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Original Article

Association between residence near surface coal mining and blood inflammation

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ABSTRACT

Previous research has documented that persons living near surface coal mining activity are at risk for poor health outcomes. The current study measured blood inflammation among adults ($N = 51$) living close to surface coal mining sites versus persons living farther away in Indiana and West Virginia, USA. None of the participants were smokers or coal miners, and none reported current acute illness. Participants completed a health interview and were tested for high sensitivity C-reactive protein as a measure of inflammation. We also collected indoor and outdoor particle counts and mass estimates at each residence. Results showed that mean C-reactive protein levels were significantly higher for residents who lived near mining, controlling for other risks (adjusted mean = 4.9 mg/L in the mining group and 0.9 mg/L in the non-mining group, $p < .03$). Mining residents also reported significantly more cardiopulmonary disease conditions and more illness symptoms. Particle counts were higher in the indoor and outdoor mining locations, and were most disparate for outdoor counts of particles in a respirable range between 0.5 μm and 5.0 μm. The results provide the first evidence that persons who live close to active surface coal mining show significantly elevated blood inflammation. Implications of results are discussed.

1. Introduction: proximity to surface coal mining and blood inflammation

Previous research has documented that persons living near coal mining sites have poor self-reported health outcomes and elevated population mortality rates (Hendryx, 2009; Hendryx and Ahern, 2009; Hendryx and Zullig, 2009). Limited evidence also documents poor environmental conditions in coal mining communities relative to control sites, including higher levels of particulate matter and relatively high presence of mining related constituents such as crystalline silica (Knuckles et al., 2013; Kurth et al., 2014b). Elevations in particulate matter from mining operations may be a contributing factor to poor population health in mining communities. However, no study to date has reported on direct biological markers of illness among people living in coal mining communities.

C-reactive protein (CRP) is an inflammatory marker produced in the liver in response to cytokine signaling (Black et al., 2004; Gabay and Kushner, 1999). High levels of CRP are predictive of an increased risk of cardiovascular disease (Ridker et al., 2000; Rutter et al., 2004) and poor lung function (Anderson, 2006; Folchini et al., 2011). High CRP may result from a variety of behavioral and environmental conditions. Smoking and obesity both increase risk for elevated CRP (Melbye et al., 2007). Environmental agents, notably particulate matter from traffic or industrial sources, has been found to be associated with elevated CRP on a chronic (Hoffmann et al., 2009) and acute (Chuang et al., 2007; Huttunen et al., 2012; Peters et al., 2001) basis, however, not all studies have observed this relationship (Rudez et al., 2009). Most studies of environmental conditions and inflammatory markers have been conducted in urban settings, and studies of associations for rural populations are relatively unexplored. Coal mining is predominately a rural-based activity and is known to increase localized ambient particulate matter (Ghose and Majee, 2007; Kolker et al., 2012; Kurth et al., 2014a,b).

The purpose of the current study was to test whether levels of C-reactive protein were significantly elevated in a rural sample living proximate to surface coal mining activity. Secondary goals were to gather additional evidence on levels of particulate matter and self-reported health for mining and non-mining community

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residents. To place the contribution of the study in perspective, we first offer a review of previous research on this topic.

2. Impacts of coal mining on public health: a review of the evidence

Despite a large body of evidence documenting occupational health problems for coal miners (Castranova and Vallyathan, 2000; Coggon and Taylor, 1998; Graber et al., 2014; Laney et al., 2012), until recently, little research had investigated possible public health consequences for persons living near coal mining activities. Among the earliest studies on this topic, Temple and Sykes (1992) showed increases in medical visits for asthma in conjunction with the opening of a surface mine in Great Britain. Also in Great Britain, Brabin et al. (1994) documented significantly higher respiratory symptoms among children exposed to coal dust, and a few years later, Pless-Mulloli and colleagues reported weak but significant associations between surface coal mining and children's respiratory health (Howel et al., 2001; Pless-Mulloli et al., 2000).

