

ORIGINAL ARTICLE

Hypertension Risk in Coal Miners after Healthy Hire Effect Elimination

SERGEY MAKSIMOV*, and GALINA ARTAMONOVA

Department Epidemiology of Cardiovascular Diseases, Institute for Complex Issues of Cardiovascular Diseases, Kemerovo, Russian Federation

Received May 28, 2014; Revised June 18, 2014; Accepted June 27, 2014

This paper is available on-line at <http://ijoh.tums.ac.ir>**ABSTRACT**

Healthy hire effect (HHE) is considered as an epidemiological phenomenon, complicating the assessment of a true cause - effect relationship between occupational exposure and health status of workers. The current study was aimed at identification and elimination of HHE in the risk assessment of arterial hypertension (AH) in coal miners. Overall, 1553 miners, working in West Siberia, were enrolled in the cross-sectional cohort study of risks for AH, the control group included 2266 subjects from other industrial sectors. The HHE identification was carried out with RR values in the age groups. A method of HHE elimination designed with analytical age-adjustment of hypertension rate in the studied group with the subsequent recalculation of the RR was implemented and tested. Initial data suggested a low risk of hypertension development in coal miners (RR = 0.58, 95 % CI 0,53:0,64) compared to workers of other industrial sectors. Elimination of HHE resulted in an increase of risk for hypertension in coal miners (RR = 1.11, 95 % CI 1,04:1,19). HHE reports that the health status of subjects, employed in the coal industry sector, is better compared to the other working population, resulting in lower incidence of hypertension. The application of the HHE elimination method resulted in the opposite results of professional affiliation impact on the incidence of hypertension compared to the initial data.

Keywords: *Healthy worker effect, Arterial hypertension, Coal miners, Epidemiology, Mathematical methods*

INTRODUCTION

Quite often epidemiological data on different health parameters of working population suggest that thought-to-be major workplace factors are not related to morbidity and mortality growth but, on the contrary, these data show inverse relationships between the mentioned factors [1]. As far back as 1885, W. Ogle noticed that mortality rates were counter intuitively lower in occupations with adverse working conditions than in occupations with more favorable working conditions or among the unemployed. In 1970s,

McMichael called this phenomenon a 'healthy worker effect'. Healthy hire effect (HHE) describes the healthy worker effect at the beginning of employment, as healthier persons are candidates for a job with adverse working conditions or a job in general compared to less healthy persons or those with impaired functional capacity [2-3]. HHE being one of the components of the healthy worker effect does not reflect possible favorable effects of working conditions on employees' health. At the same time, HHE distorts true cause-and-effect relationships and makes it seem like there is no positive correlation between workplace impacts and workers' health or, even, that there is a negative correlation, such as, the stronger the workplace factor effect, the less morbidity and mortality rates in the exposed population.

* **Corresponding author:** Sergey Maksimov, Email: m1979sa@yandex.ru

It is not difficult to identify HHE. Hypothetically, the health of young job candidates should be similar to that of the general population and the effect of the working conditions on their health is only to be seen after some exposure period (i.e., employment period). Therefore, HHE is demonstrated by the best health in young people with minimal employment record compared to the general population or people from another occupational cohort. There are enough data on HHE in people with asthma [4]: persons with asthma initially choose professions with low gas and particles exposure and prefer office employment (e.g., doctors, teachers, clerks, etc.).

No attempts were made to identify and, therefore, eliminate HHE in cardiovascular diseases and, particularly, arterial hypertension (AH). Nevertheless, some data suggest that HHE has a potentially significant effect on AH prevalence in the occupational populations with high exercise stress and high exposure to some adverse physical factors, i.e., noise, vibration [5-7]. The aim of our study was to identify and eliminate HHE while assessing AH risk in male coal miners.

MATERIALS AND METHODS

Study population and design

In 2012, all male workers of the main occupational groups from four coalmines of West Siberia were examined. The coalmines were randomly selected using a random numbers generator. The main occupational groups were coalmine shaft men, underground miners, coalmine face workers, and underground electricians.

While forming a control group to level the possible effect of a geographic factor on the study results, we did not use the data on AH prevalence in Russia. At the same time, there are no such data regarding West Siberia. Therefore, the control group was randomly selected from West Siberia male workers, excluding those working in coalmines.

People from the studied and control populations were examined during routine medical check-ups. The study was conducted under Good Clinical Practice standards and according to the principles of the Declaration of Helsinki. The study protocol was approved by the Ethics Committee of the Research Institute for Complex Issues of Cardiovascular Diseases under the Siberian Branch of Russian Academy of Medical Sciences. All of the participants signed a written informed consent to take part in the study.

