ALPHA FOUNDATION FOR THE IMPROVEMENT OF MINE SAFETY AND HEALTH

Final Technical Report

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1.0 Executive Summary:

Diesel exhaust and dust in both ambient air and occupational settings may cause chronic heart and lung disease. We do not know how much disease and death could be prevented among miners if long term exposures to diesel exhaust and dust exposures were reduced. We examined these questions in the extended follow-up of an existing cohort study – the Diesel Exhaust in Miners Study (DEMS II) – conducted jointly by NIOSH and the National Cancer Institute (NCI). The study was originally designed to examine lung cancer in non-metal miners. Our focus in this project was on chronic heart and lung disease mortality. It is also necessary to take account of the healthy worker survivor effect whereby healthier workers remain at work and exposed longer than less healthy co-workers. We have identified and addressed this potential bias and in doing so have avoided underestimation of effects. The data were made available to us by our collaborators at the National Cancer Institute (NCI).

Beyond the potential health effects of diesel exhaust exposure, we also examined deaths of despair (suicide and overdose) in this miner population. These tragic outcomes are greatly elevated in the mining and extraction industries compared to other occupations. We speculated that miners who leave work may be at increased risk of drug overdose or suicide particularly if they are younger when they leave work and if leaving work was the result of a chronic illness or injury, layoff, or mine closure.

We applied standard statistical methods for survival analysis to estimate hazard ratios for chronic obstructive pulmonary disease (COPD) and ischemic heart disease (IHD) mortality in relation to duration of employment, average intensity of exposure, and cumulative exposures to respirable elemental carbon (REC), a marker of diesel exhaust and respirable dust (RD). We applied these same standard methods to examine the risk of suicide and overdose in relation to the age at leaving work. We also evaluated the presence of the healthy worker survivor effect which is a common source of bias in occupational studies of chronic health outcomes. There is a range in the susceptibility of individuals to the adverse health effects of chemical exposures. If the more susceptible workers are more likely to leave work sooner to avoid more exposure, then those that remain at work, and exposed, will be a relatively heathier subset of the workers. This is referred to as the healthy worker survivor effect and leads to unintuitive results when comparing more and less exposed workers. Based on evidence that this was happening in our study of miners, we also applied a novel statistical approach to account for that potential bias. This novel method (referred to as g-formula) also allows us to answer questions about what would have happened if miners had been working under different permissible exposure limits than what was observed and provide results in the form of risk differences that are more interpretable for policy makers than hazard ratios.

Our results suggest that long term exposure to diesel exhaust and dust may modestly increase the risks of both COPD and CVD mortality in miners. We found that as duration of employment increased, the rate of COPD decreased, consistent with a healthy worker survivor effect. Our results for average intensity of exposure were more interpretable than our results for cumulative exposure which combines duration of exposure with intensity. We found hazard ratios (HR) for COPD and average intensity of REC and RD of 1.46 (95% confidence interval (CI): 1.12, 1.91) and 1.20 (95% CI: 0.96, 1.49), respectively. These HRs were associated with

a change in each exposure equal to the width of the interquartile range, 141.0 μg/m^3 for REC and 1.35 mg/m^3 for RD.

Based on results from the g-formula, reducing exposure to REC reduced the risk of both COPD and IHD. The risk ratio for COPD was 0.85, equivalent to an attributable risk due to diesel exhaust of 15%. The risk difference for COPD was on the order of 1% for eliminating REC. Both the risk difference and ratio for COPD were less for eliminating dust. IHD mortality, our results also suggest a modest reduction in risk of approximately 1%. In contrast with COPD, results for IHD suggest a larger impact for reducing dust than REC.