With the advent of large scale mountaintop removal (MTR) coal mining in the Appalachian US in the 1990s, anecdotal evidence (Burns, 2007; Goodell, 2006) began to emerge for its public health impacts, but empirical data were at first lacking. MTR is a particularly aggressive form of surface mining involving use of explosives and heavy machinery to remove hundreds of feet of rock and soil (i.e., overburden) above coal seams in steep terrain. The first study on the environmental public health impacts of US coal mining may be a study of hospitalization patterns associated with mining (Hendryx et al., 2007), followed shortly by an investigation of self-reported health indicators in association with higher levels of coal mining in Appalachian West Virginia (Hendryx and Ahern, 2008). A series of epidemiological studies emerged, some relying on ecological designs and/or secondary data (Ahern and Hendryx, 2012; Ahern et al., 2011a,b; Christian et al., 2011; Hendryx, 2009, 2011, 2013; Hendryx and Ahern, 2009; Hendryx and Innes-Wimsatt, 2013; Hendryx and Luo, 2014; Hendryx et al., 2008, 2012; Hendryx and Zullig, 2009; Zullig and Hendryx, 2010, 2011). All of these studies were limited to analysis of mortality statistics, morbidity self-reports, or secondary records of hospitalizations or birth certificate data. None included direct measures of environmental conditions in mining communities or direct measures of personal exposures. Nevertheless, the resulting pattern showed not only that a variety of health problems were significantly elevated in mining versus control communities after statistical control for other risks (e.g., age, sex, poverty, education, smoking, obesity, etc.) but also showed that health problems were proportional to tonnage of mining in a dose-response fashion, and that health problems were concentrated in MTR areas relative to areas where other forms of mining were practiced.

Meanwhile, a second research thread investigated the ecological impacts of coal mining on water quality (Lindberg et al., 2011; Palmer et al., 2010; Pond et al., 2008) and on air pollution (Ghose, 2007; Ghose and Banerjee, 1995; Pandey et al., 2014; Reynolds et al., 2003). Studies in this vein focused on assessments of environmental conditions rather than effects on humans and did not collect measurements specifically to assess conditions in human settlements. Regarding MTR specifically, important studies have documented serious and long term impairment to surface waters caused by MTR (Lindberg et al., 2011; Palmer et al., 2010; Pond et al., 2008). Air pollution specifically from MTR was not investigated.

Given the long history of coal mining, it is perhaps surprising that it took so long to examine what the environmental conditions in these communities actually were. A few studies have started to examine environmental conditions in residential mining communities and find evidence of elevated air and water pollution (Kurth et al., 2014a,b; Orem et al., 2012). Kurth et al. (2014a,b) have shown that MTR communities relative to controls have elevated levels of ultrafine material, and that elevated particulate matter in mining communities is predominantly a consequence of overburden removal from mining sites.

As these US studies were beginning to emerge, studies in other countries have also been published that show community health or environmental impacts from coal mining. These include a Turkish study of children's lead and cadmium exposure (Yapici et al., 2006), soil contamination in China (Liu et al., 2012; Wang et al., 2010), elevated rates of neural tube defects in China (Liao et al., 2010), impaired air quality in Columbia (Huertas et al., 2012), and environmental and health impacts in Australia (Higginbotham et al., 2010).

Most recently, going beyond mortality statistics or morbidity self-report, and beginning to merge environmental data with health data, particulate matter collected from ambient air sampling in MTR residential communities has been used in two laboratory studies. The first of these showed impaired microvascular function in rats upon respiratory exposure to MTR particulate matter (Knuckles et al., 2013). A second study found that particulate matter from MTR communities, but not from control communities, promoted tumor development and progression in human lung cells in vitro (Luanpitpong et al., 2014). Still, no US studies have collected data on biological health parameters for persons residing in communities near surface coal mining. The current study offers that new contribution.

3. Methods

3.1. Design and sample

We conducted a cross-sectional investigation of high sensitivity C-reactive protein (hsCRP) among adults living in rural communities within three miles of active surface coal mining compared to adults living farther away. Two surface coal mining areas in the eastern United States were included, one in Indiana and one in West Virginia; control participants were drawn from rural non-mining communities in the same states.

A total of 51 adults participated in the study. All samples were collected in August, 2014. Participants were recruited using a referral sampling approach beginning with initial contacts established by members of the research team. Participants then referred others who lived in mining and non-mining locations. All participants were non-smokers, not pregnant, and reported no current acute illness. No study participant used coal as a home heating source. Three adults who reported current employment as coal miners were excluded, resulting in a final sample of 48 including 23 in Indiana and 25 in West Virginia. There were 33 participants from households close to mining and 15 from non-mining control sites. Participants received modest financial incentives for participating in data collection activities. All participants underwent informed consent, and the study was reviewed and approved by the university Institutional Review Board.