In the group under study, there were 1, 553 people, and in the control group 2, 266 people.

The criteria to diagnose AH were systolic blood pressure of 140 mm Hg or higher and/or diastolic blood pressure of 90 mm Hg or higher or 'normal' blood

pressure in those taking antihypertensive drugs at the time of the study or within the previous 2 weeks.

Potential confounders and effect modifiers

Potential confounders and effect modifiers were a patient's age, body mass index and education level. These factors were chosen due to their proven effect on AH risk, on the one hand, and possibly significant differences in their prevalence in the study groups, on the other hand. Every patient's height and weight were measured and body mass index calculated taking the weight in kilograms and dividing it to the squared height in meters. Patients' education levels were self-reported and categorized into a higher education group (i.e., graduated from an institute or university) and secondary or incomplete secondary education group (i.e., finished a vocational college, technical school, high school or lyceum).

Statistical analysis

Intergroup differences between the studied and control groups were assessed with the Mann-Whitney test for quantitative variables (i.e., age and body mass index) and the Pearson's chi square (χ^2) test for dichotomized variables (AH, education level).

Quantitative variables were described as means and standard deviation.

Relative risk (RR) and 95% confidence interval (95% CI) for AH in coal miners were calculated; the control group was used for reference purposes. To estimate an effect of potential confounders (i.e., age, body mass index, and education level) on relationships between occupation and AH we performed the logistic regression analysis.

To identify HHE the RR of AH was calculated in 4 age groups: <31, 31-40, 41-50, and ≥ 51 yr. The data on the employment duration by occupation in the age groups are shown in Table 1. In the studied group, we performed analytic age-dependent adjustment of the original data on AH prevalence with subsequent recalculation of RR to eliminate HHE.

RESULTS

HHE identification from the original data

The mean age in the studied group was 39.4 \pm 9.3 yr, BMI 26.3 \pm 6.4 kg/m², and the proportion of people with higher education 11.7%. The same parameters in the control group were 42.4 \pm 10.7 yr, 26.3 \pm 4.3 kg/m², and 32.3%, respectively. Significant intergroup differences were found for age ($p < 0.0001$) and higher education level ($p < 0.0001$) but not for BMI ($p = 0.19$).

The logistic regression model demonstrated, after the adjustment for potential confounders, being a coal

Table 1. Duration of employment by occupation in different age groups

Group	n	Duration of employment by occupation					
		Mean	Standard deviation	Median	25 th percentile	75 th percentile	
Miners (studied group)	<31 yr	362	4.7	3.7	4.0	2.5	6.0
	31-40 yr	380	13.6	4.6	15.0	10.0	17.0
	41-50 yr	641	20.5	4.6	22.0	19.0	25.0
	≥51 yr	170	26.2	6.6	28.0	23.0	30.0
	All ages	1553	16.1	8.7	18.0	8.0	23.0
Non-miners (control group)	<31 yr	387	5.6	4.2	3.5	1.5	6.0
	31-40 yr	537	10.4	5.9	11.0	5.0	15.0
	41-50 yr	776	17.2	7.7	19.0	11.0	24.0
	≥51 yr	566	21.4	11.0	23.0	14.0	30.0
	All ages	2266	14.4	8.7	13.0	5.0	20.0

miner significantly ($p < 0.0001$) decreased the risk of AH (Table 2).

Lower RR of AH in coal miners was shown in all of the age groups; of note, RR increases with age (Table 3). Compared to the control group, lower RR of AH in the youngest miners (<31 yr) is the evidence of HHE.

Adjustment of the original data to eliminate HHE

RR adjustment was based on the assumption that HHE impact should be equal in all the age groups. That meant that RR level in the youngest patients, which directly reflected the presence of HHE, would not change with age. RR fluctuations in the older age groups describe the effects of another (apart from HHE) factors on AH prevalence in occupational groups. The analysis of these RR fluctuations excluding HHE-related RR components will allow eliminating HHE impact.

RR adjustment algorithm consisted of several systematic analytic corrections of the original data. Original (unadjusted) and adjusted data for the studied and control groups to calculate RR are presented in Table 4.

