimate the risk of cancer for inhalation exposure to Cr VI. CSF and URF are upper 95th confidence limit estimates.

![Graph showing risk calculation](image)

Figure E2. Increased lifetime cancer risk for each pathway and total increased lifetime cancer risk.

The enhealth (2012) formulas used to calculate the non-threshold risk estimation is shown below.

\[
ILCR = \text{Exposure Concentration (} \mu g/m^3\text{)} \times \text{URF (} \mu g/m^3\text{)}^{-1}
\]

\[
ILCR = \text{Exposure Concentration (} mg/kg/d\text{)} \times \text{CFS (} mg/kg/d\text{)}^{-1}
\]

ILCR due to Aerosol Inhalation (ILCR ai)

\[
ILCR \text{ of aerosol inhalation} = 1 \times 10^{-4} \mu g/m^3 \times 4.2 \times 10^{-2} \mu g/m^3 = 5 \times 10^{-6}
\]

ILCR due to Dust Resuspended (ILCRd)

\[
ILCR \text{ of Dust Resuspended} = 1 \times 10^{-13} \mu g/m^3 \times 4.2 \times 10^{-2} \mu g/m^3 = 4 \times 10^{-15}
\]

ILCR due to Food Contaminated (ILCRfc)

\[
ILCR \text{ due to food contaminated} = 1 \times 10^{-8} (mg/kg/d) \times 0.5 (mg/kg/d)^{-1} = 7 \times 10^{-9}
\]

ILCR due to Incidental Surface Contact (ILCRisc)
\[ \text{ILCR due to incidental surface contact} = 1 \times 10^{-7} \text{ (mg/kg/d)} \times 0.5 \text{ (mg/kg/d)}^{-1} = 6 \times 10^{-8} \]

ILCR due to ingestion of soil contaminated (ILCRsc)

\[ \text{ILCR due to ingestion of soil} = 7 \times 10^{-11} \text{ (mg/kg/d)} \times 0.5 \text{ (mg/kg/d)}^{-1} = 4 \times 10^{-11} \]

Total Increased Lifetime Cancer Risk

Total increased lifetime cancer risk (ILCR_{total})

\[ = \text{ILCRae + ILCRdr + ILCRfc + ILCRisc + ILCRsc} \]

\[ \text{ILCR}_{total} = 5 \times 10^{-6} \]

The target risk level in Australia is \( 1 \times 10^{-6} \) (enhealth, 2012); as result, increased lifetime cancer risk of the proposed project is unacceptable because the value estimated was \( 5 \times 10^{-6} \).
References


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