

# Is Job-Related Stress the Link Between Cardiovascular Disease and the Law Enforcement Profession?

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**Objective:** To determine whether job-related stress is associated with alterations in pro- and anti-atherogenic inflammatory mediators among law enforcement officers. **Methods:** Markers of vascular inflammation and the self-reported stress measures of perceived stress, vital exhaustion, job strain, effort-reward imbalance, and social support were compared between officers ( $N = 444$ ) and non-officers ( $N = 166$ ). **Results:** Officers had higher levels of IL-1 $\beta$ , IL-6, IL-10, and TNF- $\alpha$  and lower levels of C-reactive protein and fibrinogen. No more than 4% of the variability in any of the inflammatory mediators was explained by any stress measure for either the two groups or the entire sample. **Conclusions:** Law enforcement officers may be at an increased risk for cardiovascular disease due to a relatively greater pro-inflammatory vascular environment. However, this increased risk cannot be attributed to either chronic stress or the work-related stress measures assessed here.

Law enforcement officers have higher cardiovascular disease (CVD)-related morbidity and mortality than the general population.<sup>1-3</sup> This increased mortality is frequently attributed to an increased prevalence of CVD risk factors such as physical inactivity, hypercholesterolemia, hypertension, tobacco use, obesity, and hyperinsulinemia.<sup>4,5</sup> Because a significant number of individuals have these risk factors,<sup>6</sup> the effects of risk factors on CVD risk among members of the law enforcement profession may simply reflect characteristics of the general U.S. population rather than be occupationally related. Cross-sectional and longitudinal comparisons suggest that CVD risk due to traditional risk factors is similar among both groups.<sup>7</sup> Thus, the greater prevalence of CVD among officers cannot be readily explained by well-known risk factors. Moreover, the association of CVD with duration in the law enforcement profession is not explained by a disproportionate increase in conventional risk factors.<sup>8,9</sup>

Most recent reviews agree that a relationship exists between psychosocial stress and CVD.<sup>10,11</sup> This is likely also true of law enforcement officers. For example, a higher prevalence of CVD in highly stressed officers was related to the duration of employment but independent of age.<sup>8</sup> The officers in the highest quintile of stress were 2.5 times more likely to have been diagnosed with CVD than officers in the lowest quintile. The unique and pronounced effects of stress in policing are well recognized.<sup>12-14</sup> Critical incident stressors markedly affect perceived work stress,<sup>13,15</sup> yet these are typically acute, short-term stressors and mechanisms are frequently present to assist the officer in handling this stress (eg, stress debriefings). Nevertheless, chronic stressors are typically organiza-

tionally based and often ignored. It is likely that chronic stressors are more injurious to officer health than these acute stressors. Chronic stressors predict psychological distress, including posttraumatic stress disorder, more strongly than cumulative exposure to critical incidents among police.<sup>16</sup> Thus, organizational factors have the strongest influence of all types of stressors in this population.<sup>13,17</sup>

CVD is increasingly recognized as an inflammatory disease. Several inflammatory mediators are important contributors to atherogenesis.<sup>18,19</sup> Psychological stress can affect the balance of pro- and anti-inflammatory mediators.<sup>20,21</sup> These findings suggest a possible mechanism underlying the increased prevalence of CVD in law enforcement officers seen in the absence of marked elevations in more traditional CVD risk factors—namely, officers have a pro-inflammatory environment in the vasculature due to elevations in stress. This hypothesis has yet to be tested. Therefore, the purpose of the present investigation was to determine the extent to which officers have a higher risk of CVD due to alterations in pro- and anti-atherogenic inflammatory mediators that are associated with job-related stress. More specifically, this study quantified the levels of pro- and anti-atherogenic inflammatory mediators in law enforcement officers and determined whether chronic job-related stress was associated with these inflammatory mediators.