Since HHE is characterized by a certain proportion of AH prevalence in the studied and control groups in the youngest age (i.e., RR_1), a similar proportion should be observed in the older age groups. The first step was to calculate expected AH rates in all the age groups compared to AH rates in the control group (as they were the reference values) and RR_1 -value:

$$PX_n = Pc_n \times RR_1$$

Where n – the age group; PX_n – expected AH rate in the ‘ n ’ age group which should be observed at a given AH rate in the ‘ n ’ age control group and RR_1 -value; Pc_n – AH rate in the ‘ n ’ age control group; RR_1 – relative risk of AH in the youngest age group.

The second step was to convert percentages to absolute values:

$$X_n = PX_n \times a_n / Pa_n$$

where X_n – expected absolute number of people with AH in the ‘ n ’ age group which should be observed at a given AH rate in the ‘ n ’ age control group and RR_1 -value; a_n – original (non-adjusted) number of people with AH in the ‘ n ’ age group; Pa_n – original (non-adjusted) AH rate in the ‘ n ’ age group.

The next step was to measure the difference between the original (non-adjusted) and expected number of people with AH in the studied group (ΔX):

$$\Delta X_n = a_n - X_n$$

ΔX_n describes the number of people with AH in the ‘ n ’ age group, which is not related to HHE but associated with another factors. Further adjustment of the RR was based on the assumption that originally AH rates in the studied and control groups were equal in the youngest age subgroup. Hence, with age only ΔX_n should differ. To do the following calculations, we measured the expected absolute number of people with AH in the studied group (N_n), which corresponded to the AH rate in the control group (Pc_n):

$$N_n = Pc_n \times a_n / Pa_n$$

After summing up N_n and ΔX_n we got adjusted absolute number of people with AH in the ‘ n ’ age group (x_n):

$$x_n = N_n + \Delta X_n$$

A shorter way to calculate the absolute number of people with AH in the ‘ n ’ age group with the use of the 5 described equations can be presented as follows:

$$x_n = a_n \times \left(\frac{1 - RR_1}{RR_n} + 1 \right)$$

Table 2. Logistic regression model to predict AH

Parameter	Estimate	Standard error	t-value	p-value
Intercept	-6.271	0.345	-18.195	<0.0001
Age	0.050	0.004	11.880	<0.0001
Body mass index	0.123	0.103	11.960	<0.0001
Education level (comparison: Higher) Secondary	0.219	0.063	3.489	0.0005
Occupation (comparison: Non-miners) Miners	-0.567	0.081	-7.014	<0.0001

Table 3. Non-adjusted and adjusted relative risk of AH in coal miners

Age group	Non-adjusted RR		Adjusted RR	
	RR	95% CI	RR	95% CI
<31 yr	0.43	0.31 to 0.61	1.00	0.78 to 1.28
31-40 yr	0.53	0.41 to 0.68	1.09	0.91 to 1.31
41-50 yr	0.67	0.59 to 0.77	1.25	1.14 to 1.37
≥51 yr	0.78	0.66 to 0.92	1.34	1.22 to 1.48
All ages	0.58	0.53 to 0.64	1.11	1.04 to 1.19

After adjustment the total population of the studied group (A) remained unchanged, therefore, the absolute number of people without AH was calculated as shown below:

$$y_n = A_n - x_n$$

With the use of the adjusted data, it is possible to estimate the risk of AH both in the age groups and on the whole, irrespective of age.

RR of AH in coal miners after HHE was eliminated

After the elimination of HHE, RR of AH in the studied group became >1.0 compared to the control group (Table 3). RR of AH in coal miners is significantly higher than in non-miners in the age groups of 41-50 and ≥51 yr, and overall, irrespective of age. RR of AH in coal miners increases with age.

DISCUSSION

This study tested the analytical method to eliminate HHE with the use of RR of AH in coal miners. HHE elimination caused the effect of occupation on the disease risk to reverse (compared to that observed before the elimination). If the original data suggested that working in underground coalmines was associated with lower risk of AH, then the adjusted data showed that workplace factors of coal miners led to a higher risk of AH.

Study limitations and advantages

There are several relative limitations in our study. Firstly, one of the basic assumptions of the study is the hypothesis that the impact of workplace factors on workers' health is only seen after certain exposure period (duration of employment). At the same time, RR was adjusted for workers' age but not for the duration of employment by occupation, which needs to be explained. Due to possible individual particularities of the employment duration by occupation, it is quite difficult to control this factor. For example, a worker could initially start the occupation of interest after reaching the employable age or could be transferred into this occupation from a similar or completely different one in an older age. Finally, a worker might start working a while after reaching the employable age. In addition, a worker's age is a stable easily controlled factor strongly correlating with the duration of employment. As Table 1 shows, age-categorization leads to overall good grouping of workers according to their employment duration with the youngest group including the workers with the minimal employment record.

