Occupational Heart Disease

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Figure 5. Age-adjusted death rates for the 15 leading causes of death: United States, 1958-2002
我國勞工的主要死亡原因分析－惡性腫瘤(癌症)、事故傷害、心臟疾病是勞工朋友三大健康殺手
2010/2/2

勞委會勞工安全衛生研究所(以下簡稱勞研所)，分析勞保投保勞工資料，結果顯示，97年勞保投保勞工死因，以每十萬勞工死亡率(以下簡稱死亡率)為55.1之惡性腫瘤(即俗稱的癌症)列居首位外，事故傷害死亡率(18.2)仍居第二(較96年死亡率減少約十萬分之9.1)，且較特殊的是心臟疾病於96年以12.9由95年第四位躍居第三後，97年仍以9.2續列第三高。整體而言，97年死亡率較95-96年減少約8~30%。
勞工排前五大死因之死亡率趨勢變化

![Bar Chart](chart.png)

- **所有死亡原因**
- **恶性腫瘤**
- **事故傷害**
- **慢性肝病及肝硬化**
- **心臟疾病**
- **腦血管疾病**

- **91年**
- **92年**
- **93年**
- **94年**
- **95年**
- **96年**
- **97年**

91~97年
Evolution of Atherosclerosis
Causes of Heart Disease (CVD)

- Aging
- Lifestyle
- Disease
Major Independent CVD Risk Factors

- Cigarette smoking
- Elevated blood pressure
- Elevated serum total (and LDL) cholesterol
- Low serum HDL cholesterol
- Diabetes mellitus
- Advancing age

Other CVD Risk Factors

Predisposing risk factors

- Obesity
- Abdominal obesity
- Physical inactivity
- Family history of premature coronary heart disease
- Ethnic characteristics
- Psychosocial factors

Conditional risk factors

- Elevated serum triglycerides
- Small LDL particles
- Elevated serum homocysteine
- Elevated serum lipoprotein(a)
- Prothrombotic factors (eg, fibrinogen)
- Inflammatory markers (eg, C-reactive protein)

* These factors are defined as major risk factors by AHA
Previous Study Results of Potential Risk Factors and Occupational Heart Diseases
Occupational Cardiovascular Disease

- A few specific toxins encountered occupationally are known to affect the heart, most prominently:
  - carbon disulfide,
  - nitroglycerin, and
  - carbon monoxide
- There is substantial evidence that:
  - environmental tobacco smoke,
  - extreme heat, and
  - extreme cold
- Numerous studies show a relationship between heart disease and depression and exposure to stress at work.
- Other work-related exposures potentially related to cardiovascular disease include occupational noise exposure, shift work, and physical activity at work.
• Epidemiologic studies of cardiovascular disease in exposed workers from Pennsylvania, Finland, and Belgium were quoted.
• Researchers in Japan found no increased CAD in exposed workers, but a striking increase in retinal microaneurysms.
• The association between CAD and occupational exposure to nitroglycerin, ethylene-glycol-dinitrate and other aliphatic nitrates, carbon-monoxide, nonhalogenated and halogenated industrial solvents, arsenic, and cobalt, as well as blood pressure problems caused by cadmium and lead were discussed.
• Passive smoking was described as an important public health factor in deaths from CAD.
• Nonchemical factors in occupational CAD were shift work, noise, and stressors due to organization, work or psychosocial factors.
• Cardiac disease secondary to occupational lung disease was a result of pulmonary hypertension.
• The prevalence of CAD was reduced in occupations with very high levels of energy expenditure.
Risks of Occupational Heart Disease

- Carbon Disulfide
- Carbon Monoxide
- Environmental Tobacco Smoke
- Nitroglycerin
- Shift Work
- Stress

NIOSH web site
Carbon Disulfide and Heart Disease

Evidence for a direct causal relationship between carbon-disulfide and coronary artery disease (CAD) was strongest.
# Some Concentration-response Relationships in Occupational Exposure to Carbon Disulfide

