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The Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) Pilot Study: Methods and Participant Characteristics

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PURPOSE: The Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) study is one of the first population-based studies to integrate psychological, physiological, and subclinical measures of stress, disease, and mental dysfunction. This pilot study was undertaken to establish a methodology and descriptive results for a larger police study.

METHODS: A stratified sample of 100 officers was randomly selected from the Buffalo, NY Police Department. Salivary cortisol served as a stress biomarker. Flow mediated dilation (FMD) and carotid intima-media thickness (IMT) were performed with ultrasound. Dual Energy X-Ray Absorptiometry (DEXA) and anthropometric measures assessed body composition. Self-report measures of depression and posttraumatic stress disorder (PTSD) were obtained.

RESULTS: Recruitment attained for the study was 100%. Seventy-five percent showed a cortisol increase upon awakening, 90% a negative diurnal slope, and 77% an increased cortisol response after a high protein lunch challenge. Dexamethasone suppression was evident. FMD showed an increase in mean brachial artery diameter of 3.2% in men and 3.9% in women, and mean IMT was lower (male = 0.67 mm; female = 0.62 mm) compared to populations of similar age. For males, the mean body-mass index (BMI) was 29.8 kg/m^2 and total body fat 23.4%. For females, the mean BMI was 26.7 kg/m^2 and total body fat 31.5%. For all officers, 16% met criteria for depression; 36% reported elevated PTSD symptoms.

CONCLUSIONS: Compared to populations of similar age, police officers had slightly lower FMD, lower carotid IMT, elevated BMI, and higher reported rates of depression and PTSD. Standardized physiological and psychological data collection and descriptive results confirmed that the methodology of the study is feasible in a working police population.

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INTRODUCTION

Stress may be described as a process whereby environmental stimuli place an undue strain on a human being resulting in psychological and physiological changes that increase the risk for disease (1). The most commonly studied physiological systems that respond to stress are the hypothalamic–pituitary–adrenal axis (HPA) and the autonomic nervous system. The severity and impact of the stress response may be viewed by gauging physiological disruption and dysregulation of these systems in terms of "allostatic load," which refers to the wear-and-tear that the body experiences due to repeated cycles of stress (2–9).

Police work is a psychologically stressful occupation and, as a consequence, officers may have an increased risk for diseases associated with stress (10–20). Examples of stressful police encounters are involvement in shootings, witnessing violence and familial abuse, and handling dead bodies. Such

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Selected Abbreviations and Acronyms

BCOPS = Buffalo Cardio-Metabolic Occupational Police Stress study
BMI = body mass index
BSI = brief symptom inventory
CES—D = Center for Epidemiological Studies Depression scale
CHD = coronary heart disease
IMT = intima-media thickness
CVD = cardiovascular disease
DEXA = dual energy X-ray absorptiometry
FMD = flow mediated dilation
HPA = hypothalamic—pituitary-adrenal system
IES = impact of event scale
PTSD = post traumatic stress disorder

events may also leave officers susceptible to PTSD, an acute form of stress related to traumatic event exposure (21–32). The potential long term effects of PTSD may additionally lead to behavioral dysfunction, such as substance abuse, relationship difficulties, aggressive behaviors, and suicide (33–36).

Psychological stress has been identified as a possible factor in the etiology of heart disease (37–39), and recent mortality studies indicate an elevated risk of atherosclerotic heart disease in police officers (10–12). Other studies have found that a substantial number of officers were at elevated risk for atherosclerotic heart disease; 22% were smokers, 76% had elevated cholesterol, 26% had elevated triglycerides, and 60% elevated body fat composition (14–16). Public safety officers had a higher probability of developing coronary heart disease (CHD) than did the Framingham study population (14). A 22-year follow-up study of 970 Helsinki police officers found an association between hyperinsulinemia and increased CHD risk in police officers independent of other risk factors (17).

It is suggested that stress may lead to disease as a result of abnormal cortisol secretion patterns due to frequent or persistent challenges of the HPA axis. In chronic stress, this pattern may change so that levels of cortisol are not elevated upon awakening, and/or fail to return to baseline within a period of several hours. Evidence now suggests that a pathological HPA axis (i.e., low variability, poor challenge responses, poor suppression by dexamethasone) has a major impact on how the other risk factors for cardiovascular disease (CVD), type II diabetes and stroke cluster (41–43).

The Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) pilot study was conducted to establish a working methodology, protocol, and design for the first ever population-based stress study that would identify preliminary descriptive patterns of response concerning biomarkers of stress, subclinical CVD, body composition indicators, and associated psychosocial factors in the high stress occupation of police work.

RESEARCH DESIGN

Selection of Police Sample

The Buffalo, New York Police Department, a midsized urban police force, was the selected sample site. A random sample stratified on gender ($n = 100$) was generated from all police officers in the department (42 females, 58 males) using a computer-generated random number table. No specific inclusion criteria were used for the study, other than that the participant would be a sworn police officer and willing to participate in the study. However, eligibility criteria for two ultrasound procedures involving the brachial and carotid arteries were identified. Participant officers must have had no prior history of mastectomy, removal of lymph nodes, Raynaud's syndrome, diabetes with insulin pump, kidney dialysis, use of blood thinners, high doses of aspirin, and any other heart condition or circulatory disorder for the brachial ultrasound, and for carotid ultrasound, they had to be free of a history of heart attack, stroke, bypass surgery, carotid artery endarterectomy, transient ischemic attack, or physician-diagnosed CHD. The Center for Preventive Medicine, State University of New York at Buffalo, School of Public Health and Health Professions, Buffalo, N.Y., served as the data collection site. This center has been used in numerous epidemiological studies in the past, and observers are well trained in health data collection processes. Quality control of data at this facility includes daily use of a detailed manual-of-operations for all procedures, systemic training of the staff, monitoring of the lab internal control with internal standards, and periodic testing and maintenance of instruments.