Second, the study included miners of West Siberia where there surely are unique workplace conditions due to specific coal deposit features and coal extraction methods. Moreover, geographic, social, economic, and population particularities of West Siberia are probably

responsible for the fact that it is quite difficult to extrapolate our data to miners of other countries and, even, other regions of Russia, as well. The presented results need to be seen mainly as a demonstration of an analytic method to eliminate HHE on the example of a specific disease and a specific occupational group. The applicability of this method should be confirmed with other diseases and other workplace conditions as well as other cause-and-effect relationships between the health status and workplace factors.

Third, RR of AH in coal miners was only adjusted for HHE ignoring the other factors possibly having an effect on AH risk. Such factors are, first, healthy survivor effect; the impact of this effect could be observed irrespective of HHE. Nonetheless, only HHE elimination allowed obtaining the adjusted data showing tendencies contrary to those observed in the original (non-adjusted) data. Thus, even if this method would not let us have 'ultimate' true estimates of occupational risks, it would bring us much nearer.

Comparison with other studies

The majority of studies showed that AH risk commonly rose when exposed to such workplace factors, as noise, heat, carbon monoxide, or organic solvents [8-11]. In the available literature, we could not find any data on the possible HHE-interference into the effect of workplace factors on AH development. On the other hand, several studies on AH prevalence in different occupational groups demonstrated tendencies relevant to the healthy worker effect: sailors [12], blue-collar workers [6] and police officers [13]. Might not only the proposed approach help understand if there was HHE, but eliminate its impact on true cause-and-effect relationships between occupations and AH risk.

Noteworthy, there are studies on other cardiovascular diseases where the healthy worker effect is taken into account. A systematic review on workplace arsenic exposure effect on cardiovascular morbidity showed that only one study considered the healthy worker effect. After the healthy worker effect was eliminated, arsenic exposure effect on mortality associated with myocardial infarction increased [14]. The review of 23 studies demonstrated that the healthy worker effect elimination allowed proving that the profession of fire fighter is associated with post-infarct mortality and, less evidently, with myocardial infarction morbidity [15]. Nuclear industry workers were shown to have lower mortality (mainly cardiovascular), which was explained by the healthy worker effect, even though there were no attempts to adjust the data for it [1].

As for the methods to diminish the healthy worker effect, to date, substantial amount of data on this issue have been obtained. Some of these methods, which are quite extensively used to lessen the healthy worker effect, are well-known epidemiological analytical approaches (e.g., use of adjusting factors, stratification, use of time lags or exclusion of workers with short

employment record) and the specific design of experiments (e.g., maximal representatively or the use of inner comparison groups). Such methods were more or less effectively used to diminish the healthy worker effect [3, 16-18] or one of its central components, i.e., the healthy survivor effect [19-20]. Now, modern statistical methods are also used, such as, structure modeling [21], and specific methods, for example, g-estimation [20, 22].

However, all those methods allow decreasing the healthy worker effect, the healthy survivor effect or HHE to only a certain extent. The method that we used for the present study helped us eliminate HHE assuming that the effect of workplace conditions on health is only to be seen after some exposure period (i.e., employment period).

CONCLUSION

The use of the method to eliminate HHE with the original data adjustment led to the reversal of the occupational effect trend on AH prevalence compared to unadjusted results. Unadjusted RR of AH in coal miners was 0.58 irrespective of age, while the adjusted RR was 1.11. The coal miners from the youngest age group had the least non-adjusted RR of AH. Thus, the health of people seeking a job in the main occupational groups in the mining industry is better than in the general working population, therefore, AH prevalence was low. Workplace factors effect leads to a significant increase in AH morbidity in people older than 40 yr, which corresponds to the employment duration by occupation of 20 yr. However, a possible impact of other factors, primarily the healthy survivor effect, on this process cannot be ignored.

ACKNOWLEDGMENTS

The research was financed by the Russian Foundation for Humanities as part of the 'Development and implementation of primary and secondary hypertension prevention system in workers of coalmines' project, №12-06-00107. The authors declare that there is no conflict of interests.