Each individual was visited in their home where the blood sample was drawn, a health interview conducted, and air quality assessments made. The latitude and longitude of each household was recorded and the straight line distance was found between the residence and the boundary of the nearest active surface mining site. Wind speed and direction were recorded using data from the nearest weather monitoring station.

3.2. Measures

High sensitivity C-reactive protein (hsCRP) mg/L was measured using capillary finger sticks to collect dried blood spot samples. The

sampling kits were provided by ZRT Laboratory (Beaverton, OR). Sampling collection, storage and shipping protocols specified by the lab were followed. Briefly, each participant collected their own sample using a lancet and test card with assistance from the researchers as needed. Drops of blood were saved to the card, frozen at −4 °C until shipping, and shipped overnight for analysis using an enzyme-linked immunosorbent assay (ELISA). Internal laboratory quality control tests indicate that dried blood spot assay results correlate with serum samples collected at the same time at $R = 0.99$ (ZRTLab, n.d.).

In addition to hsCRP we collected measures on self-reported health indicators and residential ambient air quality. The measures of air quality included one indoor and one outdoor sample at the time of blood sampling; each of the indoor and outdoor samples included a particle count estimate and a mass estimate. Sampling was done using MetOne Aerocet 531 samplers (Grants Pass, OR). Particle counts included counts per cubic foot for particles >5.0 μm, and for particles >0.5 μm. We also calculated counts within a respirable range by finding the difference between the 0.5 μm and 5.0 μm counts. Mass estimates included PM2.5, PM10, and total suspended particles. Date, time of day, relative humidity, and temperature were recorded for each observation. Ideally, measures of particulate matter would be taken on a continuous basis for a period of at least 1–5 days prior to blood sampling for CRP (Gabay and Kushner, 1999) but the logistics and limited resources of our study did not permit this. For this reason, we would not necessarily expect levels of particulate matter to correlate to levels of hsCRP on the level of the individual person; rather, we used the air samples to gather preliminary indications as to whether particulate matter on average was higher in the mining versus the non-mining locations.

Finally, a self-report health survey was collected. The survey included information on demographics, height and weight to calculate Body Mass Index (BMI), former smoking (all participants were current non-smokers), current exposure to second hand smoke in the household, and previous occupational exposure as a coal miner. Health questions included whether the respondent had been diagnosed with myocardial infarction, angina or coronary heart disease, stroke, hypertension, diabetes, asthma (lifetime and current), or chronic obstructive pulmonary disease (COPD). In addition, participants were asked to report the presence of symptoms that they were currently experiencing or had experienced in the last month; a total of 32 symptoms were counted that represented seven types (respiratory, cardiovascular, musculoskeletal, gastrointestinal, skin, neurological, and other). Participants also completed the 10-item Perceived Stress Scale, with a higher score indicating more perceived stress (Cohen et al., 1983).

3.3. Analysis

Descriptive summaries of study variables were calculated. Group differences unadjusted for covariates were tested using Fisher's Exact test or *t*-tests. Linear or logistic regression models to control for covariates were examined. Because of the small sample size the number of possible covariates that could be modeled was limited. For this reason we first tested covariates for inclusion in linear regression models of health outcomes and hsCRP using a forward inclusion criterion with a $p < 0.10$ standard for inclusion. Residence in the mining community was not included as a variable in building the confounder models. Possible covariates included time of day, age, sex, obesity (BMI > 30), diabetes, college education (yes/no), marital status (married or not), prior occupational history as a coal miner (yes/no), former smoker (yes/no), current exposure to second hand household smoke (yes/no), and stress. Linear regression models for levels of particulate matter controlled for time of day, relative humidity and temperature; inclusion of these variables was again dependent on meeting forward inclusion criteria of $p < 0.10$.

4. Results

4.1. Descriptive summary of participants

A summary of study participants is provided in Table 1. Participants from the mining areas were on average older than participants from the non-mining control areas, and were less likely to have a college education. Differences in former smoking were significant at $p < 0.06$.