<table>
<thead>
<tr>
<th>Carbon disulfide concentration (mg/m²)</th>
<th>Duration of exposure (years)</th>
<th>Symptoms and signs</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>500-2500</td>
<td>0.5</td>
<td>Polyneuritis, myopathy, acute psychosis</td>
<td>6</td>
</tr>
<tr>
<td>450-1000</td>
<td>&lt; 0.5</td>
<td>Polyneuritis, encephalopathy</td>
<td>7</td>
</tr>
<tr>
<td>200-500</td>
<td>1-9</td>
<td>Increased ophthalmic pressure</td>
<td>12</td>
</tr>
<tr>
<td>60-175</td>
<td>5</td>
<td>Eye burning, abnormal papillary light reactions</td>
<td>13</td>
</tr>
<tr>
<td>31-137</td>
<td>10</td>
<td>Psychomotor and psychological disturbances</td>
<td>8</td>
</tr>
<tr>
<td>29-118</td>
<td>15</td>
<td>Polyneuropathy, abnormal EEG, conduction velocity slowed, psychological changes</td>
<td>9, 10</td>
</tr>
<tr>
<td>29-118</td>
<td>10</td>
<td>Increase in coronary mortality, angina pectoris, slightly higher systolic and diastolic blood pressure</td>
<td>14-17</td>
</tr>
<tr>
<td>40-80</td>
<td>2</td>
<td>Asthenospermia, hyposperma, teratosperma</td>
<td>19</td>
</tr>
<tr>
<td>22-44</td>
<td>&gt; 10</td>
<td>Arteriosclerotic changes and hypertension</td>
<td>25</td>
</tr>
<tr>
<td>30-50</td>
<td>&gt; 10</td>
<td>Decreased immunological reactions</td>
<td>26</td>
</tr>
<tr>
<td>30</td>
<td>3</td>
<td>Increase in spontaneous abortions and premature births</td>
<td>20</td>
</tr>
<tr>
<td>20-25</td>
<td>&lt; 5</td>
<td>Functional disturbances of the CNS</td>
<td>27, 28</td>
</tr>
<tr>
<td>10</td>
<td>10-15</td>
<td>Sensory polyneuritis, increased pain threshold</td>
<td>11</td>
</tr>
<tr>
<td>10</td>
<td>10-15</td>
<td>Depressed blood progesterone, increased estriol, irregular menstruation</td>
<td>21</td>
</tr>
</tbody>
</table>
Electrocardiographic abnormality for workers exposed to carbon disulfide at a viscose rayon plant

Objective: This study investigated electrocardiography (ECG) manifestations for male workers with carbon disulfide exposure at rayon manufacturing plants.

Methods: A total of 251 men in the exposure group and 226 administrative clerks in the reference group received physical examinations and completed questionnaires.

Results: The prevalence of ECG abnormalities was much higher in the carbon disulfide exposure group (25.9%, n = 65) than in the reference group (2.7%, n = 6), with an odds ratio (OR) of 12.8 (95% confidence interval [CI] = 5.4-30.2). The foremen were at the highest risk of abnormal ECG (OR = 20.6, 95% CI = 6.5-65.2), followed by filament-spinning workers (OR = 14.2 95% CI = 5.7-35.3), viscose-manufacturing workers (OR = 11.3, 95% CI = 4.3-30.1), and carbon disulfide-manufacturing workers (OR = 8.1, 95% CI = 2.7-25.6). The multivariate logistic regression analysis based, on cumulative exposure index also showed a dose-response relationship with the exposure, and the risk of ECG abnormality could be initiated at the exposure history of 31 to 57 year-ppm with an OR of 7.2 (95% CI = 1.5-36.7).

Conclusions: In general, the ECG abnormalities observed in workers at the permissible exposure level of carbon disulfide implicate the importance of environmental control of the chemical and of workers’ education in exposure prevention at work.

Carbon Monoxide and Heart Disease

Exposure of Motor Vehicle Examiners to Carbon Monoxide: A Historical Prospective Mortality Study

Stern FB, Lemen RA, Curtis RA

Arch Environ Health 1981;36(2):59-66

The effect of exhaust emissions, including carbon-monoxide (CO), on motor vehicle examiners was investigated. Mortality rates among 1,558 examiners employed for at least 6 months between 1944 and 1973 were determined from local records. CO readings were taken at 31 of 38 inspection stations and 27 examiners were administered carboxyhemoglobin (COHb) tests. The mean CO concentrations for indoor and outdoor inspection stations were 24.4 and 10.0 parts per million, respectively. The mean pre and post shift COHb concentrations were 3.3 and 4.7 percent, respectively. There were 237 deaths from all causes during the period of study, as opposed to an expected 260.4 deaths. Among the examiners, there were 124 deaths from cardiovascular diseases compared to 118.4 expected, and 52 deaths from malignant neoplasms compared to 47.8 expected. There were fewer deaths than expected from diseases of the nervous system, nonmalignant diseases of the respiratory and digestive systems, and accidents. The authors suggest that the excess of deaths due to cardiovascular diseases is due to CO exposure. The excess cancer death rate may be due to contaminants other than CO.
Environmental Tobacco Smoke and Heart Disease