MEASURES

Salivary Cortisol as a Biomarker for Stress

The HPA axis of a healthy individual would be described as having a high degree of variation with a morning peak and an evening nadir and appropriate response to challenge (e.g., physiological or psychological stressors; dexamethasone suppression). Recent evidence has indicated that changes in cortisol secretion in response to a standardized high protein meal might be used to assess the state of the HPA axis. A high protein meal challenge provokes an approximate 2–4 fold increase in healthy adult males and females when given at midday (41–43). Utilizing this methodology, salivary cortisol patterns were measured with a standardized protein meal challenge (65 grams of protein in the form of a shake) and a dexamethasone (0.5 mg) suppression test orally self-administered on the second evening after the clinic visit. Subjects were required to ingest the protein shake over a 20-minute period to better simulate the actual eating of a meal. A baseline saliva tube sample (Sarstedt Aktiengesellschaft & Company, Numbrecht,

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Germany) designed to yield approximately 0.7 ml of saliva was taken at the clinic followed by the protein cortisol challenge and four additional saliva samples at fifteen minute intervals. Officers were then provided eight additional saliva sample tubules and the dexamethasone tablet for off-clinic-site saliva testing of awakening, diurnal, and dexamethasone suppression testing of cortisol over a 2-day period. Table 1 illustrates the saliva collection and cortisol challenge protocol.

Subclinical Cardiovascular Disease

Brachial reactivity. High resolution B-mode ultrasound was used to evaluate several subclinical CVD markers. Endothelial function was measured by changes in the diameter of the brachial artery due to increased blood flow induced by inflation and then deflation of a pressure cuff on the forearm. The cuff was inflated for a period of 4 minutes and released. Indicators of brachial reactivity represent useful composite measures of overall vascular status (44). The scans were read at Wake Forest Ultrasound Reading Center, Wake Forest University, Winston-Salem, NC Mandated quality control policy of the Wake Forest Center is to select four representative scans and have each reader select and store 40 frames from each of these scans. These images provide the basis for comparing mean diameter blinded measurements from the same image by different readers (interreader reliability). The above process is then repeated using four different representative scans selected by each reader. Comparisons must meet a criteria level of $< 1.5\%$ coefficient of variation.

The overall concept was to observe changes in the artery diameter following the use of an occlusive pressure on the artery which causes reactive hyperemia in the vessel and arterial dilation upon deflation of the cuff (45–48). Flow–mediated dilation (FMD) assesses endothelium-dependent reactivity of the artery. Measures of vasodilatory responses obtained using brachial artery reactivity studies were the maximal diameter of the artery during the period of deflation, percentage change in arterial diameter compared to baseline diameter, time to maximal (peak) diameter, and the area under the dilation curve. A continuous scan was employed, as this method maintains the probe at the same location and angle of interrogation throughout the scan without being removed from the skin surface.

TABLE 1. Saliva cortisol sample timing, BCOPS study, 2001–2003

Sample number	Day	Approximate time or time related event	Challenge status or timed sample
1	1	11:10 am	Before protein drink
2	1	11:20–12:30	15 minutes after protein shake
3	1	11:20–12:30	30 minutes after protein shake
4	1	11:20–12:30	45 minutes after protein shake
5	1	11:20–12:30	60 minutes after protein shake
6	2	Waking	First waking sample
7	2	Waking	15 minutes after waking
8	2	Waking	30 minutes after waking
9	2	Waking	45 minutes after waking
10	2	Lunchtime	Immediately before eating midday meal
11	2	Dinnertime	Immediately before eating evening meal
12	2	Bedtime	Before bedtime and before taking dexamethasone tablet
13	3	Waking	Post dexamethasone waking sample

Carotid intima-media thickness. Carotid artery intima-media thickness (IMT) has been demonstrated to be a useful tool in predicting CVD outcomes, clinical coronary outcomes, and in monitoring the progression of arterial wall thickness over time (49–55). The locations along the artery that are primarily studied are the common carotid at the bifurcation of the artery into the internal and external carotid, and the internal carotid artery (found to be a common site for the occurrence of lesions and atherosclerotic plaque). Measurements are generally taken in 1 cm segments from the tip of the flow-divider at the bifurcation of the carotid artery whereby measures of the 1 cm of common carotid prior to the bifurcation, the 1 cm of the bifurcation, and 1 cm of the internal carotid are obtained. Median IMT values are in the range of 0.4–1.0 mm (55, 56). Carotid IMT was measured with B-mode ultrasound and included vessel interfaces, wall thicknesses, vessel width, and regularity of surfaces. Images of brachial reactivity and carotid IMT were recorded on high resolution video tapes for interpretation. Readers were blinded to specific hypotheses and characteristics of the study population.