## METHODS

This study was designed to compare the outcome measures of interest in a sample of law enforcement officers to an otherwise similar general population sample. These measures were inflammatory mediators (ie, C-reactive protein, fibrinogen, IL-1 $\beta$ , IL-4, IL-6, IL-10, TNF- $\alpha$ ), perceived stress, three measures of work-related stress germane to law enforcement (ie, vital exhaustion, job strain, effort-reward imbalance), a moderator of stress (ie, social support), and potential confounding factors (ie, obesity, physical activity, recent infections/illnesses, chronic aspirin ingestion, risk for CVD via the Framingham Risk Score, and presence of the metabolic syndrome).

## Participants

Sworn officers of the Iowa Department of Public Safety were invited to participate in this study as they underwent their annual medical evaluation. Of these 531 officers, 466 (88%) agreed to participate; 322 officers (69%) were members of the State Highway Patrol and the remaining 144 officers were associated with either the Division of Criminal Investigation, Division of Narcotics Enforcement, or the State Fire Marshal's office. The comparison group consisted of subjects who were demographically and socioeconomically similar to the officers but not involved in law enforcement. The comparison group ( $n = 171$ ) was recruited via four methods. First, participating officers were asked to identify several non-law enforcement friends who might be willing to participate in the study. These nominees were contacted via telephone and asked to participate. Second, employees of a local manufacturing plant were informed of the study by company intranet and invited to participate. Interested employees contacted the researchers directly. Third, and in a similar manner, local professional firefighters were recruited. Finally, participants recruited other subjects by word-of-mouth.

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Potential participants were screened and excluded from the study if they reported having a history of CVD, recent surgery, or any acute illness. Subjects were also excluded from the study if they reported ingesting large quantities of either nonsteroidal anti-inflammatory medications or any glucocorticoids, had ever been treated for an autoimmune disease, or if they provided incomplete data. As part of this initial screening, tobacco use and current medications were determined. Based on this screening, all participants were apparently healthy at the time of the study. Thus, 444 officers (22 females, 322 males) and 166 comparison participants (34 females, 132 males) were included in the analyses.

## Data Collection Procedures

### Physiological Measures

Height was measured to the nearest 0.5 cm using a stadiometer (Seca, Hanover, MD) whereas weight was assessed to the nearest 0.1 kg on a digital scale (Befour, Inc, Saukville, WI) with the participant wearing light clothing but no shoes. After 5 minutes seated rest, blood pressure was measured twice with a mercury sphygmomanometer. Venipuncture was used to obtain 30 mL of blood after a 10 hour fast. Half of this blood sample was used for assessment of lipids and glucose by a laboratory, which meets the quality control standards of the U.S. CDC Lipid Standardization Program (Quest Diagnostics, Wood Dale, IL). The remaining 15 mL of blood was allowed to clot for 2 hours with the resulting serum frozen at  $-80^{\circ}\text{C}$  for later analysis of inflammatory mediators. The latter consisted of pro-atherogenic IL-1 $\beta$ , IL-6, TNF- $\alpha$ , C-reactive protein and fibrinogen, and anti-atherogenic IL-4 and IL-10. High-sensitivity ELISA kits (Millipore, Billerica, MA) were used. All samples were run in duplicate.

### Psychological Measures

Stress was assessed using the Perceived Stress Scale (PSS).<sup>22</sup> This 14-item scale measures the degree to which a person appraises situations in his/her life as stressful although it does not indicate the source of stress. Cronbach  $\alpha$  for the PSS was 0.75.<sup>23</sup>

Three distinct forms of stress, felt to be relevant to the law enforcement profession, were also assessed. These were job strain, vital exhaustion, and effort-reward imbalance. Job strain occurs in situations where high psychological demands and low control, or decision latitude, occur simultaneously. It was assessed using the Job Content Questionnaire developed by Karasek et al.<sup>24</sup> A 25-item modified version included the following scales: a) decision latitude, also described as job control, which is divided further into decision authority and skill discretion, b) psychological demands, and c) job-related social support. This version was developed by Bosma et al.<sup>25</sup> and used in the Whitehall II study. Bosma et al.<sup>25</sup> reported Cronbach  $\alpha$  to be 0.84 for the decision latitude and psychological demands as a combined 15-item measure of job control, and 0.67 for job-related social support.