The National Institute for Occupational Safety and Health (NIOSH) has determined that environmental tobacco smoke (ETS) is potentially carcinogenic to occupationally exposed workers. In 1964, the Surgeon General issued the first report on smoking and health, which concluded that cigarette smoke causes lung cancer. Since then, research on the toxicity and carcinogenicity of tobacco smoke has demonstrated that the health risk from inhaling tobacco smoke is not limited to the smoker, but also includes those who inhale ETS. ETS contains many of the toxic agents and carcinogens that are present in mainstream smoke, but in diluted form. Recent epidemiologic studies support and reinforce earlier published reviews by the Surgeon General and the National Research Council demonstrating that exposure to ETS can cause lung cancer. These reviews estimated the relative risk of lung cancer to be approximately 1.3 for a nonsmoker living with a smoker compared with a nonsmoker living with a nonsmoker. In addition, recent evidence suggests a possible association between exposure of nonsmokers to ETS and an increased risk of heart disease.

Although these data were not gathered in an occupational setting, ETS meets the criteria of the Occupational Safety and Health Administration (OSHA) for classifying substances as potential occupational carcinogens [Title 29 of the Code of Federal Regulations, Part 1990]. NIOSH therefore recommends that ETS be regarded as a potential occupational carcinogen in conformance with the OSHA carcinogen policy, and that exposures to ETS be reduced to the lowest feasible concentration. Employers should minimize occupational exposure to ETS by using all available preventive measures.
Recent Studies of Heart Disease among Never Smoked ETS-exposed Persons

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Exposure definition</th>
<th>Relative risk (^2)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Update of Gillis et al. [1984] by Hole et al. [1989]</td>
<td>12-yr followup, 3,960 men and 4,037 women aged 45-64 in 1972-76</td>
<td>Living with smoker or ex-smoker in early 1970s</td>
<td>2.01 (CI, 1.21–3.35; 485 observed)</td>
<td>Adjusted for cardiovascular risk factors; positive dose response</td>
</tr>
<tr>
<td>Humble et al. [1990]</td>
<td>20-yr followup, 513 women aged 40+</td>
<td>Living with smoker in 1960</td>
<td>1.59 (CI, 0.99-2.57)</td>
<td>Adjusted for cardiovascular risk factors, dose response in some strata</td>
</tr>
<tr>
<td>Helsing et al. [1988]</td>
<td>12-yr followup, 4,162 men and 14,873 women aged 25+ in 1963</td>
<td>Living with smoker or ex-smoker in 1983</td>
<td>Men: 1.31 (CI, 1.1-1.6; 492 observed) Women: 1.24 (CI, 1.1-1.4; 1,530 observed)</td>
<td>Adjusted for education, positive dose response among women only</td>
</tr>
<tr>
<td>Svendsen et al. [1985,1987]</td>
<td>7-yr average followup, 1,245 men aged 35-57 in 1973-82</td>
<td>Married to smoker or ex-smoker</td>
<td>1.61 (CI, 0.96-2.71; 60 observed)</td>
<td>Adjusted for cardiovascular risk factors, positive dose response</td>
</tr>
<tr>
<td>Garland et al. [1985]</td>
<td>10-yr followup, 695 women aged 50-79 in 1972-74</td>
<td>Married to smoker or ex-smoker</td>
<td>2.9 (estimate; 19 observed)</td>
<td>Adjusted for age</td>
</tr>
<tr>
<td>Hiyama [1984]</td>
<td>16-yr followup, 91,540 women aged 40+</td>
<td>Married to smoker or ex-smoker</td>
<td>Low exposure: 1.10 (90% CI, 0.91-1.33) High exposure: 1.31 (90% CI, 1.06-1.63; 484)</td>
<td>Significant dose response</td>
</tr>
</tbody>
</table>
Passive smoking and cardiorespiratory health in a general population in the west of Scotland

David J Hole, Charles R Gillis, Carol Chopra, Victor M Hawthorne

Abstract

Objective—To assess the risk of cardiorespiratory symptoms and mortality in non-smokers who were passively exposed to environmental smoke.