Orthostatic hypotension. Blood pressure and pulse rate were measured at rest, while supine, and then after the participant was moved from a supine to a standing position. These measures were repeated at 1-, 3-, and 5-minute intervals. A decrease of 20 mm Hg or more in systolic or 10 mm Hg or more in diastolic blood pressure upon standing is a commonly used diagnostic criterion for this test (57).

Body Composition

Body composition testing was performed with a Dual Energy X-Ray Absorptiometry (DEXA) device. Assessment of total body skeletal density and body composition (distribution of fat and lean tissue) was performed overall and at several locations. In addition, weight and height were measured and body mass index (BMI) was calculated as kg per meter squared.

Psychosocial Measures

The Center for Epidemiological Studies Depression scale (CES–D) was used as a measure of psychological distress and depression in officers (58). The CES–D is a 20-item scale, with responses for each question ranging from "rarely" to "most of the time." This instrument is primarily for measuring depressive symtpomatology and has been shown to reliably measure stress. The Impact of Event scale (IES) measures stress related to traumatic events (59) and was used to detect psychological symptoms of PTSD. The Police Incident Survey contained a list of traumatic situations usually found in police work and included items such as involvement in shootings, observing dead bodies, working with abused children, and witnessing crime victims and human misery. Participants were asked the number of times they had experienced each of these events at work over the past year.

Informed Consent and Data Security

All participant officers in the study were informed of the purpose and benefits of the project, the research methods to be used, the potential risks or hazards of participation, and the right to ask for further information at any time during the research procedure. They were further informed that their choice to participate is a voluntary one, and that they are free to withdraw from the research project at any time. All phases, testing, and reports of the study were approved by The State University of New York at Buffalo's Internal Review Board and the National Institute for Occupational Safety and Health Human Subjects Review Board.

STATISTICAL ANALYSIS

Simple descriptive measures were calculated including means and standard deviations for normally distributed continuous variables, and medians and percentiles for those that were not normally distributed. Prevalence of certain discrete or categorical characteristics was also determined. Cortisol responses to several challenges were calculated as simple differences except for the diurnal cortisol pattern where linear regression was used to determine slopes based on each participant's second day cortisol samples. All results were stratified by gender. SAS version 8.2 was used for analysis (SAS Institute, Inc., Cary, NC).

RESULTS

Of the 100 randomly selected officers who were invited to participate, 100% agreed to and completed the pilot examination. Demographic characteristics of the participants in the sample are provided in Table 2 by gender. Nearly half of the sample were between 40–49 years of age (mean = 44 years), 19% were African- American, and 42% were women due to their oversampling, a majority were married and had the rank of patrol officer. Nearly 50% of police officers in the sample had served at least 15 years in the department (Table 2).

Approximately 64% of participants provided all 13 saliva samples, including on and off site testing. Salivary cortisol levels in men, women and all participants combined (Table 3) showed responses to the provocation challenges and expected awakening and diurnal responses. Some gender differences were also evident, with men having slightly higher median cortisol levels than women. A total of 66 of 96 participants (77%) showed an increased cortisol response to a high protein challenge, with median levels increasing to a greater extent in women than in men, followed by a slight reduction over the 60-minute period. Upon awakening 75% showed a cortisol increase; the proportion showing an increase was more commonly found in women (89%) than in men (65%). Similarly, the increase in median cortisol levels was greater in women (8.7 to 15.1 nmol/L) than in men (12.8 to 13.4 nmol/L). Nearly 90%

TABLE 2. Demographic characteristics by gender, BCOPS study, 2001–2003

	Men (N=58)		Women (N=42)		Total (N=100)	
Characteristic	N	%	N	%	N	%
Age group						
< 40 years	20	34.5	9	21.4	29	29.0
40–49 years	19	32.8	26	61.9	45	45.0
50 + years	19	32.8	7	16.7	26	26.0
Ethnicity						
Hispanic-American	5	8.6	0	0.0	5	5.0
African-American	9	15.5	10	23.8	19	19.0
European-American	44	75.9	32	76.2	76	76.0
Education*						
Less than 12 years	1	1.7	0	0.0	1	1.0
High School/GED	8	13.8	7	16.7	15	15.0
College < 4 years	17	29.3	12	28.6	29	29.0
College 4 + years	32	55.2	23	54.8	55	55.0
Marital status*						
Single	10	17.2	10	23.8	20	20.0
Married	43	74.1	22	52.4	65	65.0
Divorced	5	8.6	10	23.8	15	15.0
Years served*						
1–5 years	11	19.0	8	19.0	19	19.0
6–10 years	6	10.3	6	14.3	12	12.0
11–15 years	12	20.7	11	26.2	23	23.0
15+ years	29	50.0	17	40.5	46	46.0
Rank*						
Police officer	27	46.6	24	57.1	51	51.0
Sergeant/ lieutenant	9	15.5	6	14.3	15	15.0
Captain	6	10.3	2	4.8	8	8.0
Detective	11	19.0	4	9.5	15	15.0
Other	5	8.6	6	14.3	11	11.0

*Assessed during previous visit (years 1999–2000).