To better understand the drivers of suicide and overdose in mining, we examined these outcomes in relation to age at worker exit in this miner population. We observed a total of 248 suicides or overdoses from 1947 to 2015. The age adjusted rate of suicide peaked between 1980-1989 at 67.2 per 100,000 - more than double the rate in the general US male population. Since 1980, those leaving work between age 30-40 had an elevated risk of 1.46 and those who left work before age 30 had a HR for suicide or overdose of 1.94 (95% CI:1.05, 3.58) compared to those who left work after 55. We conclude that leaving work during the post-1980 decline in the US mining industry contributes to the elevated risk of suicide or overdose among male miners, particularly among younger age groups.

2.0 Problem Statement and Objective:

We know from the health effects literature on both ambient air pollution and occupational exposures that long-term exposure to high concentrations of respirable diesel exhaust and dust may cause chronic heart and lung disease. We do not know how much disease and death could be prevented among miners if the permissible exposure limit (PEL) for respirable diesel exhaust was lowered. We applied a method that allowed us to estimate the excess risk of COPD mortality in an established cohort of non-metal miners under alternative exposure limits for diesel exhaust in a format easily interpretable to policymakers. Deaths of despair due to suicides and overdoses are greatly elevated in the mining and extraction industries compared to other occupations. We speculated that miners who leave work may be at increased risk of self-injury due to drug overdose or suicide - particularly if leaving work was the result of a chronic illness or injury, layoff, or mine closure. **These questions align squarely with Priority Area 4 of the Alpha Foundation Mission, "to address the root causes of disease, injuries, and fatalities in the mining industry and, where possible, to achieve successful implementation of practical solutions derived from the research effort".**

To answer these research questions we addressed the following four Specific Aims:

Aim 1. Examine the relationships between COPD mortality and long-term exposure to diesel exhaust, measured as REC, and respirable dust (RD), based on the extended follow-up at eight non-metal mines, by applying a Cox proportional hazards model to estimate hazard ratios, adjusted for confounding by calendar year, age, and race/ethnicity.

Aim 2: Address Healthy Worker Survivor Effect (HWSE) by applying the parametric g-formula to correctly handle leaving work as a time varying confounder affected by prior exposure, when estimating the impact of reductions of diesel and dust exposures on risk of COPD and IHD mortality.

Aim 2a: Provide estimates of the number of COPD deaths avoided due to hypothetical interventions to reduce exposures below current MSHA limits for diesel exhaust.

Aim 2b: Repeat 2a with IHD as the outcome and provide estimates of the number of IHD deaths avoided due to hypothetical interventions to reduce exposures below current MSHA limits.

Aim 3. Identify the relative contributions of intensity, duration and timing of REC and dust exposure on risk of COPD mortality to identify which features of cumulative exposures are driving the risk.

Aim 3a: Fit non-linear distributed lag models for diesel exhaust and dust to characterize the relative weights of different exposure periods.

Aim 3b: Use results of Aim 3a to partition cumulative exposures and incorporate the most biologically relevant time windows of exposure into a refined g-formula. Repeat for dust.

Aim 4: Identify the work-related predictors of self-injury mortality (suicide or drug overdose) by considering layoffs, mine closures, location (underground or surface), and diesel and dust exposure as potential risk factors.

Aim 4a. Examine the impact of temporary lay-offs and partial mine closures at the eight non-metal mines during the study period on mortality rates of miners, specifically fatal self-injury (suicide and drug overdose), as well as IHD, particularly acute myocardial infarction, and stroke.

Aim 4b: If suicide appears to block some of the diesel-related risk of IHD or COPD, reassess Aim 2 to see how many deaths would have been avoided by reducing diesel exposure if we could also intervene to prevent self-injury

3.0 Research Approach:

We studied the impact of long-term exposures to two specific agents, respirable diesel exhaust and dust (particulate matter (PM)), on chronic heart and lung disease mortality in the Diesel Exhaust in Miners Study (DEMS II). Our analytic approach was rooted in a causal framework and focused on evaluating the number of deaths that could be avoided by complying with hypothetical interventions to reduce exposures below a series of specified levels. Our proposal leveraged our past (Alpha funded) work with the DEMS cohort mortality study of 12,315 miners at 8 non-metal U.S. mines (DEMS I), and took advantage of new follow-up data recently collected by our collaborators at the National Cancer Institute (NCI). With extended vital status follow-up from 1997 through 2015, we had access to more than twice the numbers of deaths due to Ischemic Heart Disease (IHD) and Chronic Obstructive Pulmonary Disease (COPD) mortality.