Design—Prospective study of cohort from general population first screened between 1972 and 1976 and followed up for an average of 11·5 years, with linkage of data from participants in the same household.

Setting—Renfrew and Paisley, adjacent burghs in urban west Scotland.

Subjects—15399 Men and women (80% of all those aged 45-64 resident in Renfrew or Paisley) comprised the original cohort; 7997 attended for multiphasic screening with a cohabitee. Passive smoking and control groups were defined on the basis of a lifelong non-smoking index case and whether the cohabitee had ever smoked or never smoked.

Main outcome measure—Cardiorespiratory signs and symptoms and mortality.

Results—Each of the cardiorespiratory symptoms examined produced relative risks >1·0 (though none were significant) for passive smokers compared with controls. Adjusted forced expiratory volume in one second was significantly lower in passive smokers than controls. All cause mortality was higher in passive smokers than controls (rate ratio 1·27 (95% confidence interval 0·95 to 1·70)), as were all causes of death related to smoking (rate ratio 1·30 (0·91 to 1·85)) and mortality from lung cancer (rate ratio 2·41 (0·45 to 12·83)) and ischaemic heart disease (rate ratio 2·01 (1·21 to 3·35)). When passive smokers were divided into high and low exposure groups on the basis of the amount smoked by their cohabitees those highly exposed had higher rates of symptoms and death.

Conclusion—Exposure to environmental tobacco smoke cannot be regarded as a safe involuntary habit.
TABLE V—Age and sex adjusted mortality per 10 000 per year by category of exposure to cigarette smoke. Figures in parentheses are actual numbers of deaths

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Passive smoking</th>
<th>Single smoking</th>
<th>Double smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes</td>
<td>83·1 (99)</td>
<td>97·4 (164)</td>
<td>160·0 (420)</td>
<td>155·6 (734)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>1·6 (2)</td>
<td>5·0 (7)</td>
<td>23·2 (54)</td>
<td>21·4 (93)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>27·3 (30)</td>
<td>47·7 (54)</td>
<td>61·0 (171)</td>
<td>60·7 (260)</td>
</tr>
<tr>
<td>All causes of death related to smoking</td>
<td>60·8 (71)</td>
<td>72·2 (104)</td>
<td>130·4 (362)</td>
<td>129·9 (592)</td>
</tr>
</tbody>
</table>

TABLE VI—Age adjusted prevalence of respiratory and cardiovascular symptoms and age standardised mortality per 10 000 per year for women in control and passive smoking groups. Figures in parentheses are numbers of actual cases

<table>
<thead>
<tr>
<th></th>
<th>Controls (n=489)</th>
<th>Low exposure (n=754)</th>
<th>High exposure (n=541)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory symptoms:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infected sputum</td>
<td>2·1 (10)</td>
<td>2·4 (18)</td>
<td>3·1 (17)</td>
</tr>
<tr>
<td>Persistent sputum</td>
<td>6·4 (31)</td>
<td>5·8 (45)</td>
<td>8·6 (46)</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>12·7 (60)</td>
<td>11·2 (84)</td>
<td>16·2 (88)</td>
</tr>
<tr>
<td>Hypersecretion</td>
<td>4·1 (19)</td>
<td>3·8 (29)</td>
<td>5·7 (30)</td>
</tr>
<tr>
<td><strong>Cardiovascular symptoms:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina</td>
<td>3·6 (17)</td>
<td>4·1 (32)</td>
<td>5·8 (31)</td>
</tr>
<tr>
<td>Major abnormality found on electrocardiogram</td>
<td>0·4 (2)</td>
<td>1·1 (8)</td>
<td>0·5 (2)</td>
</tr>
<tr>
<td><strong>Mortality:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All causes</td>
<td>58·3 (32)</td>
<td>64·6 (70)</td>
<td>87·8 (54)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>3·2 (1)</td>
<td>2·5 (2)</td>
<td>5·7 (3)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>6·8 (3)</td>
<td>14·2 (14)</td>
<td>28·0 (16)</td>
</tr>
<tr>
<td>All causes of death related to smoking</td>
<td>34·9 (17)</td>
<td>35·2 (39)</td>
<td>47·3 (30)</td>
</tr>
</tbody>
</table>
Nitroglycerin and Heart Disease

Cardiovascular Mortality among Munitions Workers Exposed to Nitroglycerin and Dinitrotoluene