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TABLE 3. Salivary cortisol levels and characteristics by time of sample and gender, Buffalo Police Pilot Health Study, 2001–2003

	Men ($N=55$)			Women ($N=41$)			Total ($N=96$)		
Cortisol Samples	N	Median	Percentile (25^{th}, 75^{th})	N	Median	Percentile (25^{th}, 75^{th})	N	Median	Percentile (25^{th}, 75^{th})
Day one									
Before protein shake	52	7.5	(4.2, 13.3)	35	4.2	(3.2, 5.7)	87	5.4	(3.4, 9.2)
15 min after shake	52	7.2	(5.0, 15.0)	37	5.1	(3.5, 6.3)	89	6.1	(4.1, 10.1)
30 min after shake	52	7.6	(4.6, 12.9)	36	5.7	(4.2, 7.6)	88	6.2	(4.3, 10.1)
45 min after shake	50	6.6	(4.2, 10.9)	35	4.7	(3.4, 7.3)	85	5.8	(3.9, 9.3)
60 min after shake	51	5.4	(3.9, 10.4)	34	5.1	(3.8, 8.0)	85	5.2	(3.9, 8.6)
Day two									
At wakening	51	12.8	(8.3, 19.0)	35	8.7	(6.1, 12.8)	86	11.1	(7.3, 18.0)
15 min after waking	49	13.4	(8.1, 24.3)	33	11.6	(7.4, 18.3)	82	13.0	(7.9, 21.4)
30 min after waking	49	10.6	(6.4, 21.2)	39	15.1	(9.6, 22.0)	88	13.4	(7.9, 21.6)
45 min after waking	51	10.9	(6.3, 18.5)	37	14.8	(9.0, 22.9)	88	13.0	(6.8, 20.3)
Before lunch	52	6.8	(3.6, 10.9)	37	5.5	(2.7, 8.4)	89	6.2	(3.5, 10.1)
Before dinner	49	3.6	(1.9, 5.2)	39	2.7	(1.6, 5.1)	88	3.4	(1.8, 5.2)
Before bedtime & Dexamethasone	50	1.4	(0.6, 3.2)	39	1.8	(1.2, 6.1)	89	1.7	(0.9, 3.7)
Day three									
At wakening after Dexamethasone	47	1.4	(0.5, 4.3)	35	1.7	(1.0, 2.8)	82	1.5	(0.7, 3.8)

Cortisol values expressed as nmol/L.

demonstrated a negative diurnal slope with median levels decreasing from 11.1 nmol/L on awakening to 1.7 nmol/L before bedtime. Notable dexamethasone suppression was evident in the 82 participants whose median cortisol level on awakening was 1.5 nmol/L, a level similar to the median before bedtime (1.7 nmol/L) for 89 participants.

Mean levels and percentages of anthropometric and subclinical CVD measures are provided for male and female police officers in Table 4 . Mean BMI was 29.8 kg/m^2 for men and 26.7 kg/m^2 for women, while total body fat was 23.4% for men and 31.5% for women. Although whole body-bone density was similar for men and women, mean total lean body mass was greater in men (69.9 kg) than in women (49.1 kg). Mean systolic and diastolic blood pressure were somewhat higher in men than in women. Mean heart rate and diastolic blood pressure increased by approximately 10 beats per minute and 5 mm Hg respectively with standing. Orthostatic hypotension was rare with only one male officer meeting the criteria. Of the 100 participants, 81 of the brachial scans were of sufficient quality to read, 13 were insufficient, and six participants were ineligible for scans due to proscribed medical reasons. Mean brachial artery diameter increased from 4.97 mm to 5.13 mm (3.2%) in men and 3.72 mm to 3.87 mm (3.9%) for women. Mean carotid IMT was 0.67 mm in men and 0.62 mm in women officers.

Table 5 presents a summary of psychosocial measures by gender. Mean scores of distress and depression (CES–D) and impact of traumatic events (IES) were higher for women officers. Mean levels of depression were higher for women officers than for men (9.79 vs. 7.93). Mean scores on IES subscales of intrusiveness of thoughts and avoidance were slightly higher for women than men officers. Men and women officers had similar scores in terms of trauma symptom severity. On average, male officers reported experiencing more traumatic events at work (4.67 events vs. 3.44 events over the last year in men and women, respectively).

DISCUSSION

The BCOPS study is one of the first population-based studies to integrate psychological, physiological and subclinical pathology measures of stress, disease, and mental dysfunction. Descriptive results from the present pilot study reinforced the use of its design and methodology for a planned larger BCOPS project.

We were successful in obtaining a working sample of police officers, as 100% of the generated stratified random sample who were invited participated in the study. This demonstrates that we have a motivated and willing population with which to conduct a large-scale population based study involving this police department. Our data were successfully obtained from reliable, standardized, and quality controlled measures widely used in clinical and epidemiologic research.