Vital exhaustion characterizes feelings of excessive fatigue or lack of energy, irritability, and demoralization, usually attributed to overwork or problems at work. It was assessed using a 9-item version of Form B of the Maastricht Questionnaire developed by Appels<sup>26</sup> and modified by Kopp et al.<sup>27</sup> Cronbach  $\alpha$  for the 9-item version was 0.83, and the modified scale correlated strongly with the original 21-item version ( $r = 0.93$ ).

Effort-reward imbalance was determined using a 17-item scale,<sup>28</sup> which assessed intrinsic effort or "overcommitment," extrinsic effort, and rewards. Intrinsic effort was assessed by items that tap a) need for approval, b) competitiveness and latent hostility, c) impatience and disproportionate irritability, and d) inability to withdraw from work obligations. Extrinsic effort was assessed by items that tap a) the frequency of being stressed by time pressure, b) responsibility, c) overtime work, and d) increasing responsibility.

Rewards were assessed by a) perceived esteem by colleagues and superiors, b) the lack of job insecurity, and c) the lack of undesirable job changes.

Finally, social support may affect the relationship between job-related stress and inflammatory mediators. It was assessed using the 24-item version of the Social Provisions Scale.<sup>29</sup> This scale has been associated with psychological and physical health outcomes in other groups, which serve the public, such as nurses.<sup>30</sup> It provides a global index of social support based on the subscales of attachment, social integration, reassurance of worth, reliable alliance, guidance, and opportunity for nurturance. Reliability is good ( $\alpha = 0.85$ ).<sup>29</sup>

## Data Analyses

Height and weight were used to calculate body mass index (BMI,  $\text{kg} \cdot \text{m}^{-2}$ ). Gender, age, LDL-cholesterol, HDL-cholesterol, blood pressure, smoking status (defined as having regularly used tobacco within the past 12 months), and presence of diabetes (defined as a fasting plasma glucose  $>126 \text{ mg} \cdot \text{dl}^{-1}$  or currently using antidiabetic medications) were used to determine each subject's Framingham Risk Score.<sup>31</sup> This score is an estimate of the probability that the subject will have clinically significant CVD within the next 10 years.

Risk factors for the metabolic syndrome were identified according to the National Cholesterol Education Program Adult Treatment Panel III criteria.<sup>32</sup> However, a BMI  $>30 \text{ kg} \cdot \text{m}^{-2}$  was used instead of waist circumference as the criterion for abdominal obesity.<sup>33</sup> Thus, the risk factors were BMI  $\geq 30 \text{ kg} \cdot \text{m}^{-2}$ , triglycerides  $\geq 150 \text{ mg} \cdot \text{dl}^{-1}$ , HDL-cholesterol  $<40 \text{ mg} \cdot \text{dl}^{-1}$ , blood pressure  $\geq 130 \text{ mm Hg}$  systolic and/or  $\geq 85 \text{ mm Hg}$  diastolic or currently using antihypertensive medications, and fasting plasma glucose  $\geq 110 \text{ mg} \cdot \text{dl}^{-1}$  or currently using antidiabetic medications.

All data were log transformed, as needed, to improve the distribution of the scores. Differences between the two groups were initially assessed using independent sample *t*-tests. To determine the extent to which psychosocial factors were most associated with changes in these inflammatory mediators, the measures of stress (ie, perceived stress, vital exhaustion, job strain, effort-reward imbalance) and social support were examined as predictors of the measures of pro- and anti-atherogenic inflammatory mediators using hierarchical regression analyses. Possible confounding variables in these relationships, such as obesity, physical activity, shift work patterns, recent infections/illness, and the Framingham Risk Score, were entered into the regression equation in step 1 and measures of stress and social support entered in step 2. Separate regression analyses were conducted for each of the pro- and anti-atherogenic inflammatory mediators. Multiple regression analyses tested whether or not the relationships between the measures of stress and social support and the inflammatory mediators varied for the two groups of participants (ie, officers and non-police participants). This procedure permitted testing for interactions between group membership and the predictor variables. All data are presented as mean  $\pm$  SD with statistical significance set at  $P < 0.05$ .