Stayner LT, Dannenberg AL, Thun M, Reeve G, Bloom TF, Boeniger M, Halperin W


A study of cardiovascular mortality among munitions workers exposed to nitroglycerin and dinitrotoluene (DNT) was conducted. The cohort consisted of 15654 current or former white male employees at a United States Army munitions factory in Radford, Virginia. A total of 5529 were potentially exposed to nitroglycerin and 4989 to DNT; 5136 were exposed to neither. The vital status of the subjects was determined on December 31, 1982. Death certificates were reviewed, and standardized mortality ratios (SMRs) and rate ratios (SRRs) were computed using the general United States population as the reference. Data were also analyzed by Poisson regression techniques. Mortality from all causes was close to that expected for the nitroglycerin and DNT exposed and unexposed subjects; SMRs were 1.03, 1.00, and 0.99, respectively. Mortality from cerebrovascular disease was less than expected in nitroglycerin and DNT exposed subjects; SMRs were 0.90 and 0.95, respectively. Death due to ischemic heart disease (IHD) was close to that expected in DNT exposed subjects, but slightly increased in subjects exposed to nitroglycerin (SMRs 0.98 and 1.07). When expressed as SRRs, IHD mortality was significantly increased in nitroglycerin subjects under the age of 35 (SRR 5.46). Cerebrovascular mortality was elevated in subjects 55 to 59 years old exposed to DNT (SRR 4.46). Poisson regression analysis showed a significant interaction between age and nitroglycerin exposure for IHD mortality. The strongest effect was observed in workers actively exposed to nitroglycerin before the age of 45 (SRR 3.30).
Table 8. Standardized rate ratios (SRR) and 95% confidence intervals (95% CI) for ischemic heart disease by time since last exposure to nitroglycerin.

<table>
<thead>
<tr>
<th>Time since last exposure to nitroglycerin&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Observed deaths (N)</th>
<th>SRR&lt;sup&gt;a&lt;/sup&gt;</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active</td>
<td>19</td>
<td>1.17</td>
<td>0.23–5.92</td>
</tr>
<tr>
<td>1 month—&lt;5 years</td>
<td>36</td>
<td>0.76</td>
<td>0.32–1.82</td>
</tr>
<tr>
<td>≥5 years</td>
<td>289</td>
<td>1.22</td>
<td>0.82–1.71</td>
</tr>
</tbody>
</table>

<sup>a</sup> Standardized for age, calendar, and employment status (active versus retired) with the use of the unexposed group as the reference group and the person-years distribution of the unexposed group as the weights.

<sup>b</sup> Active = less than one month since last exposure to nitroglycerin, 1 month—<5 years = between 1 month and less than 5 years since last exposure to nitroglycerin, ≥5 years = 5 or more years since last exposure to nitroglycerin.

Figure 1. Mortality rate ratios for ischemic heart disease calculated from the Poisson regression for active nitroglycerin exposure by age in years (number of cases) (**P<0.0001**). The model controls for age, year, employment, and exposure to dinitrotoluene.
Shift Work and Heart Disease

- **Definition of shift work**
  - Shift work involves working outside the daylight hours.

- **Atypical work hours**
  - In many cases working hours follow a well-defined schedule, which can be changed only in exceptional circumstances (child sick, unexpected family event, etc).
  - In situations with non-negotiable working hours, the degree of predictability becomes a determinant element.

Shift Work and Heart Disease

Health and safety effects of shift work
- Immediate effects
  - Sleep
    - Circadian rhythm, performance and safety
    - Interference social and family life
  - Long-term health effects
    - Digestive problems
    - Heart disease
Shift Work and Heart Disease

Shift Work, Shift Change, and Risk of Death From Heart Disease at Work

Steenland K, Fine L


The effect of current shift and recent shift change, on risk of contracting ischemic heart disease, was studied among workers at heavy equipment factories. A nested case/control study was conducted in a cohort of 21,000 men at four heavy equipment factories. The study compared 163 men who died of ischemic heart disease at work or within a week of having worked to control workers. The cases had no prior history of heart disease. Each case was matched with five controls, based on age, factory and race. Shifts worked by cases and controls were determined from personnel records. Data were analyzed by conditional logistic regression with retained matching. Mean time worked on the current shift, at the time of case death, was 9 years, with 72% of study subjects working on the first shift, 22% on second shift and 6% on third shift. Differences in heart disease associated with current shifts were not detected. Recent change from second or third, to first shift lowered the risk of heart disease initially, but the effect decreased with time. There was no corresponding negative effect associated with changing from first to second or third shifts. The authors conclude that there was little evidence for effects on risk of death from heart disease, due to current shift. Their data suggest a temporary lowering of risk after change from second or third to first shift.
Stress and Heart Disease