Brachial artery FMD in this police sample showed an increase from 4.97 mm to 5.13 mm (3.2%) in men and 3.72 mm to 3.87 mm (3.9%) in women. Other studies have shown FMD increases in healthy individuals from 2.4% to 7.01% and have attempted to establish a cutoff value of 4.5% or below to identify the presence of endothelial

TABLE 4. Anthropometric and cardiovascular characteristics by gender, Buffalo Police Pilot Health Study, 2001–2003

	Men ($N=58$)		Women ($N=42$)		Total ($N=100$)	
Characteristic	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD
Age (years)	58	44.0 ± 8.7	42	44.0 ± 5.7	100	44.0 ± 7.6
Height (cm)	58	178.8 ± 9.5	42	166.3 ± 5.9	100	173.5 ± 10.2
Weight (kg)	58	94.5 ± 14.2	42	73.8 ± 14.6	100	85.8 ± 17.6
BMI (kg/m^2)	58	29.8 ± 5.8	42	26.7 ± 5.0	100	28.5 ± 5.7
Total body fat (%)[a]	56	23.4 ± 5.4	42	31.5 ± 6.4	98	26.9 ± 7.1
Whole body bone mineral density, g/cm^2[a]	56	1.3 ± 0.1	42	1.2 ± 0.1	98	1.2 ± 0.1
Total lean body mass, kg[a]	56	69.9 ±6.7	42	49.1 ±6.5	98	61.0 ±12.2
Resting heart rate (beat/min)	58	59.2 ± 8.3	42	63.0 ± 7.9	100	60.8 ± 8.2
Systolic BP (mm Hg)	58	122.6 ± 14.9	42	112.6 ± 12.6	100	118.4 ± 14.8
Diastolic BP (mm Hg)	58	81.2 ± 10.4	42	73.0 ± 8.0	100	77.8 ± 10.3
Postural change [b]						
Heart rate (beat/min)	58	9.4 ± 5.4	42	10.3 ± 6.6	100	9.8 ± 6.0
Systolic BP (mm Hg)	58	1.2 ± 7.1	42	0.6 ± 6.4	100	1.0 ± 6.8
Diastolic BP (mm Hg)	58	4.8 ± 5.5	42	4.2 ± 5.0	100	4.5 ± 5.3
Orthostatic hypotension (%)[c]	58	1.7	42	0.0	100	1.0
Brachial artery diameter						
Baseline (mm)	46	4.92 ± 0.54	35	3.71 ± 0.49	81	4.40 ± 0.79
Pre-cuff release (mm)	46	4.97 ± 0.54	35	3.72 ± 0.51	81	4.40 ± 0.82
Maximum post-cuff release (mm)[d]	46	5.13 ± 0.53	35	3.87 ± 0.51	81	4.58 ± 0.81
Carotid IMT (mm)	51	0.67 ± 0.12	42	0.62 ± 0.09	93	0.64 ± 0.10

BMI, body mass index; BP, blood pressure; IMT, intimal-medial thickness.
[a]Measured by Dual Energy X-Ray Absorptiometry (DEXA).
[b]Postural change in heart rate, systolic BP, and diastolic BP were determined by subtracting the supine from the standing measures at 3 minutes after standing.
[c]Orthostatic hypotension defined as reduction in systolic BP of ⩾ 20 mm Hg or in diastolic BP of ⩾ 10 mm Hg within 3 minutes of standing.
[d]Estimated from a nonparametric model of each subjects brachial curve.

dysfunction (46), however, such dilation levels are presently a matter of debate (71–74).

Although BMI measures among all officers (28.5 kg/m^2) were on average above the 25 kg/m^2 conventional definition for "overweight," this convention does not take into account significant biological differences in adipose distribution between males and females. In addition, it is possible that the elevated BMI in police officers could represent increased muscle mass due to strength training, which may occur to a greater extent in this occupation than in the general population. Based on this convention, other police studies which demonstrate 83% of officers being "overweight" compared to 71% of the general population may be misleading as to potential risk for metabolic and

TABLE 5. Summary of psychosocial measures by gender. BCOPS study, 2001–2003

	Men ($N=58$)		Women ($N=42$)		Total ($N=100$)	
Variable	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD
CES-D score (0-60)[a]	58	7.93 ± 6.56	41	9.79 ± 8.93	99	8.70 ± 7.64
Impact of events score (0-75)[b]	58	19.67 ± 15.09	41	23.29 ± 13.94	99	21.17 ± 14.66
Intrusive subscale score (0-35)	58	9.02 ± 8.33	41	11.51 ± 7.84	99	10.05 ± 8.18
Avoidance subscale score (0-40)	58	10.66 ± 7.94	41	11.78 ± 7.84	99	11.12 ± 7.88
Level of symptoms from impact of events						
Subclinical (0-8)	18 (31.0%)		8 (19.5%)		26 (26.3%)	
Mild (9-25)	20 (34.5%)		18 (43.9%)		38 (38.4%)	
Moderate (26-43)	15 (25.9%)		10 (24.4%)		25 (25.3%)	
Severe (44-75)	5 (8.6%)		5 (12.2%)		10 (10.1%)	
Police incident score (0-9)[c]	58	4.67 ± 2.38	41	3.44 ± 2.46	99	4.16 ± 2.48

[a]Consists of the sum of 20 items from the CES-D scale, with higher scores indicating major depressive disorder.
[b]Consists of the sum of 15 items from the Impact of Event Scale, with higher scores indicating the intensity of post-traumatic stress symtomatology.
[c]Consists of the sum of 9 items from the Police Work Traumatic Incident Survey.

cardiovascular subclinical disease (14,15). The BCOPS study is well positioned to address the relative merits of using BMI compared to direct measurements of adiposity, bone mass, and lean body mass—for men and for women. Public safety officers had a higher probability of developing CHD than did the Framingham study population (14–17). Interestingly, atherosclerotic heart disease rates were higher in officers with fewer years of service (11). This is infrequently the case in healthy worker populations like the police (40). This has been found only in mortality cohorts, and limited information is available on lifestyles and work exposures. However, previous literature demonstrates that police officers are immediately and continuously exposed to considerable stress and traumatic events in their work, as well as shift work, poor diet, and poor exercise habits (14,15). These could be precipitating factors for cardiovascular disease (39).

