Modelling the health risks of exposure to respirable crystalline silica from hydraulic fracturing operations in the USA shale plays

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Received: May 22, 2015 Accepted: July 7, 2015 Online Published: July 28, 2015

DOI: 10.5430/jbei.v1n1p25 URL: http://dx.doi.org/10.5430/jbei.v1n1p25

Abstract

Respirable crystalline silica (RCS) is a known human carcinogen and a contaminant of potential concern. Proppants are used during the process of well stimulation (hydraulic fracturing) as additives in the fluid cocktail and sand is often used as a proppant which contains high percentage of silica determined by the quartz content. Empirical occupational exposure risk models were employed in this study to assess the potential health consequences from chronic RCS exposures based on RCS data from NIOSH and risk assessment formulas. Evaluating the lifetime (LT) excess cancer risk (LCR) potential, based on a risk target of 10\(^{-5}\), the job titles that are likely to experience any substantial potential effect of cancer induction are the sand mover (LCR = 16.1 \times 10^{-5}) and transfer belt (LCR = 19.2 \times 10^{-5}) operators. The sand truck driver and data Van operators are among the job functions with a cumulative disease burden of 7.2% that are unlikely to be affected by < 2% carcinogenic disease burden. The chemical truck, sand mover and transfer belt (T-belt) operators may potentially be at risk of other occupational nonmalignant respiratory diseases with hazard quotient (HQ) of 0.65, 1.79, and 2.13 respectively. It is recommended that continuous occupational health monitoring of potentially exposed workers should be included as part of the project plan and the engineering risk controls that have been put in place should be ranked to highlight the effectiveness of any risk reduction/prevention methodology employed.

Key words
Silicosis, Respirable crystalline silica, Hydraulic fracturing, Energy, Disease, Cancer

1 Introduction

The earth’s composition is made up of about 75% silicon and oxygen making them the most abundant elements. The chemical combination of a silicon atom and dual oxygen atoms form a compound called silica mineral (SiO\(_2\)). This is a noncombustible material with a melting point of 16,000 C (29,120 F); SiO\(_2\) is odorless and has no representative color \([1]\). SiO\(_2\) molecules are ordered in a continuous repeating three-dimensional pattern, which align to form a crystalline structure. Crystalline SiO\(_2\) exist in nature with three stable polymorphs—quartz, -cristobalite and tridymite. Quartz sub-polymorph known as the alpha quartz is the most abundant of all crystalline SiO\(_2\) polymorphs. Respirable crystalline silica (RCS) from occupational sources are classified as: Group 1 substances, by IARC, which are carcinogenic to humans \([2]\), representing health risk factors to exposed workers.
The human physiology reacts to fibrogenic dusts such as RCS due to its biological toxicity. Occupational definition of respirable dust ($\text{Res}_{\text{Dust}}$) entails particles categorized having potential thoracic effect, with sizes less than 5 μm. ACGIH, 2004 and the ISO 7708 method [3], further define $\text{Res}_{\text{Dust}}$, as inhalable substance with 50% cut-point at < 5 μm aerodynamic diameter, shown in Figure 1 [4, 5]. These minute materials have the capabilities to evade the respiratory tract and penetrate deeply directly into the lungs, causing disabilities and fatalities in various exposed workers worldwide. Chronic exposure to high concentrations of RCS affects the lungs, leading to the widespread accumulation of fibrous tissues which eventually result to silicosis and lung cancer [6, 7].

There is a wide range of applications of SiO₂ in the industrial and manufacturing processes, including glass, cement, abrasives, electronics etc. [8]. Occupational exposures to RCS have been traditionally in the coal, metal mining, agriculture and highway construction industries [9], recent technologically advancement in the petroleum industry has increased workers exposure to RCS during oil and gas extraction.

The hydraulic fracturing technique ($HF_{\text{tech}}$) involves stimulating reservoirs of tight formations for optimal recovery of oil or gas, creating cracks (fractures) in the rock matrix and allowing a free flow of oil or gas through the wellbore to the surface. $HF_{\text{tech}}$ is becoming more prevalent in the petroleum extractive industry as the demand for energy increases worldwide. Modern improvements in technology for unconventional oil and gas reserves have made deep formations with very low permeability accessible through 3-D microseisimcs and directional drilling coupled with pressure pumping, a process called high-volume- hydraulic fracturing technique ($HVHF_{\text{tech}}$). The importance of the $HF_{\text{tech}}$ cannot be overemphasized; it has been used to recover over 600 trillion cubic feet (Tcf) of natural gas and 7 billion barrels of crude oil since the technology was developed approximately 70 years ago [10, 11]. The United States has a natural gas reserve estimate of approximately 1800 Tcf which are technically recoverable and estimated to sufficiently supply energy to the United States for upwards of 116 years [12].