A Follow-up Study of Job Strain and Heart Disease Among Males in the NHANES1 Population

Steenland K, Johnson J, Nowlin S


The relationship between job strain and heart disease was evaluated via the National Health and Nutrition Survey 1 (NHANES1) completed by a sample of US workers from 1971 to 1975. Because of the lack of job strain scores for women, the study was restricted to 3,575 men with no history of heart disease. Analyses were conducted using Cox regression by means of SAS PHREG. Two principal scores were analyzed: job control (decision authority and latitude) and job demand. Job control and job demand were strongly and positively correlated, and both were positively correlated with education and income, and negatively correlated with systolic blood pressure. Job demand was negatively correlated with age, but job control showed no correlation. Job demand remained highly correlated with job control after adjusting for education, age, and race for white and blue collar workers. Blue collar workers in high control and high demand jobs had a significantly decreased risk of heart disease compared with other blue collar workers. The same trend for white collar workers did not appear. The authors conclude that there is no evidence from this study of an increased risk of heart disease for those with high strain jobs, however, those with the highest job control may have a significantly decreased risk of heart disease. The authors suggest that the variable of job demand should be measured in future studies.
Stress and Heart Disease
What Workers Say about Stress on the Job

Survey by Northwestern National Life
Percentage of workers who report their job is “very or extremely stressful.”

Survey by the Families and Work Institute
Percentage of workers who report they are “often or very often burned out or stressed by their work.”

Survey by Yale University
Percentage of workers who report they feel “quite a bit or extremely stressed at work.”
Stress and Heart Disease

NIOSH Model of Job Stress

Stressful Job Conditions

Individual and Situational Factors

Risk of Injury and Illness
Recently Study Results

Chemical compounds
Magnetic fields
Atypical work hours
Stress and endocrine function
Nano-particles
Production of Synthetic Organic Chemicals and the Percentage of Overweight Adults in the US

Tributyltor Chloride (TBT) on Adipocyte Differentiation and Obesity
Exposure Assessment

Job titles in the NLMS were classified according to 1970 Bureau of Census (BOC) occupational codes. To determine potential MF exposure for each job reported in the NLMS, we first converted the 1970 BOC occupational codes to 1980 BOC occupational codes. Subsequently, these 1980 BOC codes were linked with a job-exposure matrix (JEM) developed previously by Bowman and colleagues (7). Personal monitoring and spot measurements on power-frequency magnetic fields were compiled based on a comprehensive search of the peer-reviewed literature and unpublished sources. Workday average magnetic field measurements were pooled to estimate arithmetic and geometric mean exposure for 32.1% of the 502 1980 BOC occupations. Estimations based on categories judged to have similar magnetic field exposures were assigned to those occupations lacking direct measurements (52.7%). Where reliable exposure estimates were not possible (15.2% of the 1980 BOC occupations), subjects holding these jobs in the NLMS (3.6% of eligible population) were excluded from our analyses. Given the high correlation between geometric and arithmetic means in this JEM (data not shown), we linked each 1980 BOC occupation only to the arithmetic mean estimates of magnetic field exposure.

In the current analysis, magnetic field exposures were divided a priori into three intervals (<0.15, 0.15 to <0.20, \( \geq 0.20 \) \( \mu T \)), analogous to prior studies. Furthermore, given the sizable number of CVD deaths in the greater than 0.20 \( \mu T \) exposure group, we also examined exposure–response relationships using four categories (<0.15, 0.15 to <0.20, 0.20 to <0.30, and \( \geq 0.30 \) \( \mu T \)).
Atypical Work Hours and Metabolic Syndrome Among Police Officers

John M. Violanti, PhD; Cecil M. Burchfiel, PhD, MPH; Tara A. Hartley, MPA, MPH; Anna Mnatsakanova, MS; Desta Fekedulegn, PhD; Michael E. Andrew, PhD; Luenda E. Charles, PhD, MPH; Bryan J. Vila, PhD

ABSTRACT: This study examined whether atypical work hours are associated with metabolic syndrome among a random sample of 98 police officers. Shift work and overtime data from daily payroll records and reported sleep duration were obtained. Metabolic syndrome was defined as elevated waist circumference and triglycerides, low HDL cholesterol, hypertension, and glucose intolerance. Multivariate analysis of variance and analysis of covariance models were used for analyses. Officers working midnight shifts were on average younger and had a slightly higher mean number of metabolic syndrome components. Stratification on sleep duration and overtime revealed significant associations between midnight shifts and the mean number of metabolic syndrome components among officers with less sleep ($p = .013$) and more overtime ($p = .007$). Results suggest shorter sleep duration and more overtime combined with midnight shift work may be important contributors to the metabolic syndrome.