Mean carotid IMT for the total police sample was 0.64 mm. Cross-sectional analysis from the Atherosclerosis Risk in Communities (ARIC) study suggests that age related increase in wall thickness averages approximately 0.010 per year (48). Findings from the ARIC study data show a mean thickness of 0.72 mm in participants without stroke (baseline age 45–64 years), a population slightly older on average than the present police sample (mean age = 44 years). ARIC data from 1987–1995 also suggested that a mean IMT of 0.8–1.0 mm posed at least a twofold greater risk of subsequent ischemic stroke (75).

A recent review on IMT measurements in normal subjects found that the risk of first myocardial infarction increases with an IMT of 0.82 mm or more and the risk of stroke with a mean IMT of 0.75 mm or more. A progression rate of IMT of 0.034 mm per year or greater increased the risk of future events significantly (55). The median wall thickness for all ages in this sample ranged from 0.5–1.0 mm. A study on younger healthy subjects (age 37 $\pm$ 4 years) reported an average IMT thickness of 0.75 mm (60–62).

A recent study of PTSD in police officers found that 25% of officers who scored more than 20 on the IES left the force within one year (26). 35% of officers in our sample scored over 25 on the IES, with approximately 73% reporting at least some level of PTSD symptomatology. Biological changes in physiological reactivity may occur as a result of traumatic exposure, and trauma may impact allostatic load. In particular, urinary and plasma-cortisol levels are considerably lower in PTSD patients than in non–PTSD trauma survivors and normal controls. The reduction in cortisol levels results from an enhanced negative feedback by cortisol, which is secondary to an increased sensitivity of receptors in target tissues (63–67).

The CES–D scale included components of depression such as depressed mood, feelings of guilt, helplessness, loss of appetite, and sleep disturbance. Approximately 16% of the officers scored at or above a critical level of depression as defined by this measure (mean score of 16.0 or greater) (58).

Salivary cortisol levels in men, women, and all participants combined in our police sample (Table 3) showed responses to provocation challenges with awakening and diurnal responses. In a review of awakening cortisol response studies, increases upon awakening ranged from 50%–156%, with a mean increase of 91% (68), which represent a somewhat greater magnitude of increase than in our police sample (21%). The magnitude of dexamethasone suppression observed in this study was similar to that of other studies (41, 42, 67).

The primary limitations of this pilot study are the relatively small sample size of 100 officers and the uniqueness of the occupational population under study. Although police officers may not be representative of the population at large, findings from this study may be generalizable to other police officers and perhaps to similar occupational groups. In addition, choice of the police occupation offers an excellent opportunity to study a population continuously exposed to chronic and acute stress. This population represents a large occupational group of approximately 708,000 sworn police officers in the United States (69). The assessment of subclinical CVD and biological markers of stress at a single point in time provides descriptive health-related characteristics of a midsized city police department. Larger prospective studies are presently being planned to assess such markers over time and differential exposures to other psychosocial conditions such as prior life stressors, depression, and coping skills.

The strengths of this study are the availability of several subclinical CVD markers such as brachial and carotid IMT data along with other risk factors and psychosocial measures for this sample, the use of a standardized protocol, and high response rates and cooperation in the study. Measures such as cortisol biomarkers and standardized cortisol challenge tests for stress may provide a more quantitative physiologic measure than questionnaires and therefore circumvent the potential problem of under reporting of stress or misclassification by participants. Stress biomarkers can also facilitate a better understanding of change over time (70).

Overall, police officers showed a relatively low brachial artery FMD and a lower carotid IMT compared to other populations of similar mean ages. A moderate response to cortisol provocation challenges, good awakening and diurnal responses, and notable dexamethasone suppression were observed. BMI levels were on average higher than recommended body weight norms, although BMI may not reflect differences in muscle mass or biological differences in adipose distribution between males and females. Sixteen percent of the police sample met the case criteria for depression on the CES–D measure, and approximately 36% reported moderate to high PTSD symptoms on the IES. This

pilot project also demonstrated that collection of standardized physiological and psychological data is feasible for assessing associations involving cortisol, subclinical cardiovascular disease, body composition, and psychosocial measures in a large police population.