REFERENCES

1. McGeoghegan D, Binks K, Gillies M, Jones S, Whaley S. The non-cancer mortality experience of male workers at British Nuclear Fuels plc, 1946-2005. *Int J Epidemiol* 2008; 37: 506-518.
2. Arrighi HM, Hertz-Picciotto I. The evolving concept of the healthy worker survivor effect. *Epidemiology* 1994; 5: 189-196.
3. Li CY, Sung FC. A review of the healthy worker effect in occupational epidemiology. *Occup Med (Lond)* 1999; 49: 225-229.
4. Le Moual N, Kauffmann F, Eisen EA, Kennedy SM. The healthy worker effect in asthma: work may cause asthma, but

- asthma may also influence work. *Am J Respir Crit Care Med* 2008; 177: 4-10.
5. Enterline PE. Comments on the "healthy worker effect" in occupational epidemiology. In: Reports to the Workers' Compensation Board on the Healthy Worker Effect. Toronto, Canada: Ministry of Labour of the Government of Ontario, ISDP Report 3. 1988; Available from: <http://www.canoshweb.org/odp/html/JUL1988.htm>.
 6. Skrobonja A, Kontosic I. Arterial hypertension in correlation with age and body mass index in some occupational groups in the harbor of Rijeka, Croatia. *Ind Health* 1998; 36: 312-317.
 7. Maksimov SA, Skripchenko AE, Indukaeva EV et al. Age features of epidemiology of arterial hypertension in workers of the coal-mining enterprises. *Adv Gerontol* 2011; 24: 697-700.
 8. Jovanovic J, Jovanovic M. Occupational stress and arterial hypertension. *Med Pregl* 2004; 57: 153-158.
 9. Narlawar UW, Surjuse BG, Tharke SS. Hypertension and hearing impairment in workers of iron and steel industry. *Indian J Physiol Pharmacol* 2006; 50: 60-66.
 10. Vangelova KK, Deyanov CE. Blood pressure and serum lipids in industrial workers under intense noise and a hot environment. *Rev Environ Health* 2007; 22: 303-311.
 11. Chang TY, Wang VS, Hwang BF et al. Effects of co-exposure to noise and mixture of organic solvents on blood pressure. *J Occup Health* 2009; 51: 332-339.
 12. Kirkutis A, Norkiene S, Grieciene P et al. Prevalence of hypertension in Lithuanian mariners. *Proc West Pharmacol Soc* 2004; 47: 71-75.
 13. Idahosa PE. Hypertension: an ongoing health hazard in Nigerian workers. *Am J Epidemiol* 1987; 125: 85-91.
 14. Navas-Acien A, Sharrett A, Sibergeld EK et al. Arsenic exposure and cardiovascular disease: a systematic review of the epidemiologic evidence. *Am J Epidemiol* 2005; 162: 1037-1049.
 15. Choi BC. A technique to re-assess epidemiologic evidence in light of the healthy worker effect: the case of firefighting and heart disease. *J Occup Environ Med* 2000; 42: 1021-1034.
 16. McMichael AJ. Standardized mortality ratios and the «healthy worker effects»: scratching beneath the surface. *J Occup Med* 1976; 18: 165-168.
 17. Qiao R, Wang M, Wang Z. Methodology for controlling healthy worker effect on coal miners' mortality. *J West China University Med Sci* 1996; 27: 90-93.
 18. Sterling TD, Weinkam JJ. Observations on possible sources, extent, persistence, constancy, and corrections for the healthy worker effect. In: Reports to the Workers' Compensation Board on the Healthy Worker Effect. Toronto, Canada: Ministry of Labour of the Government of Ontario, ISDP Report 3. 1988; Available from: <http://www.canoshweb.org/odp/html/JUL1988.htm>.
 19. Eisen EA, Holcroft CA, Greaves IA et al. A strategy to reduce healthy worker effect in a cross-sectional study of asthma and metalworking fluids. *Am J Ind Med* 1997; 31: 671-677.
 20. Chevrier J, Picciotto S, Eisen EA. A comparison of standard methods with G-estimation of accelerated failure-time models to address the healthy-worker survivor effect: application in cohort of autoworkers exposed to metalworking fluids. *Epidemiology* 2012; 23: 212-219.
 21. Dumas O, Le Moual N, Siroux V et al. Marginal structural models to quantify and control for the healthy worker effect in asthma: results from the EGEEA study. *Am J Respir Crit Care Med* 2012; 185: A1175.
 22. Arrighi HM, Hertz-Picciotto I. Controlling the healthy worker survivor effect: an example of arsenic exposure and respiratory cancer. *Occup Environ Med* 1996; 53: 455-462.