A perforating gun is typically passed down through the directional drilled hole and then detonated, after which a cocktail of fracturing fluids are pumped into the formation at very high pressure to further extend the cracks and prevent it from shutting in. The hydraulic fluids required for the stimulation process are primarily made up of water (≈ 93%), proppants – mostly silica sand which is ≈ 6% in total volume and emulsifiers, acids, inhibitors, cross link breakers and other components (≈ 1%) (see Figure 2) [13]. The use of proppant is essential in the process since the open fractures are kept open for fluid transport through the formation, therefore requiring large proportion of sand with high quartz content. The quantity of proppant required to complete a fracturing job largely depends on the number of stages that the $HF_{\text{tech}}$ is required in the well. The mechanical process involved with preparing the sand for use, generates RCS dusts that are of potential occupational health concerns to the exposed field workers [14]. Several other potential risks are associated with the use of the $HF_{\text{tech}}$ [11, 15, 16].
There are currently approximately 35,000 wells using the $HF_{tech}$ for well improvements each year and a total of over 1,000,000 wells hydraulically fractured since the inception of the $HF_{tech}$ [12]. The number of workers employed in the petroleum industry has increased exponentially in the past seven years, at the end of 2012, over 193,000 extraction workers, 90,000 drilling workers and 102,000 field support workers were hired and the workforce statistics for the oil and gas extraction industry grew to over 202,000 as of December 2013 [17]. Fifty percent of these workers have support responsibilities that are susceptible to the hazard of RCS exposures.

Occupational exposures to RCS are linked with pulmonary diseases affecting humans [18-20]. The total number of workers exposed to RCS during the application of $HF_{tech}$ is largely estimated. The population potentially at risk of RCS is approximately 100,000, based on the data of employed field support technicians in the petroleum industry at the end of December 2013 [17]. The exposure risks are higher for the sand mover and transfer belt operators. Other job titles have lesser exposures, but the chronic and continuous exposure to the operational RCS, is of potential concern.

### 1.1 Pathophysiology of RCS Exposures

The initial physiological reaction to the inhalation of RCS include the irritation of the respiratory tracts and lungs inflammation, acute exposures could result to silicoproteinosis [21], while other conditions, such as coughing, asphyxiation, protracted airflow restriction and consequently, chronic bronchitis, pathologic emphysema [18, 22], pulmonary alveolar lipoproteinosis [23] and fibrotic lesions [24], would develop due to the chronic exposures. The most commonly associated disease due to RCS exposure is called silicosis, which is typified by fibrotic lesions and or the occurrence of histologically distinct silicotic lumps in the lungs causing oxygen intake restrictions. The silicotic development mechanism is illustrated in Figure 3, the RCS dusts infiltrate into the lower respiratory tract (LRT), since the alveolar macrophages (AM) cannot digest the dust particles, they are rapidly ingested into phagosomes, which according to Ding et al. [25] would result to severe AM toxicity in the lungs from the release of reactive oxygen species (ROS).

Continued exposures to RCS consequently lead to increased protracted inflammatory episodes (phagocytosis), which allows the dust to penetrate the AM and killing the epithelial cells (Type 1), this excites the fibroblasts through the epithelial cell repair mechanism [26], which facilitates the production and spread insoluble fibrous proteins (collagen) that are present in connective tissues. Cytokines are subsequently released from the interaction between the phagosomes and lysosomes. The ROS initiates the disease by inducing inflammations since they function as growth agents thereby making it possible for neutrophils to migrate to the inflammation region. The distinctive scarring patterns associated with silicotic conditions are set by the concentric deposition of collagen fibers on the lungs [27].

### 1.2 Types of effects

**Simple Silicosis** is classified by visible opacities at the upper lungs that are rounded and stand alone. The disease progression is known to occur with proliferation of the opacities and enlargement in sizes.
**Conglomerated Nodular Silicosis** occurs as the progression of restrictive diseases, due to the merging of silicotic nodules to form an opaque bilateral massive fibrotic body, usually represented as angel’s wings on a radiographic film of chest x-rays. This condition could also result to emphysema, dyspnea, cardiopulmonary arrest, silicotuberculosis, scleroderma and kidney disease, Caplan’s syndrome.