REFERENCES

1. Cohen S, Kessler RC, Gordon LU. Measuring Stress. New York: Oxford University Press; 1997; 3–28.
2. Haddy RI, Clover RD. The biological processes in psychological stress. Fam, Sys & Health. 2001;19:291–302.
3. Chrousos GP, Gold PW. The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. JAMA. 1992;267: 1244–1252.
4. Black PH. Central Nervous System Interactions: Psychoneuroimmunology of stress and its immune consequences. Antimicro Agents Chemo. 1994;8:1–6.
5. McEwen BS, Seeman TE. Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. Ann of New York Acad Sci. 1999;896:30–47.
6. McEwen BS. Protective and damaging effects of stress mediators. NEJM. 1998;338:171–179.
7. Krantz DS, McCeney MK. Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. Ann Rev Psych. 2002;53:341–369.
8. Chrousos G. Editoral: A healthy body in a healthy mind—and vice versa: The damaging power of "uncontrollable" stress. J Clin Endo Metab. 1998;83:1842–1845.
9. Rosmond R, Bjorntorp P. The hypothalamic—pituitary-adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes and stroke. J Internal Med. 2000;2188–2197.
10. Violanti JM, Vena JE, Marshall JR. Disease risk and mortality among police officers: New evidence and contributing factors. J Pol Sci Admin. 1986;14:17–23.
11. Violanti JM, Vena JE, Petralia S. Mortality of a police cohort: 1950–1990. Am J Ind Med. 1998;33:366–373.
12. Williams MA, Petratis MM, Baechle TR, Ryschon KL, Campain JJ, Sketch MH. Frequency of physical activity, exercise capacity, and atherosclerotic heart disease risk factors in male police officers. J Occup Med. 1987;29:596–600.
13. Pollock ML, Gettman LR. Coronary Risk Factors and Level of Physical Fitness in Police Officers. Dallas, Texas: Institute for Aerobics Research; 1986.
14. Franke WD, Cox DF, Schultz DP, Anderson DF. Coronary heart disease risk factors in employees of Iowa's department of public safety compared to a cohort of the general population. Am J Ind Med. 1997;31:733–777.
15. Ramey SL, Franke WD, Shelley MC II. Relationship among risk factors for nephrolithiasis, cardiovascular disease, and ethnicity: Focus on a law enforcement cohort. AAOHN J. 2004;52:116–121.
16. Eli DL, Mostardi RA. The effect of recent life events, stress, life assets, and temperament pattern on cardiovascular risk factors for Akron city police officers. J Human Stress. 1986;12:77–91.
17. Pyorala M, Miettinen H, Laakso M, Pyorala K. Hyperinsulemia predicts coronary heart disease risk in healthy middle-aged men: The 22-year follow-up results of the Helsinki policemen study. Circulation. 1998;98:398–404.
18. Violanti JM. Dying for the job: Stress and police mortality. NY: Prentice-Hall; 2004.
19. Why YP, Bishop GD, Tong EMW, Diong SW, Enkelmann HC, Khader M, Ang J. Cardiovascular reactivity of Singerporean male police officers as a function of task, ethnicity and hostility. Int J Psychopath. 2003;49:99–110.
20. Piercecchi MD, Leonetti G, Pelissier AL, Conrath J, Cianfarani F, Valli M. Evaluation of biological stress markers in police officers. Med Law. 1999;18:125–144.
21. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders; 4th Ed. Washington, DC: American Psychiatric Association; 1994 24–42.
22. Violanti JM, Paton D. Police Trauma: Psychological Aftermath of Civilian Combat. Springfield, IL: Thomas; 1999 1–25.
23. Paton D, Smith LM. Psychological Trauma in Critical Occupations: Methodological and assessment strategies. In: Paton D, Violanti JM, eds. Traumatic Stress in Critical Occupations: Recognition, Consequences, and Treatment. Springfield, IL: Thomas; 1996;125–139.
24. Violanti JM. Trauma Stress in Police Work. In: Paton D, Violanti JM, eds. Traumatic Stress in Critical Occupations: Recognition, Consequences and Treatment. Springfield, IL: Thomas; 1996:137–143.
25. Violanti JM. Residuals of police occupational trauma. Aust J. Dis Trauma St. 1997;3:1–7.
26. Paton D, Violanti JM. Chronic Exposure to Risk and Trauma: Addiction and Separation Issues in Police Officers. Police Psychology Symposium publication. Wellington, NZ: New Zealand Psychological Society; 1997.
27. Paton D, Violanti JM. Long Term Exposure to Traumatic Demands in Police Officers: Behavioral Addiction and its Management. In: Haberman G, ed. Looking Back, Moving Forward: Fifty years of New Zealand Psychology. Wellington, NZ: New Zealand Psychological Society; 1997.
28. Violanti JM, Gehrke A. Police trauma encounters: Precursors of compassion fatigue. Int J Emerg Mental Health. 2004;6:75–80.
29. Alexander C. Police Psychological Burnout and Trauma. In: Violanti JM, Paton D, eds. Police Trauma: Psychological Aftermath of Civilian Combat. Springfield, IL: Thomas; 1999;54–63.
30. Gershon RM, Lin S, Li X. Work stress in aging police officers. J Occup Environ Med. 2002;44:160–167.
31. Hodgins GA, Creamer M, Bell R. Risk factors for posttrauma reactions in police officers: A longitudinal study. J Nervous Ment Dis. 2001;189: 541–547.
32. Wilczak C. Three perspectives on trauma from New York City police officers. Division 17 Newsletter, Division of Counseling Psychology of the American Psychological Association. 2002;3:13–15.
33. van der Kolk BA, McFarlane AC. The Black Hole of Trauma. In: van der Kolk BA, McFarlane AC, Weisaeth L, eds. Traumatic Stress: The Effects of Overwhelming Experience on Mind, Body and Society. New York: Guilford; 1996:5–23.
34. van der Kolk BA. The body keeps score: Approaches to The Psychobiology of Posttraumatic Stress Disorder. In: van der Kolk BA, McFarlane AC, Weisaeth L, eds. Traumatic Stress: The Effects of Overwhelming Experience on Mind, Body and Society. New York: Guilford; 1996: 214–241.
35. Violanti JM. Predictors of police suicide ideation. Suic Life Threat Beh. 2004;4:277–283.
36. van der Kolk BA. Stress Versus Traumatic Stress: From Acute Homeostatic Reactions to Chronic Psychopathology. In: van der Kolk BA, McFarlane AC, Weisaeth L, eds. Traumatic Stress: The Effects of Overwhelming Experience on Mind, Body and Society. New York: Guilford; 1996;77–101.
37. Henry JP. Mechanisms by which stress can lead to coronary heart disease. Postgrad Med J. 1986;62:687–693.
38. Eysenck HJ, Grossarth-Maticek R, Everitt B. Personality, stress smoking, and genetic predisposition as synergistic risk factors for cancer and coronary heart disease. Integr Physio Behav Sci. 1999;26:309–322.