Before controlling for covariates, mining area participants reported a greater number of symptoms and were more likely to report the presence of one or more cardiopulmonary disease conditions. Cardiopulmonary conditions were measured dichotomously as the presence of one or more of myocardial infarction, angina, stroke, hypertension, current asthma, or COPD. Mining area residents had higher average hsCRP levels (4.3 versus 2.8 mg/L), but this difference did not achieve statistical significance before covariate adjustment. When hsCRP was divided into three categories (<1, 1–3, or >3 mg/L), mining area residents were significantly more likely to fall into the higher CRP categories ($p < 0.02$).

4.2. Particulate matter, meteorology and distance

Mean particle counts per cubic foot were higher for households near mining compared to households farther away. Particle counts were statistically higher controlling for time of day, relative humidity and temperature for all comparisons except larger indoor particles (see Table 2). Results for mass estimates (not shown) did not find significant differences on any of the indoor or outdoor samples between mining and non-mining households.

Wind speeds were generally light (mean = 2 mph, range = 0–9 mph). Temperature averaged about 25 °C, and relative humidity on average was 51% for indoor samples and 64% for outdoor (range = 34–89%). Average distance between households and mine boundaries was 1.1 miles in the mining group (sd = 0.7, range = 0.2–2.8). Among the non-mining group the mean distance was 40.2 miles (sd = 13.1, range = 6.4–48.0).

Table 1
Descriptive summary of study participants by mining location and total.

Variable	Mining	Non-mining	Total
Covariates			
Mean age*	55.1	39.3	50.2
Percent female	57.6	60.0	58.3
Percent obese	48.5	46.7	47.9
Percent married	78.8	73.3	77.1
Percent with college education*	54.6	86.7	64.6
Percent former smoker	42.4	13.3	33.3
Percent second hand smoke exposure	9.1	13.3	10.4
Percent with prior work experience as a coal miner	15.2	0	10.4
Mean stress score	11.9	11.6	11.8
Dependent variables			
Mean hsCRP mg/L	4.3	2.8	3.8
Percent with hsCRP*			
<1.0 mg/L	42.4	66.7	50.0
1.0–3.0 mg/L	27.3	6.7	20.8
>3.0 mg/L	30.3	26.7	29.2
Percent with one or more cardiopulmonary conditions*	60.6	6.7	43.8
Mean number of symptoms*	5.2	2.3	4.3

* $p < 0.05$ based on 2-tailed Fisher's Exact test or two-tailed *t*-test.

Table 2
Mean particle counts per cubic foot, indoor and outdoor samples in mining and non-mining locations. Means adjusted for time, relative humidity, and temperature.

	Mining	Non-mining
Indoor		
Particles ≥ 0.5 μm*	469,609	151,018
Particles ≥ 5.0 μm	3768	2715
Respirable range (0.5 μm–5.0 μm)*	466,159	147,507
Outdoor		
Particles ≥ 0.5 μm**	594,061	334,071
Particles ≥ 5.0 μm*	2031	829
Respirable range (0.5 μm–5.0 μm)**	591,899	333,600

* $p < 0.05$.
** $p < 0.01$.

4.3. HsCRP levels and health survey results adjusted for covariates

The forward inclusion linear regression model for hsCRP identified age and female sex as covariates meeting the $p < 0.10$ criterion. The final model with residence near the mining site, age and sex as independent variables identified living near mining as a significant independent risk for higher hsCRP (Model F = 6.09, df = 3.44, $p < 0.002$). The least squares adjusted means for hsCRP were 4.9 mg/L in the mining group and 0.6 mg/L in the non-mining group ($p < 0.03$).

Forward inclusion linear and logistic regression models for current symptoms and cardiopulmonary conditions, respectively, identified the following covariates: age, diabetes, second hand smoke exposure, stress and obesity (symptoms); age, female sex, and college education (cardiopulmonary conditions). With the inclusion of these covariates, living in a mining area was significantly associated with both health outcomes. In the case of symptoms, the mining variable was significant at $p < 0.006$, and the least squares adjusted means were 4.6 symptoms in the mining group compared to 1.8 symptoms in the non-mining group. The odds ratio for one or more cardiopulmonary conditions associated with residence in a mining area was 10.7 (95% CI = 1.1–107.9).