KEYWORDS: cardiovascular disease, overtime, police officers, shift work, sleep

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Cortisol patterns and brachial artery reactivity in a high stress environment

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ABSTRACT

Chronic stress can result in frequent or persistent challenges of the hypothalamic-pituitary-adrenal (HPA) axis resulting in abnormal cortisol patterns and increased risk for cardiovascular disease (CVD). Police work is an environment replete with stress. The present article describes associations between cortisol, a biomarker of stress, and brachial artery flow mediated dilation (FMD) in police officers. A random sample stratified on gender (n = 100, 33% women) was generated from officers in a mid-sized urban department. Four salivary cortisol parameters were derived: after awakening, following a standardized high protein meal challenge, during the entire day, and after a dexamethasone suppression test. Continuous scan B-Mode ultrasound was used to measure percent change in brachial artery FMD following occlusion and release. Elevated cortisol secretion after awakening was significantly associated with impaired FMD in women, reflected by an inverse trend. Adjustment for age, smoking, and alcohol consumption did not appreciably alter this trend. A similar result was not evident among male officers. Responses of other cortisol challenges to the HPA axis were not associated with FMD. In conclusion, increased cortisol secretion after awakening was independently associated with impaired FMD in female police officers only, indicating a possible link between HPA axis stress response and subclinical CVD. However, because associations were not found with other cortisol parameters and were not evident in male officers, replication of these findings with a prospective study design may be warranted.

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Potential *in vitro* effects of carbon nanotubes on human aortic endothelial cells

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**Abstract**

Respiratory exposure of mice to carbon nanotubes induces pulmonary toxicity and adverse cardiovascular effects associated with atherosclerosis. We hypothesize that the direct contact of carbon nanotubes with endothelial cells will result in dose-dependent effects related to altered cell function and cytotoxicity which may play a role in potential adverse pulmonary and cardiovascular outcomes. To test this hypothesis, we examined the effects of purified single- and multi-walled carbon nanotubes (SWCNT and MWCNT) on human aortic endothelial cells by evaluating actin filament integrity and VE-cadherin distribution by fluorescence microscopy, membrane permeability by measuring the lactate dehydrogenase (LDH) release, proliferation/viability by WST-1 assay, and overall functionality by tubule formation assay. Marked actin filament and VE-cadherin disruption, cytotoxicity, and reduced tubule formation occurred consistently at 24 h post-exposure to the highest concentrations [50–150 μg/10⁶ cells (1.5–4.5 μg/ml)] for both SWCNT and MWCNT tested in our studies. These effects were not observed with carbon black exposure and carbon nanotube exposure in lower concentrations [1–10 μg/10⁶ cells (0.04–0.4 μg/ml)] or in any tested concentrations at 3 h post-exposure. Overall, the results indicate that SWCNT and MWCNT exposure induce direct effects on endothelial cells in a dose-dependent manner.

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Original Contribution

Vascular Function, Inflammation, and Variations in Cardiac Autonomic Responses to Particulate Matter Among Welders

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Patients with health conditions associated with impaired vascular function and inflammation may be more susceptible to the adverse health effects of fine particulate (particulate matter with a mass median aerodynamic diameter of ≤2.5 μm (PM$_{2.5}$)) exposure. In 2006, the authors conducted a panel study to investigate directly whether vascular function and inflammation (assessed by C-reactive protein) modify PM$_{2.5}$-associated reductions in heart rate variability among 23 young male workers (mean age, 40 years) from Massachusetts. Concurrent 24-hour ambulatory electrocardiogram and personal PM$_{2.5}$ exposure information was collected over a total of 36 person-days, including either or both welding and nonwelding days. Linear mixed models were used to examine the 5-minute standard deviation of normal-to-normal intervals (SDNN) in relation to the moving PM$_{2.5}$ averages in the preceding 1–4 hours. C-reactive protein levels and 3 measures of vascular function (augmentation index, mean arterial pressure, and pulse pressure) were determined at baseline. The authors observed an inverse association between the 1-hour PM$_{2.5}$ and 5-minute SDNN. Greater SDNN declines were observed among those with C-reactive protein ($P_{\text{interaction}} < 0.001$) and augmentation index ($P = 0.06$) values at or above the 75th percentile and pulse pressure values below the 75th percentile ($P < 0.001$). Systemic inflammation and poorer vascular function appear to aggravate particle-related declines in heart rate variability among workers.