The additional statistical power offered us the opportunity to corroborate the generally positive results of our previous exposure-response studies with more specificity and greater

confidence. The additional follow-up data also allowed us to examine the potential impact of leaving work on self-injury – suicide and drug overdose – and the extent to which these outcomes, tragic in themselves, may also be obscuring some of the exposure-related risk of chronic disease mortality in mining populations.

To achieve the aims, we applied Cox proportional hazards models to estimate hazard ratios for each chronic disease outcome, comparing high to low exposed miners, adjusted for confounding. All Cox models included both REC and RD, with one measured as intensity and the other as cumulative exposure to avoid co-linearity. All models included age, state, year or birth, race and work location (surface/underground) to adjust for confounding. For workers who remained at work after exposure follow-up ended in 1997, exposures after 1997 were assumed to be zero, cumulative REC and RD levels remained at their highest levels. We flexibly modelled the exposure-lag-response using distributed-lag non-linear (DLNM) models to disentangle exposure intensity, duration and timing of REC and RD exposure on risk of COPD mortality. Specifically, we fit generalized additive Poisson models in order to approximate a cox proportional hazards model in a GAM framework, allowing us to implement penalized distributed lag non-linear models as described by Gasparrini et al. (2017). We also applied Cox models to examine risk of suicide or overdose and age at worker exit (leaving work).

To address healthy worker survivor bias and answer questions about what the COPD and IHD risks would have been if the miners had been working under different (unobserved) exposure limits, we applied the parametric g-formula. The implementation of parametric g-formula followed four basic steps: (1) Parametric model fitting for time-varying variables. (2) Applying the parametric models on baseline data to simulate the natural course scenario under no intervention. (3) Simulate risks under hypothetical interventions. (4) Compute bootstrap confidence intervals for the measure of association. Close approximation of the natural course scenario to the observed data is an indicator for the quality of parametric model forms. Results are presented as risk differences between different exposure scenarios.

Employment records were not updated beyond 1997. To deal with the gap, we were compelled to either lag exposure by 18 years, appropriate for cancer but not so much for chronic disease, or else truncate follow-up, or assume that all miners still employed in 1997 when records ended were unexposed after that point.

4.0 Research Findings and Accomplishments:

Aim 1: Standard analysis of COPD

We first examined risk of COPD mortality in relation to both average intensity and cumulative exposure to diesel exhaust (REC) and dust (RD) in a standard survival analysis using Cox models based on the extended follow-up (DEMS II). We anticipated that cumulative exposure would be more relevant to risk because this metric combines intensity of daily exposure with the duration of exposure which we believe to be biologically relevant to the development of COPD. However, we found surprising inverse associations (hazard ratios below 1.0) between COPD mortality and cumulative exposures to both respirable dust and respirable elemental carbon.

The key to understanding these counterintuitive results is to appreciate the counterintuitive relationship between duration of employment and COPD. The median duration of employment was 7.8 years, and the interquartile range was 3-19 years. The hazard ratio (HR) for COPD decreased steadily as duration increased and was lowest (HR=0.71) for those in the highest category who had been employed more than 23 years. We believe that more cumulative exposure is associated with a lower rate of COPD for the same reason - miners who remained at work longer had lower risk of COPD. This provides strong evidence that healthier miners tend to remain at work while their less healthy co-workers leave work, ie, the healthy worker survivor effect (HWSE) is operating here. To account for HWSE, we needed to apply the parametric g-formula.