**SiO₂-Related Lung Cancer:** Lung cancer might be induced in exposed workers either with preexisting conditions of silicosis or not. SiO₂ is known to induce cancer in humans, therefore the direct chronic exposure to RCS and the associated accumulative effect might be accountable for elevated occupational risk of lung cancer. Data from several individual epidemiological studies, present significant statistically associations between lung cancer mortality and RCS exposures \(^{28,29}\).

**Nonmalignant Respiratory Disease (NMRD):** Emphysema appears as an irregular air spaces expansion with damaging alterations to the walls of the alveolar \(^{18}\). Immunologic abnormalities that can potentially burden humans with silicosis and additional forms of chronic renal and autoimmune ailments, have been described in other relevant literature; which include ataxic sensory neuropathy, rheumatoid arthritis, dermatomyositis, monoclonal gammopathy, glomerulonephritis \(^{30,31}\).

The silica particles can also result to extrapulmonary silicosis \(^{32}\), when migrated from the lungs in experimental animals to different other organs such as; the kidney, liver, spleen etc. \(^{33}\). In humans however, peritoneal silicosis was diagnosed as described in the study by Tschopp et al. \(^{34}\).

Fibrotic lesions (scarring) in the lungs are diagnosed as visible opacities on computerized tomography or from chest x-ray films. The International Labor Organization standardized the classification of these opacities based on the characteristic density, shape and size that correlate with the effect on the lungs \(^{35}\).

In this study, the potential occupational health effects from chronic RCS exposures on field workers using the \(H_{F_{\text{tech}}}\) were assessed and a prediction of the cancer risk and the hazard quotient were made based on population assumptions.

![Figure 3. Theoretical Inflammation-based mechanism for carcinogenic RCS on Humans](http://jbei.sciedupress.com)
2 Methodology

2.1 Exposed populations and exposure levels

The job titles and exposure levels at risk of potential adverse health complications as a result of RCS were identified in a NIOSH study \[14\]. The study evaluated the occupational exposures to RCS during the use of HF\textsubscript{tech}. Eleven sites with HF\textsubscript{tech} operations were investigated and one hundred and eleven (111) samples were obtained from the personal breathing zone (PBZ) of the field exposed workers during a complete 12 hour shift. Sample collection techniques and analyses were presented in details in the published literature \[14\].

Each well site had about 12 dedicated workers in place for the implementation of the HF\textsubscript{tech}. The proppant delivery, distribution and mixing were usually handled by 4-6 operators. The sand mover is a conveyor system that receives the discharged sand from the truck and subsequently connected to the blender. Both the sand mover and the blender are controlled by operators that maintain constant communication to determine the characteristic nature of the sand before it is mixed with other fluids and pumped into the wellbore. In this study, for the purpose of assessing the potential health effects of RCS exposures on support field workers, an estimation of the average number of workers directly exposed to equal RCS exposure throughout the shift for each well was chosen based on the exposure assessment from the NIOSH study \[14\].

The Occupational Safety and Health Administration (OSHA) (OSHA 29 CFR 1910.1000) recommended permissible exposure limit (PEL) for all samples with detectable quartz fraction were calculated based on the medium silica percentage of 53% in the PBZ samples collected, the PEL in this study was determined as 0.18 mg/m\textsuperscript{3} using the OSHA PEL equation Table 1 \[36\]. For extended work times more than the standard 8-hr shift, there are various numerical models that are applicable for adjusting the exposure standards, which include the Brief and Scala Model \[37\], OSHA Model \[38\] and Pharmacokinetic Model \[39\]. The Brief and Scala daily correction formula (equation 1) for exposure TWA was used to adjust the values obtained by Esswein \textit{et al.} \[14\] since the formula compensates for both the exposure and recovery times and the 8-hr adjusted was not included in the study;

\[
\varphi = \frac{8 \times (24-h) \times \text{Exposure Standard (8-hr TWA)}}{16 \times h}
\]

(1)

Where \(\varphi = \) TWA corrected exposure standard; \(h = \) total number of hours on the job per day assume 40-hr work week. Table 1 shows the adjusted exposure standard median values and the severity values modified from Esswein \textit{et al.} \[14\] and the equivalent severity values are illustrated in Figure 4.