## RESULTS

CVD risk characteristics are presented in Table 1. The officers and non-officer participants differed significantly on some of the traditional CVD risk factors. However, neither global CVD risk, expressed as the Framingham Risk Score, nor the number of metabolic syndrome risk factors differed between the two groups. With the exception of IL-4, the inflammatory mediators differed significantly between the two groups. Concentrations of four of the remaining six mediators were approximately two to three times higher in the officers (range = 1.8 to 5.2). The other two mediators,

**TABLE 1.** CVD Risk Factors and Inflammatory Mediators of this Risk

Variable	Law Enforcement	Control	Cohen's <i>d</i>
CVD risk factors			
Age (y)	37.4 ± 9.2	42.0 ± 11.3**	0.45
BMI (kg · m <sup>-2</sup> )	28.6 ± 4.0	28.3 ± 5.9	
Total cholesterol (mg · dl <sup>-1</sup> )	193.3 ± 5.2	192.7 ± 36.3	
LDL-cholesterol (mg · dl <sup>-1</sup> )	119.4 ± 0.3	117.7 ± 28.9	
HDL-cholesterol (mg · dl <sup>-1</sup> )	47.4 ± 1.2	49.7 ± 13.9*	0.21
LDL/HDL ratio	2.6 ± 0.9	2.5 ± 0.8	
Triglycerides (mg · dl <sup>-1</sup> )	134.7 ± 7.2	133.3 ± 84.4	
Glucose (mg · dl <sup>-1</sup> )	92.5 ± 9.7	93.3 ± 22.0	0.15
Mean arterial pressure (mm Hg)	99.3 ± 7.8	95.9 ± 0.6**	0.30
Framingham risk score (%)	5.7 ± 3.1	5.1 ± 4.2	
Metabolic syndrome risk (no. factors)	1.4 ± 1.2	1.3 ± 0.4	
Proatherogenic inflammatory mediators			
IL-1β (pg · ml <sup>-1</sup> )	6.3 ± 14.9	1.1 ± 2.0**	0.49
IL-6 (pg · ml <sup>-1</sup> )	29.8 ± 40.7	15.1 ± 0.3**	0.45
TNF-α (pg · ml <sup>-1</sup> )	10.5 ± 18.2	5.8 ± 5.6**	0.35
C-reactive protein (pg · ml <sup>-1</sup> )	7.2 ± 1.7	10.6 ± 18.0**	0.27
Fibrinogen (pg · ml <sup>-1</sup> )	0.28 ± 0.14	0.34 ± 0.24**	0.32
Antiatherogenic inflammatory mediators			
IL-4 (pg · ml <sup>-1</sup> )	176.1 ± 223.4	147.6 ± 235.4	
IL-10 (pg · ml <sup>-1</sup> )	46.5 ± 13.4	20.5 ± 73.2**	0.25

Data adjusted for age.  
\**P* < 0.05; \*\**P* < 0.01.