5. Discussion

We observed that persons living within three miles of active surface coal mining operations had significantly elevated levels of C-reactive protein compared to persons living farther away. All persons tested were non-smokers and non-miners without reported acute illness, and statistical analyses controlled for a set of potential confounders. A cut-point above 3.0 mg/L has been used to identify persons at risk for cardiovascular disease and other conditions (Ridker, 2003); we found that the average adjusted C-reactive protein level for residents near mining operations exceeded this cut-point at 4.9 mg/L, compared to 0.6 mg/L in the non-mining control sample.

We also observed that persons living in communities near surface mining had significantly elevated odds of a self-reported cardiopulmonary condition, a significantly greater number of current or recent reported symptoms across organ systems, and had significantly higher counts of respirable particles in ambient indoor and outdoor air samples. Mass estimates were not significantly elevated in the mining samples during these one-time sampling observations.

Our study was limited in number of ways. The sample was small and may not be representative of all members of the study populations. We were only able to collect one-time air quality samples at the same time as the assessments of C-reactive protein. The cross-sectional nature of the design does not allow us to conclude definitively whether elevated CRP levels result from exposure to particulate matter or result from other unmeasured influences. Future research should conduct ambient air assessments on a more continuous basis for a period of several days before assessing CRP. Our air quality assessments were also limited by the instrumentation that was available to us; previous research has suggested that ultrafine (aerodynamic diameter <0.1 μm) particle counts may be especially elevated in coal mining communities (Kurth et al., 2014b) but in the current study the available instruments were able to detect particles only larger than 0.5 μm. It is interesting, however, that the observed disparity in particle counts near mining operations was most pronounced for the smaller particle range in outdoor samples. Higher particle counts in the smaller size distribution, rather than conventional mass estimates, appear to be of more potential concern in rural mining communities. Mining operations generate particulate matter from blasting, draglines, coal crushing, wind erosion, and fossil fuel combustion from motor vehicles and other equipment (Ghose and Majee, 2007; Kurth et al., 2014b). The smaller particles may result from primary sources from explosives and machinery and secondary organic sources (Devlin et al., 2014).

Despite the limitations, this study is the first of its kind to demonstrate an elevated biological marker of poor health among persons who live near surface coal mining operations. All tested persons were non-smokers and non-miners. Results cannot be attributed to other risk variables including age, sex, co-occurring diabetes, obesity, education, exposure to second hand smoke, and others. C-reactive protein is an inflammatory marker that is predictive of risk for cardiovascular disease (Ridker et al., 2000; Rutter et al., 2004), respiratory illness (Anderson, 2006; Folchini et al., 2011) and other conditions, and may be a marker of exposure to mining activities.

6. Conclusion

An analogy of a partially completed jigsaw puzzle may serve as illustration of the overall state of evidence in this area. Some of the puzzle pieces represent environmental evidence for impaired air and water quality caused by mining and present in mining communities. Some represent epidemiological evidence from mortality and morbidity data. Some represent laboratory evidence of biological harm caused by particulate matter from mining communities. The newly discovered piece presented in this paper shows evidence for biological impact among people living in mining communities. All of these pieces are not yet put together into a single picture. The missing connectors will measure environmental exposure, dose, and biological impact all among the same persons who live in mining communities versus controls who do not. But even though all of the pieces are not connected, we can look at the partially completed puzzle and know what the picture is, if not yet all of the details. It is a picture of human health harm caused by surface mining to the people who live near the mining sites. The precautionary principle in environmental science argues that prudent steps are required when there is evidence of environmental harm and corresponding public health problems, even if all causal links are not understood (Kriebel et al., 2001). Such is certainly the case for surface mining of coal. Prudent steps include more effective regulatory control over surface mining practices, or in the case of MTR, the complete termination of the practice altogether (Palmer et al., 2010).

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References

Ahern, M., Hendryx, M., 2012. Cancer mortality rates in Appalachian mountaintop coal mining areas. J. Occup. Environ. Sci. 1 (2), 63–70.

Ahern, M., Hendryx, M., Conley, J., Fedorko, E., Ducatman, A., Zullig, K., 2011a. The association between mountaintop mining and birth defects among live births in Central Appalachia, 1996–2003. Environ. Res. 111 (6), 838–846, http://dx.doi.org/10.1016/j.envires.2011.05.019.

Ahern, M., Mullett, M., MacKay, K., Hamilton, C., 2011b. Residence in coal-mining areas and low-birth-weight outcomes. Matern. Child Health J. 15 (7), 974–979, http://dx.doi.org/10.1007/s10995-009-0555-1.