augmentation; C-reactive protein; disease susceptibility; heart rate; inflammation; particulate matter; vascular diseases; welding
Circulating adhesion molecules after short-term exposure to particulate matter among welders

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ABSTRACT
Background: Studies from several countries indicate that welders experience increased risk of mortality and morbidity from ischemic heart disease. Although the underlying mechanisms are unclear, vascular responses to particulate matter contained in welding fumes may play a role. To investigate this, we studied the acute effects of welding fume exposure on the endothelial component of vascular function, as measured by circulating adhesion molecules involved in leukocyte adhesion (sICAM-1 and sVCAM-1) and coagulation (vWF).

Methods: A panel of 26 male welders was studied repeatedly across a 6 h work-shift on a high exposure welding day and/or a low exposure non-welding day. Personal PM₂₅ exposure was measured throughout the work-shift. Blood samples were collected in the morning (baseline) prior to the exposure period, immediately after the exposure period, and the following morning. To account for the repeated measurements, we used linear mixed models to evaluate the effects of welding (binary) and PM₂₅ (continuous) exposure on each blood marker, adjusting for baseline blood marker concentration, smoking, age and time of day.

Results: Welding and PM₂₅ exposure were significantly associated with a decrease in sVCAM-1 in the afternoon and the following morning and an increase in VWF in the afternoon.

Conclusions: The data suggest that welding and short-term occupational exposure to PM₂₅ may acutely affect the endothelial component of vascular function.

What this paper adds

- Welders are occupationally exposed to fine particulate matter (PM₂₅) and experience increased risk of mortality and morbidity from ischemic heart disease.
- However, the mechanisms linking PM₂₅ inhalation with adverse cardiovascular health are unclear.
- To date, few studies have investigated exposure–response relationships between occupational PM₂₅ exposure and subclinical cardiovascular outcomes.
- Findings from this study suggest that short-term occupational exposure to welding fume and PM₂₅ may acutely affect the endothelial component of vascular function, as measured by circulating adhesion molecules.
- This study contributes to a growing body of literature on the cardiovascular health effects of occupational exposure to PM₂₅ and evidence that alterations in vascular function may play a role in PM-associated cardiovascular disease.
Special Article

Occupation as Socioeconomic Status or Environmental Exposure? A Survey of Practice Among Population-based Cardiovascular Studies in the United States

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Decisions about how occupation is used in epidemiologic research can affect conclusions about the importance of socioeconomic and environmental factors in explaining disparities for outcomes such as cardiovascular disease. A review of practices in the collection and use of occupational data was conducted among population-based cardiovascular studies in the United States. Studies were identified for review from the National Heart, Lung, and Blood Institute website and the biomedical database, Computer Retrieval of Information on Scientific Projects, by use of selected criteria. Data collection instruments and study publications were retrieved and reviewed for 30 of 33 studies (91%). Most of the studies (63%) collected at least descriptive occupational data, and more than half (60%) collected data on workplace hazards. The reviewed studies produced 80 publications in which occupational data were used in analyses, most often as an indicator of socioeconomic status. Authors rarely acknowledged known conceptual and empirical links among socioeconomic status, employment stability, and working conditions. Underutilization of data on workplace conditions was found. Existing data could be used more effectively to examine the contribution of work-related social and environmental conditions to the development of modifiable cardiovascular disease through multiple pathways.

cardiovascular diseases; environment and public health; epidemiologic research design; occupations; social class
Future Occupational Heart Disease

**Exposure**
- Nano particle
- Stress
- Work hour
- Physical
- Chemical
- Biological
- ...

**Outcome**
- CAD/CVA/SD
- HTN (BP)
- Endothelial
- Arrhythmia (ECG)
- MetS/Endocrine
- Atherosclerosis
- Cardiomyopathy

Potential Confounders
Thank You for Your Attention!