**TABLE 2.** Stress Markers

Variable (Possible Range)	Law Enforcement	Control	Cohen's <i>d</i>
Perceived stress (0–56)	16.7 ± 6.1	21.5 ± 6.7**	0.79
Job strain (1–16)	3.4 ± 0.7	3.6 ± 0.9**	0.24
Job demands (4–16)	10.4 ± 1.8	11.1 ± 2.7**	0.39
Job control (18–72)	56.6 ± 6.4	55.4 ± 8.8	
Vital exhaustion (9–27)	13.8 ± 4.5	15.3 ± 4.4**	0.36
Sense of exhaustion (1–3)	1.3 ± 0.7	1.6 ± 0.9**	0.37
Effort-reward imbalance (0.25–4.0)	0.88 ± 0.14	0.79 ± 0.20**	0.54
Extrinsic effort (5–20)	15.5 ± 1.8	13.2 ± 2.7**	0.96
Intrinsic effort (4–16)	9.3 ± 2.2	9.7 ± 2.3*	0.18
Rewards (8–32)	28.5 ± 2.7	27.2 ± 2.8**	0.47
Social support (20–96)	81.3 ± 9.9	80.7 ± 9.8	

Data adjusted for age.  
\**P* < 0.05; \*\**P* < 0.01.

fibrinogen and C-reactive protein, were 25% and 33% lower in the officers than the non-officer participants.

Perceived stress was higher in the control participants than the officers (Table 2). Job strain and vital exhaustion were also higher in the control participants. Effort-reward imbalance was higher in the officers, primarily due to relatively greater extrinsic effort. Social support was high and did not differ between the two groups.

Nevertheless, in assessing the extent to which group differences in stress markers explain differences in the inflammatory mediators, regression analyses indicated that less than 4% of the variance in any of the inflammatory mediators was explained by any of the stress measures. This remained the case when the data were analyzed either within groups or across the entire cohort.

Finally, because there may be gender-related differences in these findings, the data were also analyzed after stratifying by

gender. For some of the inflammatory and stress variables, the women differed from the men. Specifically, the women had higher C-reactive protein levels (15.9 ± 14.0 vs 8.0 ± 16.4 pg · ml<sup>-1</sup>), vital exhaustion (16.1 ± 4.7 vs 14.0 ± 4.5), and social support (83.2 ± 8.0 vs 80.8 ± 10.1) than the men but lower levels of extrinsic effort (13.2 ± 2.9 vs 15.1 ± 2.1). More importantly, these differences were not unique to either the law enforcement or the comparison group and do not alter the group differences presented in Tables 1 and 2.

## DISCUSSION

The purpose of the present investigation was to determine the extent to which law enforcement officers have a higher risk of CVD due to alterations in pro- and anti-atherogenic inflammatory

mediators, which are associated with job-related stress. There were group differences among the inflammatory mediators as well as group differences among the stress measures. Indeed, group membership (ie, law enforcement or control) had a relatively strong association with many of these biological and psychological factors. Nevertheless, group differences in job-related stress were neither associated with, nor predictive of, group differences in the inflammatory mediators. This study therefore suggests that the law enforcement profession may be at an increased risk for CVD through its association with a pro-inflammatory vascular milieu. The mechanism responsible for this association does not appear to be job-related stress. It is also not due to elevations in traditional CVD risk factors, because neither the Framingham Risk Score nor the prevalence of Metabolic Syndrome risk factors differed between the two groups.

Of the seven inflammatory mediators assessed here, five are generally considered pro-atherogenic (ie, IL-1 $\beta$ , IL-6, TNF- $\alpha$ , C-reactive protein, fibrinogen) whereas two are anti-atherogenic (ie, IL-4 and IL-10). Three pro-atherogenic mediators were higher in the officers whereas two were elevated in the comparison group; the magnitude of these differences was considerably larger in the former. One anti-atherogenic mediator did not differ between the two groups whereas the second was higher in the officers. Thus, there was not a consistent trend in the group differences and, because of the relatively large sample sizes used here, some of these statistically significant differences may be spurious.

Clarifying the extent to which these mediators differentially affect CVD risk between the two groups may be aided by comparing the effect sizes of these differences. Larger effect sizes imply larger and therefore, more likely “real” differences between the groups. Effect sizes, expressed as Cohen’s *d*,<sup>34</sup> were subsequently calculated (Table 1). Of the six inflammatory mediators, the largest effect sizes were found in the three pro-atherogenic inflammatory mediators that were higher in the officers than the non-officers. Consequently, the data suggest that the officer group had a more pronounced pro-atherogenic inflammatory milieu than the comparison group.