Anderson, G.P., 2006. COPD, asthma and C-reactive protein. Eur. Respir. J. 27 (5), 874–876, http://dx.doi.org/10.1183/09031936.06.00029306.

Black, S., Kushner, I., Samols, D., 2004. C-reactive protein. J. Biol. Chem. 279 (47), 48487–48490, http://dx.doi.org/10.1074/jbc.R400025200.

Brabin, B., Smith, M., Milligan, P., Benjamin, C., Dunne, E., Pearson, M., 1994. Respiratory morbidity in Meyerside schoolchildren exposed to coal dust and air pollution. Arch. Dis. Child. 70, 305–312.

Burns, S.S., 2007. Bringing Down the Mountains. West Virginia University Press, Morgantown.

Castranova, V., Vallyathan, V., 2000. Silicosis and coal workers' pneumoconiosis. Environ. Health Perspect. 108 (Suppl. 4), 675–684.

Christian, W.J., Huang, B., Rinehart, J., Hopenhayn, C., 2011. Exploring geographic variation in lung cancer incidence in Kentucky using a spatial scan statistic: elevated risk in the Appalachian coal-mining region. Public Health Rep. 126, 789–796.

Chuang, K.J., Chan, C.C., Su, T.C., Lee, C.T., Tang, C.S., 2007. The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults. Am. J. Respir. Crit. Care Med. 176 (4), 370–376, http://dx.doi.org/10.1164/rccm.200611-1627OC.

Coggon, D., Taylor, A.N., 1998. Coal mining and chronic obstructive pulmonary disease: a review of the evidence. Thorax 53, 398–407.

Cohen, S., Kamarck, T., Mermelstein, R., 1983. A global measure of perceived stress. J. Health Soc. Behav. 24 (4), 385–396.

Devlin, R.B., et al., 2014. Controlled exposure of humans with metabolic syndrome to concentrated ultrafine ambient particulate matter causes cardiovascular effects. Toxicol. Sci. 140 (1), 61–72, http://dx.doi.org/10.1093/toxsci/kfu063.

Folchini, F., Nonato, N.L., Feofiloff, E., D'Almeida, V., Nascimento, O., Jardim, J.R., 2011. Association of oxidative stress markers and C-reactive protein with multidimensional indexes in COPD. Chron. Respir. Dis. 8 (2), 101–108, http://dx.doi.org/10.1177/1479972310391284.

Gabay, C., Kushner, I., 1999. Acute-phase proteins and other systemic responses to inflammation. N. Engl. J. Med. 340 (6), 448–454, http://dx.doi.org/10.1056/NEJM199902113400607.

Ghose, M.K., 2007. Generation and quantification of hazardous dusts from coal mining in the Indian context. Environ. Monit. Assess. 130, 35–45.

Ghose, M.K., Banerjee, S.K., 1995. Status of air pollution caused by coal washery projects in India. Environ. Monit. Assess. 38 (1), 97–105.

Ghose, M.K., Majee, S.R., 2007. Characteristics of hazardous airborne dust around an Indian surface coal mining area. Environ. Monit. Assess. 130, 17–25.

Goodell, J., 2006. Big Coal. Houghton Mifflin, Boston.

Graber, J.M., Stayner, L.T., Cohen, R.A., Conroy, L.M., Attfield, M.D., 2014. Respiratory disease mortality among US coal miners: results after 37 years of follow-up. Occup. Environ. Med. 71 (1), 30–39.

Hendryx, M., 2009. Mortality from heart, respiratory, and kidney disease in coal mining areas of Appalachia. Int. Arch. Occup. Environ. Health 82, 243–249.

Hendryx, M., 2011. Poverty and mortality disparities in Central Appalachia: mountaintop mining and environmental justice. J. Health Dispar. Res. Pract. 4 (3), 44–53.

Hendryx, M., 2013. Personal and family health in rural areas of Kentucky with and without mountaintop coal mining. J. Rural Health 29, S79–S88.

Hendryx, M., Ahern, M., 2008. Relations between health indicators and residential proximity to coal mining in West Virginia. Am. J. Public Health 98, 669–671.

Hendryx, M., Ahern, M., 2009. Mortality in Appalachian coal mining regions: the value of statistical life lost. Public Health Rep. 124, 541–550.