Aim 2: G-formula analysis for COPD and IHD

COPD: We applied the parametric g-formula to avoid healthy worker survivor bias by correctly handling leaving work as a time varying confounder on the causal pathway. This approach examines the impact of hypothetical exposure interventions for diesel exhaust and dust on risk of COPD mortality. The impact was measured by comparing the numbers of observed deaths due to COPD and ischemic heart disease, to the numbers we would expect to see under the hypothetical intervention. The current MSHA limit for diesel exhaust is equivalent to 106 ug/m3 respirable elemental carbon. Currently MSHA regulates PM as respirable Particulate Not Otherwise Specified (PNOS), based on the obsolete 1973 ACGIH TLV of 5 mg/m^3. Our results provide evidence below in Table 1 that interventions on diesel exhaust and/or dust exposure would reduce COPD mortality risk below that observed under current conditions.

Table 1: Lifetime cumulative risk of COPD mortality under the natural course and under intervention reducing REC and RD exposures in male workers of the DEMS II cohort followed for mortality between 1947 – 2015. Results based on g-computation analysis.

Intervention	Risk (%)	Risk Ratio (95% CI)	Risk Difference (95% CI)
Natural course (ref.)	7.3 (ref.)	1.00 (ref)	0.0 (ref)
REC<=106 μg/m3	7.2	0.99 (0.94, 1.06)	-0.1 (-0.5, 0.5)
REC<=25 μg/m3	6.6	0.90 (0.38, 1.09)	-0.7 (-10.9, 0.6)
REC=0	6.2	0.85 (0.55, 1.06)	-1.1 (-4.3, 0.4)
RD<=1 mg/m3	7.3	1.00 (0.68, 1.12)	0.0 (-4.6, 0.9
RD<=0.5 mg/m3	7.2	0.99 (0.62, 1.20)	-0.1 (-5.4, 1.5)
RD=0	6.7	0.93 (0.56, 1.31)	-0.5 (-5.9, 2.3)

Abbreviations: CI, confidence interval; COPD, chronic obstructive pulmonary disease; DEMS II, diesel exhaust in miners study II; REC, respirable elemental carbon; RD, respirable dust

IHD: After extending outcome follow-up from 1997 to 2015, the number of IHD deaths among male miners hired after dieselization increased from 297 to 951. An interquartile range (IQR) increase in average REC intensity (141.0 μg/m^3) was associated with higher risks of IHD death, HR = 1.21 (95% CI: 1.06 – 1.38); an IQR increase in average RD intensity (1.35 mg/m^3) was also associated with higher risks of IHD deaths, HR = 1.16, (95% CI: 1.04 – 1.31).

After restricting to ever-underground workers, both the highest average REC exposure group (HR = 1.35, 95% CI: 1.07 – 1.70) and the highest average RD exposure group (HR = 1.49, 95% CI: 1.13 – 1.96) showed increased IHD death risk after adjusting for covariates. No significant association was found among surface-only workers who were exposed to relatively low levels of EC and RD from their occupational environment. These patterns are consistent with the previously published analysis using outcomes before 1997 (Costello, 2018), with tighter confidence intervals due to higher number of cases and attenuated point estimates due to lack of exposure information between 1997 and 2015.

We also conducted two sensitivity analyses. First, we included workers who were employed before dieselization, whose cumulative RD exposure were likely to be underestimated because their RD exposure prior to dieselization were not captured. The trends of higher exposure groups associated with higher IHD risks remained consistent with the main analysis, while the HR point estimates slightly increased for average REC (HR = 1.24 compared to 1.13) and attenuated for average RD (HR = 1.37 compared to 1.62), which was expected due to the RD exposure misclassification. On top of that, we also attempted an alternative assumption for exposure after 1997: the annual exposures workers who remained at work in 1997 stayed the same as their last follow-up year levels until they reached 55 years old (estimated retirement) and became 0 after that. The estimates for both average EC (HR = 1.26 compared to 1.24) and average RD (1.40 compared to 1.37) remained almost identical.