![Figure 4. PBZ Severity based on OSHA calculated PEL data \[14\]](image)
Table 1. Calculated and guidance RCS exposure values

<table>
<thead>
<tr>
<th>Occupational Exposure Guidelines</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Component</td>
<td>Units</td>
<td>OSHA PEL</td>
<td>8-HR TWA</td>
<td>10-HR TWA</td>
<td>ACGH TLV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RCS</td>
<td>mg/m³</td>
<td>10 mg/m³</td>
<td>% SiO₂ + 2</td>
<td>0.025</td>
<td>0.05</td>
<td></td>
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</tbody>
</table>

The adjusted OSHA PEL exposure and severity value (Adapted from Esswein et al. [14])

| Job Titles |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
|------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| #Sample    | 16  | 3   | 2   | 5   | 3   | 1   | 1   | 1   | 4   | 10  | 1   | 50  | 6   | 7   | 1   | 1   | 111 |     |     |     |     |     |     |     |     |     |     |     |     |
| Median TWA | 0.10| 0.14| 0.04| 0.04| 0.04| 0.04| 0.02| 0.01| 0.02| 0.06| 0.04| 0.38| 0.45| 0.06| 0.01| 0.11|     |     |     |     |     |     |     |     |     |     |     |
| GM         | 0.09| 0.12| 0.04| 0.07| 0.05| 0.04| 0.02| 0.01| 0.02| 0.05| 0.04| 0.26| 0.33| 0.05| 0.01| 0.12|     |     |     |     |     |     |     |     |     |     |     |
| * mg/m³    | 7.34| 10.01| 3.10| 3.17| 4.97| 3.10| 1.51| 0.94| 1.44| 4.39| 2.95| 27.43| 32.62| 4.03| 0.50| 7.85|     |     |     |     |     |     |     |     |     |     |     |
| **Severity | 0.41| 0.56| 0.17| 0.18| 0.28| 0.17| 0.08| 0.05| 0.08| 0.24| 0.16| 1.52| 1.81| 0.22| 0.03| 0.44|     |     |     |     |     |     |     |     |     |     |     |

Note: GM = Geometric Mean, * 8-hr TWA adjusted, ** Severity = Median TWA/ OSHA PEL-TWA; if severity > 1, > OSHA PEL. A=Blender Operator, B=Chemical Truck Operator, C=Fueler, D=Hydration Unit Operator, E=Mechanic, F=Operator, Data Van, G=Pump Truck Operator, H=QC Tech, I=Roving Operator, J=Sand Coordinator, K=Sand Truck Driver, L=Sand Mover Operator, M=T-belt Operator, O=Water Tank Operator

The highest median occupational exposures of RCS after the adjustment to the standard 8-hr TWA, were the (L) Sand Mover and (M) T-belt Operators, with median exposures of 27.43 mg/m³ and 32.62 mg/m³ respectively, which is consistent with the NIOSH study [14].

2.2 Theoretical occupational carcinogenic risk and hazard quotient

The inhalation reference exposure ($\delta$) value of 0.003 mg/m³ [3, 4] was used in this study to predict the total LT carcinogenic risk ($\psi$) in the hydraulic fracturing industry, provided the worker continuously inhaled the RCS dust at the calculated dust concentration ($\gamma$) during the exposure period. The product summation of the total occupational exposure to the calculated median 8-hr TWA for each job title ($\gamma$) throughout the employment period for the job ($T_j$) gives the cumulative LT dust concentration intake ($C_{DC}$) as given in equation 2:

$$C_{DC} = \sum_{j=1}^{n} (C_j \times T_j) = mg/m³ - yrs$$

Where $T_j$ was calculated using equation 3:

$$T_j = \left( \frac{EF \times ED}{LT} \right) = yrs$$

The exposure frequency (EF) and worker’s lifetime (LT) were assumed to be 45 years and 70 years respectively. While the exposure duration (ED) was calculated as: $\left[ \left( \frac{8}{24} \text{ hours} \right) \times \left( \frac{5}{7} \text{ days} \right) \times \left( \frac{48}{52} \text{ weeks} \right) \right]$. Therefore; the potential excess lifetime carcinogenic risk ($L_{CR}$) using the $HF_{tech}$ was determined using equation 4:

$$L_{CR} = C_{DC} \times I_r, \; L_{CR} > 10^{-4} = Risk$$

Exposure effects with the potentials of non-carcinogenic health complications can be estimated using the hazard quotient equation (eq. 5).
The potential occupational carcinogenic and non-carcinogenic risks from continuous exposure to RCS through the use of the $H_{F_{tech}}$ are presented in Table 2. These risk outcomes did not take into account the synergistic effects from other potentially deleterious substances the workers may have been exposed to at the same period as the RCS exposure. The results presented herein also assumed that the workers continued on the same job with constant exposure levels until the end of the follow-up year.