Hendryx, M., Ahern, M.M., Nurkiewicz, T.R., 2007. Hospitalization patterns associated with Appalachian coal mining. J. Toxicol. Environ. Health A 70 (24), 2064–2070.

Hendryx, M., Innes-Wimsatt, K.A., 2013. Increased risk of depression for persons living in coal mining areas of Central Appalachia. Ecopsychology 5 (3), 179–187.

Hendryx, M., Luo, J., 2014. An examination of the effects of mountaintop removal coal mining on respiratory symptoms and COPD using propensity scores. Int. J. Environ. Health Res. 1–12, http://dx.doi.org/10.1080/09603123.2014.938027.

Hendryx, M., O'Donnell, K., Horn, K., 2008. Lung cancer mortality is elevated in coal mining areas of Appalachia. Lung Cancer 62, 1–7.

Hendryx, M., Wolfe, L., Luo, J., Webb, B., 2012. Self-reported cancer rates in two rural areas of West Virginia with and without mountaintop coal mining. J. Community Health 37, 320–327, http://dx.doi.org/10.1007/s10900-011-9448-5.

Hendryx, M., Zullig, K., 2009. Higher coronary heart disease and heart attack morbidity in Appalachian coal mining regions. Prev. Med. 49, 355–359.

Higginbotham, N., Freeman, S., Conner, L., Albrecht, G., 2010. Environmental injustice and air pollution in coal affected communities, Hunter Valley, Australia. Health Place 16 (2), 259–266.

Hoffmann, B., et al., 2009. Chronic residential exposure to particulate matter air pollution and systemic inflammatory markers. Environ. Health Perspect. 117 (8), 1302–1308, http://dx.doi.org/10.1289/ehp.0800362.

Howel, D., Pless-Mulloli, T., Darnell, R., 2001. Consultations of children living near open-cast coal mines. Environ. Health Perspect. 109 (6), 567–571.

Huertas, J.I., Huertas, M.E., Izquierdo, S., Gonzalez, E.D., 2012. Air quality impact assessment of multiple open pit coal mines in northern Colombia. J. Environ. Manage. 93 (1), 121–129, http://dx.doi.org/10.1016/j.jenvman.2011.08.007.

Huttunen, K., et al., 2012. Low-level exposure to ambient particulate matter is associated with systemic inflammation in ischemic heart disease patients. Environ. Res. 116, 44–51, http://dx.doi.org/10.1016/j.envres.2012.04.004.

Knuckles, T., et al., 2013. Air pollution particulate matter collected from an Appalachian mountaintop mining site induces microvascular dysfunction. Microcirculation 20, 158–169.

Kolker, A., et al., 2012. Atmospheric particulate matter in proximity to mountaintop coal mines. In: Paper Presented at the 22nd VM Goldschmidt Conference: Earth in Evolution, Montreal, 24 June 2012.

Kriebel, D., et al., 2001. The precautionary principle in environmental science. Environ. Health Perspect. 109, 871–876.

Kurth, L., et al., 2014a. Atmospheric particulate matter in proximity to mountaintop coal mines: sources and potential environmental and human health impacts. Environ. Geochem. Health, http://dx.doi.org/10.1007/s10653-014-9669-5.

Kurth, L.M., McCawley, M.A., Hendryx, M., Lusk, S., 2014b. Atmospheric particulate matter size distribution and concentration in West Virginia coal mining and non-mining areas. J. Expo. Sci. Environ. Epidemiol. 24, 405–411, http://dx.doi.org/10.1038/jes.2014.2.

Laney, A.S., Wolfe, A.L., Petsonk, E.L., Halldin, C.N., 2012. Pneumoconiosis and advanced occupational lung disease among surface coal miners – 16 states, 2010–2011. Morb. Mortal. Wkly. Rep. 61, 431–434.

Liao, Y., et al., 2010. Spatial analysis of neural tube defects in a rural coal mining area. Int. J. Environ. Health Res. 20 (6), 439–450, http://dx.doi.org/10.1080/09603123.2010.491854.

Lindberg, T.T., et al., 2011. Cumulative impacts of mountaintop mining on an Appalachian watershed. PNAS 108 (52), 20929–20934, http://dx.doi.org/10.1073/pnas.1112381108.