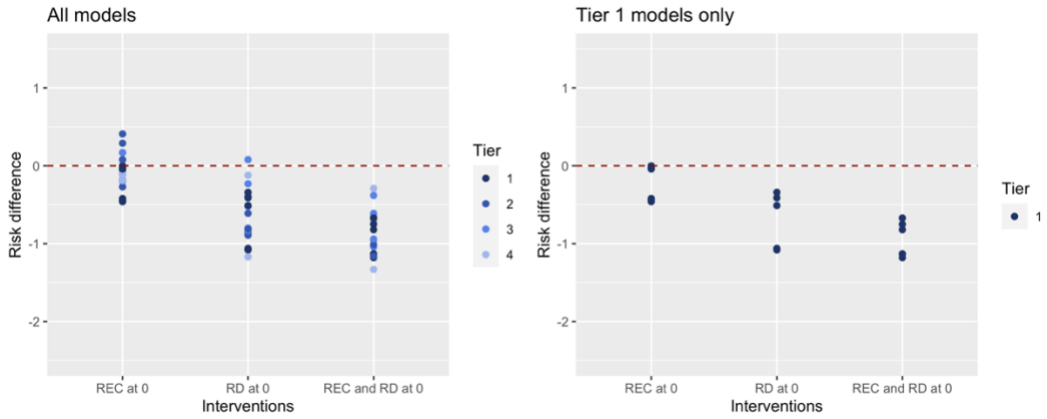
IHD and g-formula: We also investigated the effect of occupational REC and RD exposure on IHD mortality in the DEMS II cohort using parametric g-formula, a causal inference method that accounts for healthy worker survivor bias via simulation of hypothetical interventions. We restricted to 10,780 male workers hired after dieselization and assumed that those who remained at work when job record ended in 1997 left work in the next year, setting their exposures to zero in the following years. This assumption affected 51,043 (13.3%) person-years contributed by 3,200 (29.7%) workers, whose average age at last job record was 44.5 years old.

While implementing these steps, we attempted 20 sets of parametric models and noticed that different model specifications can largely affect the natural course quality, and the subsequent estimation of the etiologic risk differences. Thus, we qualitatively assessed the natural course quality based on graphical comparisons of the observed vs natural course simulation of time-varying variables over the follow-up years, with descending priorities for IHD mortality, annual REC and RD exposures, employment status variables and other variables. The 20 model sets were categorized into 4 quality tiers with tier 1 indicating the best performing natural courses.

Although subjective, our assessment of natural course qualities was helpful in reducing the variations in etiologic risk differences under hypothetical interventions (Figure 1). Results from the best performing model suggest that (1) setting REC exposure to 0 will reduce IHD mortality risk by 0.42% (95% CI: -0.90%, 1.74%), (2) setting RD exposure to 0 will reduce IHD mortality risk by 0.51% (95% CI: -1.24%, 1.74%), and (3) setting both REC and RD

exposure to 0 will reduce IHD mortality risk by 0.82% (95% CI: -0.56%, 2.21%). The previous finding by Neophytou et al in the DEMS1 cohort estimated that risk reductions for the three hypothetical interventions to be 2.0% (95% CI: -1.5, 5.5%), 1.6% (95% CI: -0.6, 3.8%), and 3.0% (95% CI: 0.3, 5.7%), respectively (Neophytou et al 2019). Compared to the DEMS1 results, the new results are qualitatively consistent but closer to the null, which is expected due to the exposure misclassification for those remained at work when job records ended.

Figure 1: Percent (%) Risk differences by model quality tiers for three hypothetical interventions (1) setting REC at 0, (2) setting RD at 0, and (3) setting both REC and RD at 0.