For several reasons, the measures of stress assessed here were not associated with the pro-inflammatory environment seen in the officers. First, the comparison group was more stressed than the officers. Besides having a higher mean perceived stress score, a disproportionately high number of controls were in the highest quartile of stress (ie, 45% of the control group vs 17% of the officers). However, none of the inflammatory mediators differed between the highest quartile of subjects and the lowest quartile. Second, three of the four stress measures were higher in the non-officer comparison group than the officers, yet none of the stress measures were predictive of the inflammatory variables. Thus, chronic job-related stress does not appear to contribute to CVD in law enforcement officers by altering their inflammatory profile.

The mechanism(s) underlying the pro-inflammatory profile of the officers is uncertain. One candidate is shift work. Shift work is associated with CVD and this association often remains after traditional CVD risk factors are accounted for.<sup>35,36</sup> More than 77% of the officers reported having either shift work or working irregular, unscheduled hours compared with just 27% of the comparison group. It is unclear how shift work may contribute to increased CVD, although Sookian et al<sup>35</sup> recently reported that leukocyte count, a marker of inflammation, was associated with shift work. Early atherosclerosis, evidenced by a 2.2-fold higher risk of carotid plaques and higher carotid artery intima-media thickness, has been found in relatively young shiftworkers.<sup>36</sup> Of relevance to this study, this relationship remained after accounting for C-reactive protein and job strain. Police officers were also found to have higher carotid intima-media thicknesses than a relatively similar comparison

group and this difference remained after controlling for age, gender, CVD risk factors, and depression.<sup>37</sup> As acknowledged by these authors, a limitation of this study was that they did not assess any other psychosocial traits, such as work-related stress, or other biological mechanisms, such as inflammatory mediators. Based on these collective findings, we speculate that differences in shift work, rather than work-related stress, may affect inflammatory markers associated with an increased CVD risk.

A second, albeit related, factor may be differences in sleep quality. Poor sleep quality is associated with inflammation<sup>38</sup> and CVD mortality.<sup>39</sup> It is also more common in law enforcement officers than non-officers.<sup>40,41</sup> Although we did not assess sleep quality, anecdotal evidence suggests that many officers in this study routinely experience various markers of poor sleep, such as difficulty falling asleep, fitful sleep, or waking up tired. Accordingly, as with shift work, we speculate that differences in sleep quality may contribute to the differences in inflammatory markers seen here.

There are limitations to this study, which likely affect interpretation of the data. First, neither the officers nor the comparison group were highly stressed. The PSS score for the entire cohort was similar to that of 1235 subjects employed in diverse occupations<sup>23</sup> ( $18.0 \pm 6.6$  vs  $18.9 \pm 6.9$ , respectively). For each of the other three stress measures assessed here, the mean was less than half the range of possible scores. Thus, the association between work-related stress and mediators of inflammation may have been stronger had more stressed subjects been included. Second, there are numerous measures of work-related stress,<sup>42</sup> and it is possible that the measures used in this study were not the most appropriate for achieving the aims of the study. Nevertheless, in developing the study, care was taken to identify those constructs which seemed most relevant to police work. Conversations with seasoned law enforcement officers, both before and after the data collection, reinforced this view. Finally, the bulk of law enforcement officers assessed here were members of the highway patrol, or troopers. The job responsibilities of these officers differ somewhat from municipal police officers and this difference may affect the generalizability of the findings. However, the stress constructs used here assess aspects of chronic job-related stress that were felt to be universal to the law enforcement profession. This assertion is supported by the observation that neither the stress levels nor the inflammatory mediators of the troopers differed significantly from their non-trooper colleagues. The responsibilities of the latter are more comparable to those seen of their peers in municipal police departments. Thus, our findings are likely generalizable to other law enforcement officers.