### Table 2. Potential carcinogenic and non-carcinogenic occupational risk due to RCS dust

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>G</th>
<th>H</th>
<th>I</th>
<th>J</th>
<th>K</th>
<th>L</th>
<th>M</th>
<th>N</th>
<th>O</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td># Samples</td>
<td>16</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>10</td>
<td>1</td>
<td>50</td>
<td>6</td>
<td>7</td>
<td>1</td>
<td>111</td>
</tr>
<tr>
<td>Median TWA</td>
<td>0.10</td>
<td>0.14</td>
<td>0.04</td>
<td>0.04</td>
<td>0.07</td>
<td>0.04</td>
<td>0.02</td>
<td>0.01</td>
<td>0.02</td>
<td>0.06</td>
<td>0.04</td>
<td>0.38</td>
<td>0.45</td>
<td>0.06</td>
<td>0.01</td>
<td>0.11</td>
</tr>
<tr>
<td>$C_{DC}$</td>
<td>1.44E-02</td>
<td>1.96E-02</td>
<td>6.08E-03</td>
<td>6.22E-03</td>
<td>9.75E-03</td>
<td>6.08E-03</td>
<td>2.97E-03</td>
<td>1.84E-03</td>
<td>2.83E-03</td>
<td>8.62E-03</td>
<td>5.79E-03</td>
<td>5.33E-02</td>
<td>6.40E-02</td>
<td>7.91E-03</td>
<td>9.89E-04</td>
<td>2.11E-01</td>
</tr>
<tr>
<td>$L_{CB}$</td>
<td>4.32E-05</td>
<td>5.89E-05</td>
<td>1.82E-05</td>
<td>1.86E-05</td>
<td>2.92E-05</td>
<td>1.82E-05</td>
<td>8.90E-06</td>
<td>5.51E-06</td>
<td>8.48E-06</td>
<td>2.59E-05</td>
<td>1.74E-05</td>
<td>1.61E-04</td>
<td>1.92E-04</td>
<td>2.37E-05</td>
<td>2.97E-06</td>
<td>6.33E-04</td>
</tr>
<tr>
<td>H-Q&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.48</td>
<td>0.65</td>
<td>0.20</td>
<td>0.21</td>
<td>0.32</td>
<td>0.20</td>
<td>0.10</td>
<td>0.06</td>
<td>0.09</td>
<td>0.29</td>
<td>0.19</td>
<td>1.79</td>
<td>2.13</td>
<td>0.26</td>
<td>0.03</td>
<td>7.03</td>
</tr>
</tbody>
</table>

### Table 3. Likelihood of Occurrence of LCR based on Occupational RCS using the $H_{F_{tech}}$

<table>
<thead>
<tr>
<th>Job Title</th>
<th>O</th>
<th>H</th>
<th>I</th>
<th>G</th>
<th>K</th>
<th>C</th>
<th>F</th>
<th>D</th>
<th>N</th>
<th>J</th>
<th>E</th>
<th>A</th>
<th>B</th>
<th>L</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>LCR</td>
<td>0.3</td>
<td>0.6</td>
<td>0.8</td>
<td>0.9</td>
<td>1.7</td>
<td>1.8</td>
<td>1.8</td>
<td>1.9</td>
<td>2.4</td>
<td>2.6</td>
<td>2.9</td>
<td>4.3</td>
<td>5.9</td>
<td>16.1</td>
<td>19.2</td>
</tr>
<tr>
<td>Likelihood of Occurrence</td>
<td>Rare Possible</td>
<td>$\sum = 2.6%$</td>
<td>Unlikely Possible</td>
<td>$\sum = 7.2%$</td>
<td>Possible</td>
<td>$\sum = 7.9%$</td>
<td>Likely</td>
<td>$\sum = 45.6%$</td>
<td></td>
<td></td>
<td></td>
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The workers dedicated to job functions L and M were found to have the highest susceptibility of developing cancer after retirement due to the chronic RCS exposures. The model predicts about 2 cancer cases per 10,000 workers for both job functions (Sand-mover and Transfer-belt operators) on average. Based on the level of exposure of other field workers to RCS, the potential risk of cancer development after a follow-up period of 45 years is minimal compared to L and M jobs.
The water tank operator job function will have the least effect of carcinogenicity \( (3.0 \times 10^6) \) arising from hydraulic fracturing RCS. The cumulative dust exposure may result to other health complications other than lungs cancer. The potential effects of RCS leading to non-carcinogenic human health problems were assessed using the hazard quotient (HQ); which shows the same trend as the lifetime cancer risk, based on the overall RCS exposure. HQ > 1 poses significant health risk to humans, at the current level of RCS exposure, only the sand mover operator and transfer belt (T-belt) operator may potentially be at risk of other occupational nonmalignant respiratory diseases with HQ of 1.79, and 2.13 respectively.