Aim 3: **Distributed exposure lag and COPD**

Results from distributed-lag non-linear models (DLNM) suggest a non-constant lag-response with elevated effect estimates associated with exposures occurring 30-50 years prior to the risk period for both REC and RD exposures. Results using the more sensitive definition of the outcome (with 284 cases of COPD identified) yielded somewhat higher effect estimates particularly for RD than REC, with the RD exposure effect appearing statistically significant at lags 38-42 years prior to death. However, all exposure-response results for both exposures were non-monotonic. These inconclusive results are likely due to the fact that work histories were not extended (as we expected) beyond 1997 when the mortality follow-up was extended to 2015. Because the DLNM approach was less illuminating than we had anticipated, we did not incorporate them into the g-computation analysis as originally intended.

Aim 4: **Job loss and Suicide**

We leveraged the extended mortality follow-up (1947-2015) in the Diesel Exhaust in Miners Study II (DEMS II) to compare age-adjusted suicide and overdose rates between men in the mining industry and the general US male population. We then investigated the association between age at leaving work and suicide or overdose. The 1980s began with two recessions in three years, and the manufacturing and mining industries never quite recovered production levels. We found that leaving work during the contraction of the US mining industry after 1980 contributed to the elevated risk of suicide and overdose among male miners, particularly among younger age groups. Based on the 248 suicides or

overdoses observed from 1947 to 2015, the rate of suicide peaked between 1980-1989 at 67.2 per 100,000, more than double the rate in the general population.

As seen in Table 2 below, the HRs were null before 1980. However post-1980, those leaving work between age 30-40 had an elevated risk of 1.46 and those who left work before age 30 had a HR for suicide or overdose of 1.94 compared to those who left work after 55. To understand the difference in results before and after 1980, consider that as the US mining industry declined and mines were downsized or closed, leaving work was more likely to be *involuntary* job loss after 1980. We believe that the economic and psychosocial disruption related to loss of a job contributes to the elevated risk of suicide or overdose mortality we observed among male miners, particularly among younger age groups.

Table 2. Adjusted hazard ratio estimates for suicide and overdose after leaving work by age at leaving work (N=8,213).

Age at leaving work	Entire Cohort[a] N Cases=189			Leaving Work before 1980 N cases=97			Leaving Work After 1980 N Cases=92		
	N	HR	(95% CI)	N	HR	(95% CI)	N	HR	(95% CI)
55 years or older	36	1.00	-	18	1.00	-	18	1.00	-
40-54 years old	32	0.93	0.57, 1.52	17	0.63	0.32, 1.27	15	1.19	0.59, 2.40
30-39 years old	52	1.05	0.67, 1.65	26	0.58	0.30, 1.12	26	1.46	0.78, 2.72
<30 years old	69	1.10	0.71, 1.71	36	0.48	0.26, 0.90	33	1.94	1.05, 3.58

Cox models use time since leaving work as the scale and are adjusted for 5-year calendar interval, work location (surface, underground), race (white, unknown and other), mine state.
[a] Population is restricted to persons who left work. Excludes those whose employment follow-up is censored and persons who died at work.

Diesel exhaust exposure does not directly increase suicide risk so suicide cannot block any diesel-related risk of IHD or COPD mortality.

5.0 Publication Record and Dissemination Efforts

Presented:
Ferguson J, Eisen E, Colbeth H, Costello S, Neophytou A, Koutros S, Silverman D. S-236 Suicide and Job Loss in the Diesel Exhaust in Miners Study II (DEMS II). Abstract presented at SER Symposium 2021.

Submittted:
Lu W, Picciotto S, Costello S, Keil A, Koutros S, Silverman D, Eisen E. Assessing Natural Course Simulation Quality of Parametric G-formula: an Illustration Using the Diesel Exhaust in Miners Study (DEMS). Abstract submitted to Society of Epidemiology (SER). January 2023.

In preparation:

Neophytou AM, Ferguson JM, Costello S, Picciotto S, Balmes JM, Koutros S, Silverman DT, Eisen EA. Diesel exhaust and respiratory dust exposure in miners and chronic obstructive pulmonary disease (COPD) mortality in DEMS II. Submitted to National Cancer Institute Working Group on DEMS. January 2023.