In summary, this study suggests that law enforcement officers have an altered inflammatory profile that may be associated with the increased prevalence of CVD seen in this occupation. Although the mechanism underlying this pro-inflammatory milieu remains uncertain, it does not appear to be due to work-related stress.

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#### REFERENCES

1. Calvert GM, Merling JW, Burnett CA. Ischemic heart disease mortality and occupation among 16- to 60-year-old males. *J Occup Environ Med.* 1999; 41:960–966.
2. Dubrow R, Burnett CA, Gute DM, Brockert JE. Ischemic heart disease and acute myocardial infarction mortality among police officers. *J Occup Med.* 1988;30:650–654.
3. Franke WD, Collins SA, Hinz PN. Cardiovascular disease morbidity in an Iowa law enforcement cohort, compared with the general Iowa population. *J Occup Environ Med.* 1998;40:441–444.

4. Pyörälä M, Miettinen H, Halonen P, Laakso M, Pyörälä K. Insulin resistance syndrome predicts the risk of coronary heart disease and stroke in healthy middle-aged men: the 22-year follow-up results of the Helsinki policemen study. *Arterioscler Thromb Vasc Biol.* 2000;20:538–544.
5. Williams M, Pettratis M, Baechle T, Ryschon K, Campain J, Sketch M. Frequency of physical activity, exercise capacity and atherosclerotic heart disease risk in male police officers. *J Occup Med.* 1987;29:596–600.
6. Lloyd-Jones D, Adams R, Carnethon M, et al; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2009 update. A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2009;119:e21–e181.
7. Franke WD, Cox DF, Schultz DP, Anderson DF. Coronary heart disease risk in employees of Iowa's Department of Public Safety compared to a cohort of the general population. *Am J Ind Med.* 1997;31:733–737.
8. Franke WD, Ramey SL, Shelley MC II. Relationship between cardiovascular disease morbidity, risk factors, and stress in a law enforcement cohort. *J Occup Environ Med.* 2002;44:1182–1189.
9. Ramey SL, Downing NR, Franke WD. Milwaukee police department retirees: cardiovascular disease risk and morbidity among law enforcement officers. *AAOHN J.* 2009;57:448–453.
10. Hemingway H, Marmot M. Evidence based cardiology: psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. *BMJ.* 1999;318:1460–1467.
11. Krantz DS, McCeney MK. Effects of psychological and social factors on organic disease: a critical assessment of research on coronary heart disease. *Annu Rev Psychol.* 2002;53:341–369.
12. Finn P, Tomz JE. *Developing a Law Enforcement Stress Program for Officers and Their Families.* Washington, D.C.: National Institute of Justice; 1997.
13. Gershon RRM, Barocas B, Canton AN, Li X, Vlahov D. Mental, physical, and behavioral outcomes associated with perceived work stress in police officers. *Crim Justice Behav.* 2009;36:275–289.
14. Abdollahi MK. Understanding police stress. *J Forens Psychiatry Psychol.* 2002;2:1–24.
15. Gershon RM, Lin S, Li X. Work stress in aging police officers. *J Occup Environ Med.* 2002;44:160–167.
16. Liberman AM, Best SR, Metzler TJ, Fagan JA, Weiss DS, Marmar CR. Routine occupational stress and psychological distress in police. *Policing.* 2002;25:421–439.
17. Storch JE, Panzarella R. Police stress: state-trait anxiety in relation to occupational and personal stressors. *J Crim Justice.* 1996;24:99–107.
18. Libby P. Inflammation in atherosclerosis. *Nature.* 2002;420:868–874.
19. Ridker PM. Novel risk factors and markers for coronary disease. *Adv Intern Med.* 2000;45:391–418.
20. Miller D, O'Callaghan J. Neuroendocrine aspects of the response to stress. *Metabolism.* 2002;51:5–10.