Liu, J., Liu, G., Zhang, J., Yin, H., Wang, R., 2012. Occurrence and risk assessment of polycyclic aromatic hydrocarbons in soil from the Tiefa coal mine district, Liaoning, China. J. Environ. Monit. 14 (10), 2634–2642, http://dx.doi.org/10.1039/c2em30433c.

Luanpitpong, S., et al., 2014. Appalachian mountaintop mining particulate matter induces neoplastic transformation of human bronchial epithelial cells and promotes tumor formation. Environ. Sci. Technol. 48 (21), 12912–12919, http://dx.doi.org/10.1021/es504263u.

Melbye, H., et al., 2007. Bronchial airflow limitation, smoking, body mass index, and statin use are strongly associated with the C-reactive protein level in the elderly. The Tromso Study 2001. Respir. Med. 101 (12), 2541–2549, http://dx.doi.org/10.1016/j.rmed.2007.07.018.

Orem, W., et al., 2012. Water chemistry in areas with surface mining of coal. In: Paper Presented at the Geological Society of America Annual Meeting and Exposition, Charlotte, NC, November 2012.

Palmer, M.A., et al., 2010. Mountaintop mining consequences. Science 327, 148–149.

Pandey, B., Agrawal, M., Singh, S., 2014. Coal mining activities change plant community structure due to air pollution and soil degradation. Ecotoxicology 23, 1474–1483.

Peters, A., et al., 2001. Particulate air pollution is associated with an acute phase response in men; results from the MONICA-Augsburg Study. Eur. Heart J. 22 (14), 1198–1204, http://dx.doi.org/10.1053/euhj.2000.2483.

Pless-Mulloli, T., et al., 2000. Living near opencast coal mining sites and children's respiratory health. Occup. Environ. Med. 57, 145–151.

Pond, G.J., Passmore, M.E., Borsuk, F.A., Reynolds, L., Rose, C.J., 2008. Downstream effects of mountaintop coal mining: comparing biological conditions using family- and genus-level macroinvertebrate bioassessment tools. J. N. Am. Benthol. Soc. 27 (3), 717–737.

Reynolds, L., Jones, T.P., BeruBe, K.A., Wise, H., Richards, R., 2003. Toxicity of airborne dust generated by opencast coal mining. Mineral. Soc. G. B. Irel. 67 (2), 141–152.

Ridker, P.M., 2003. Cardiology patient page. C-reactive protein: a simple test to help predict risk of heart attack and stroke. Circulation 108 (12), e81–e85, http://dx.doi.org/10.1161/01.CIR.0000093381.57779.67.

Ridker, P.M., Hennekens, C.H., Buring, J.E., Rifai, N., 2000. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N. Engl. J. Med. 342 (12), 836–843.

Rudez, G., et al., 2009. Effects of ambient air pollution on hemostasis and inflammation. Environ. Health Perspect. 117 (6), 995–1001, http://dx.doi.org/10.1289/ehp.0800437.

Rutter, M.K., Meigs, J.B., Sullivan, L.M., D'Agostino Sr., R.B., Wilson, P.W., 2004. C-reactive protein, the metabolic syndrome, and prediction of cardiovascular events in the Framingham Offspring Study. Circulation 110 (4), 380–385, http://dx.doi.org/10.1161/01.CIR.0000136581.59584.0E.

Temple, J.M.F., Sykes, A.M., 1992. Asthma and open cast mining. Br. Med. J. 305, 396–397.

Wang, R., Liu, G., Chou, C.L., Liu, J., Zhang, J., 2010. Environmental assessment of PAHs in soils around the Anhui Coal District, China. Arch. Environ. Contam. Toxicol. 59 (1), 62–70, http://dx.doi.org/10.1007/s00244-009-9440-6.

Yapici, G., Can, G., Kiziler, A.R., Aydemir, B., Timur, I.H., Kaypmaz, A., 2006. Lead and cadmium exposure in children living around a coal-mining area in Yatagan, Turkey. Toxicol. Ind. Health 22, 357–362.
ZRTLab. (n.d.) Blood spot test specifications: high sensitivity C-reactive protein. Beaverton, OR.
Zullig, K., Hendryx, M., 2011. Health-related quality of life among Central Appalachian residents in mountaintop mining counties. Am. J. Public Health 101, 848–853.
Zullig, K.J., Hendryx, M., 2010. A comparative analysis of health-related quality of life for residents of U.S. counties with and without coal mining. Public Health Rep. 125, 548–555.