Colbeth HL, Ferguson JM, Costello S, Picciotto S, Neophytou AM, Koutros S, Silverman DT, Eisen EA. Risk of suicide and overdose in a cohort of non-metal miners. Submitted to National Cancer Institute Working Group on DEMS. March 2023.

Lu W, Picciotto S, Costello S, Keil A, Koutros S, Silverman D, Eisen E. Assessing Natural Course Simulation Quality of Parametric G-formula: Ischemic heart disease and diesel exhaust in a cohort of non-mental miners in the Diesel Exhaust in Miners Study (DEMS II).

6.0 Conclusions and Impact Assessment:

Our findings, based on extended follow-up of DEMS II, a large established cohort of non-metal miners, suggest that long term, cumulative exposures to both diesel exhaust and dust may modestly increase the risk of both COPD and IHD mortality. We also found evidence of considerable survivor bias in evaluating COPD mortality in this setting. Duration of exposure, as well as cumulative exposure (as opposed to average exposure intensity) were inversely associated with risk of COPD mortality when examined in a traditional regression framework. This counterintuitive finding is indicative of healthy worker survivor bias whereby workers least affected by exposure remain at work, and exposed, longer.

Healthy worker survivor bias is common in occupational epidemiology but can be mitigated using the parametric g-formula to evaluate risk under potential exposure interventions. In analysis based on the parametric g-formula, we found that interventions to mitigate diesel exhaust exposure, measured as respirable elemental carbon (REC), may lead to decreases in COPD mortality risk in this population, though 95% confidence intervals included the null. Increasing average intensity of respirable dust was also related to increased risk of COPD mortality, although results were stronger for REC. Many of the exposures in this study were higher than the current MSHA PEL for diesel exhaust. Our results suggest that interventions to mitigate this exposure may lead to decreases in mortality risk due to COPD in this population. Results for IHD mortality and diesel exhaust in this extended follow-up of the DEMS cohort were less conclusive based on the g-formula approach.

In comparison to age-adjusted rates of suicide among US males, rates of suicide among male miners in the DEMS II cohort remained consistently elevated over the extended follow-up period, indicating that it remains a long-standing public health crisis in this industry. We found that in the more recent period, miners who left work at younger ages were at increased risk of suicide or overdose. We believe that leaving work during the more recent period was likely tied to the downsizing and closures of mining facilities. Our longitudinal study of fatal suicide and overdose adds to the limited available research among US miners (Pizarro & Fuenzalida 2021). Our findings suggest that the mining industry, which cycles through economic boom-and-bust years, has the opportunity to

establish counseling and educational awareness programs that can reduce consistently elevated rates of suicide that peak during times of economic downturn.

In addition to the substantive findings, this work contributes to the methods for occupational epidemiology. Like all parametric methods, the parametric g-formula assumes correct specification of all the models involved. In g-formula applications, this assumption is usually assessed by comparing the observed risk with the simulated risk with no exposure intervention. However, there is no consensus on how to determine whether the simulated risk is close enough to the observed, and the literature does not discuss whether other covariates should also be considered. We found that close approximation of the observed risk does not guarantee good simulation of other covariates: poor predictions of exposures and time-varying covariates may still exist and cause biased effect estimation of potential interventions. Based on our findings, we recommend reporting graphic predictions over time of the outcome variables, intervened exposure variables, and other time-varying covariates when applying parametric g-formula. We further recommend that objective criteria for model misspecification be developed.

7.0 Recommendations for Future Work:

Next steps might be to help the policymakers for the mining industry understand the implications of how healthy worker survivor bias influences results of epidemiology studies and risk assessment. It is common in workplace studies and leads to attenuation of results that in turn can lead to exposure limits that are not adequately protective of miners' health. We are currently funded by Alpha on a translation project designed to learn how best to communicate epidemiology to improve protection of miners' health. Once we have produced translation material through focus groups, further improvement and wider dissemination to the mining community would be the focus of future work.

8.0 References:

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