Occupational exposures to carcinogenic materials are undesirable; the model in this study presents quantitative risk to workers directly in contact with RCS contained in the fracturing sand based, on the median permissible exposure limits (PEL) calculated in compliance with OSHA regulation. In similar studies, acute silicosis symptoms have been found to develop due to short time exposure to high RCS, however, other epidemiological studies have shown that the development of chronic symptoms have occurred even long after occupational RCS exposures ended \[40\]. There is approximately 1% chance for workers with an exposure duration of 45 years to develop other non-carcinogenic diseases from occupational exposure to RCS, and 50% of these cases may develop complications due to Mycobacterium tuberculosis, consequently leading to tuberculosis disease \[41\]. Increased mortality rates have been reported from other forms of non-carcinogenic consequences of RCS exposure, such as chronic obstructive pulmonary disease (COPD) and pneumoconiosis \[42\]. Statistical significant differences have been presented in recent epidemiological data obtained from silica-exposed workers, which showed increased cases of mortality from immunologic and autoimmune ailments \[41\] and systematic impairment of the local immune function in the human lungs with amplified nuclear factor-κB activation \[43\].

Oil and gas operators must however, be concerned about every level of exposure and the associated potential effects on the health of the workers. Companies hiring workers to carry out job task such as the sand-mover and T-belt operators during the hydraulic fracturing process, should pay close attention to protecting all workers with the possibility of exposure to RCS. They must also ensure that every engineering control that can possibly and thoroughly safeguard workers health quality from harmful exposures, are properly designed and implemented.

### 4 Conclusion

In this study, the health effect descriptive epidemiologic analysis was used to appraise the potential health effects due to occupational exposures to RCS during the use of the HFtech. The sand mover operator and the transfer belt operator both have LCR values of \( 1.6 \times 10^6 \) and \( 1.9 \times 10^6 \) respectively; and HQ of 1.79 and 2.3 respectively. These values are higher than the LCR target risk level of \( 10^5 \) and HQ = 1 \[44\], therefore, these two job functions are considered to have the likelihood of disease burden occurrence. The projected risks were based on some occupational assumptions and also considering the PEL levels for each job function, although silica exposure is dose-dependent. It is quintessential to underscore the benefits obtainable from preventing workers from any potential continuous RCS dust exposures. This is substantial in reducing the potentiality of either a lifetime excess risk or other nonmalignant health conditions, such as the negative modification of polycyclic aromatic hydrocarbons (PAH) induced Cytochrome P-4501A1 (CYP1A1) actions in the lungs, causing pulmonary inflammations \[45\].

Providing more effective engineering controls, (which should begin from the design stage) for field operations during hydraulic fracturing, such as the use of alternatives measures to the pneumatic sand transfer activities, between the sand movers to the conveyor, will result to occupational exposure reduction and consequently, risk reduction/elimination. The application of substitution, elimination and administrative control methodology will bring about optimal reduction in occupational risk to workers that can be potentially exposed to the carcinogenic silica material. An example is the use of an alternative proppant material such as the resin coated sand and also reducing the amount of time a particular worker performs the task of either as a transfer belt or sand mover operator. It is suggested that effective health monitoring of
workers who may be potentially exposed to RCS and other toxic substances should be prioritized together with the implementation of operational engineering risk controls, enhanced trainings and emphasis on the proper use of respirators.

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