21. Steptoe A, Humer M, Chida Y. The effects of acute psychological stress on circulating inflammatory factors in humans: a review and meta-analysis. *Brain Behav Immun.* 2007;21:901–912.
22. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J Health Soc Behav.* 1983;24:385–396.
23. Cohen S, Williamson G. Perceived stress in a probability sample of the United States. In: Spacapan S, Oskamp S, eds. *The Social Psychology of Health.* Newbury Park, CA: Sage; 1988:31–67.
24. Karasek R, Brisson C, Kawakami N, Houtman I, Bongers P, Amick B. The Job Content Questionnaire (JCQ): an instrument for internationally comparative assessments of psychosocial job characteristics. *J Occup Health Psychol.* 1998;3:322–355.
25. Bosma H, Marmot MG, Hemingway H, Nicholson AC, Brunner E, Stansfeld SA. Low job control and risk of coronary heart disease in Whitehall II (prospective cohort) study. *BMJ.* 1997;314:558–565.
26. Appels A. Loss of control, vital exhaustion, and coronary heart disease. In: Steptoe A, Appels A, eds. *Stress, Personal Control, and Health.* New York, NY: John Wiley & Sons; 1989:215–235.
27. Kopp MS, Falger PRJ, Appels A, Szedmak S. Depressive symptomatology and vital exhaustion are differentially related to behavioral risk factors for coronary artery disease. *Psychosom Med.* 1998;60:752–758.
28. Peter R, Siegrist J. Chronic psychosocial stress at work and cardiovascular disease: the role of effort-reward imbalance. *Int J Law Psychiatry.* 1999;22:441–449.
29. Cutrona CD, Russell DW. The provisions of social relationships and adaptation to stress. *Adv Pers Relat.* 1987;1:37–67.
30. Constable JF, Russell D. The effect of social support and the work environment upon burnout among nurses. *J Human Stress.* 1986;12:20–26.
31. Wilson P, D'Agostino R, Levy D, Belanger A, Silbershatz H, Kannel W. Prediction of coronary heart disease using risk factor categories. *Circulation.* 1998;97:1837–1847.
32. National Institutes of Health. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): Final Report. Bethesda, MD: National Institutes of Health, National Heart, Lung, and Blood Institute; 2002.
33. Farin HMF, Abbasi F, Reaven GM. Comparison of body mass index versus waist circumference with the metabolic changes that increase the risk of cardiovascular disease in insulin-resistant individuals. *Am J Cardiol.* 2006;98:1053–1056.
34. Cohen J. *Statistical Power Analysis for the Behavioral Sciences.* 2nd ed. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988.
35. Sookian S, Gemma C, Fernandez Gianotti T, et al. Effects of rotating shift work on biomarkers of metabolic syndrome and inflammation. *J Intern Med.* 2007;261:285–292.
36. Puttonen S, Kivimäki M, Elovainio M, et al. Shift work in young adults and carotid artery intima-media thickness: the cardiovascular risk in young Finns study. *Atherosclerosis.* 2009;205:608–613.
37. Joseph PN, Violanti JM, Donahue R, et al. Police work and subclinical atherosclerosis. *J Occup Environ Med.* 2009;51:700–707.
38. McNicholas WT. Obstructive sleep apnea and inflammation. *Prog Cardiovasc Dis.* 2009;51:392–399.
39. Mallon L, Broman J-E, Hetta J. Sleep complaints predict coronary artery disease mortality in males: a 12-year follow-up study of a middle-aged Swedish population. *J Intern Med.* 2002;251:207–216.
40. Vila B. Impact of long work hours on police officers and the communities they serve. *Am J Ind Med.* 2006;49:972–980.
41. Neylan TC, Metzler TJ, Best SR, et al. Critical incident exposure and sleep quality in police officers. *Psychosom Med.* 2002;64:345–352.
42. Tabanelli MC, Depolo M, Cooke RM, et al. Available instruments for measurement of psychosocial factors in the work environment. *Int Arch Occup Environ Health.* 2008;82:1–12.