39. Ross R. The pathogenesis of atherosclerosis: A perspective for the 1990's. Nature. 1993;362:801–809.

40. McMichael AJ. Standardized mortality ratios and the healthy worker effect: Scratching beneath the surface. J Occ Med. 1976;18:165–168.

41. Rosmond R, Bjorntorp P. Endocrine and metabolic aberrations in men with abdominal obesity in relation to anxio-depressive infirmity. Metabolism. 1998;47:1187–1193.

42. Rosmond R, Dallman MF, Bjorntorp P. Stress-related cortisol secretion in men: Relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. J Clin Endocrin Metab. 1998;83:1853–1859.

43. Gibson EL, Checkley S, Papadopoulos A, PoonDaley S, Wardle J. Increased salivary cortisol reliably induced by a protein-rich midday meal. Psychosom Med. 1999;61:214–224.

44. Vogel RA. Measurement of endothelial function by brachial artery flow-mediated vasodilation. Am J Cardiol. 2001;88(Suppl I):31–34.

45. Celermajer DS, Sorensen KE, Georgakopoulos D, Bull C, Thomas O, Robinson J, Deanfield JE. Vascular endothelial responses, prostanoids, and flow: Cigarette smoking is associated with dose-related and potentially reversible impairment on endothelium-dependent dilation in healthy adults. Circulation. 1993;88:2149–2155.

46. Vogel RA, Corretti MC, Plotnick GD. A comparison of brachial artery flow–mediated vasodilation using upper and lower arm arterial occlusion in subjects with and without coronary risk factors. Clin Cardiol. 2000; 23:571–575.

47. Hashimoto M, Eto M, Akishita M, Kozaki K, Ako J, Iijima K, et al. Correlation between flow-mediated vasodilation of the brachial artery and intima-media thickness of the carotid artery in men. Arterioscler Thromb Vasc Biol. 1999;9:2795–2800.

48. Chambless LE, Zhong MM, Arnett D, Folsom AR, Riley WA, Heiss G. Variability in B–Mode ultrasound measurements in the atherosclerosis risk in communities (ARIC) study. Ultrasound in Med & Biol. 1996; 22:545–555.

49. Jensen-Urstad K, Rosfors S. A methodological study of arterial wall function using ultrasound techniques. Clin Physio. 1997;17:557–567.

50. Salonen R, Salonen JT. Determinants of carotid intima-media thickness: A population-based ultrasonography study in eastern Finnish men. J Intern Med. 1991;229:225–231.

51. Grobbee DE, Bots ML. Carotid artery intima-media thickness as an indicator of generalized atherosclerosis. J Intern Med. 1994;236:567–573.

52. Chambless LE, Heiss G, Folsom AR, Rosamond W, Szklo M, Sharrett AR, Clegg LX. Association of coronary heart disease incidence with carotid arterial wall thickness and major risk factors: The atherosclerosis risk in communities (ARIC) study, 1987–1993. Am J Epidemiol. 1997;146:483–494.

53. Hodis HN, Mack WJ, LaBree L, Selzer RH, Liu CR, Liu CH, et al. The role of carotid arterial intima-media thickness in predicting clinical coronary events. Ann Intern Med. 1998;128:262–269.

54. O'Leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK Jr, for the Cardiovascular Health Study Collaborative Research Group. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. N Eng J Med. 1999;340:14–22.

55. Kanters SD, Algra A, van Leeuwen MS, Banga J. Reproducibility of in-vivo carotid artery intima–media thickness measurements: A review. Stroke. 1997;28:665–671.

56. Chambless LE, Folsom AR, Davis V, Sharrett R, Heiss G, Sorlie P, Szklo M, Howard G, Evans G. Risk factors for progression of common carotid atherosclerosis: The Atherosclerosis Risk in Communities Study, 1987–1998. Am J Epidemiol. 2002;155:38–47.

57. The Consensus Committee of the American Autonomic Society and the American Academy of Neurology. Consensus statement on the definition of orthostatic hypotension, pure autonomic failure, and multi-system atrophy. Neurology. 1996;46:1470.

58. Ratloff LS. The CES–D scale: A Self-report depression scale for research in the general population. Appl Psych Meas. 1977;1:385–401.

59. Horowitz M, Wilner M, Alvarez W. Impact of Event Scale: A measure of subjective stress. Psychosomatic Med. 1979;41:209–218.

60. Aminbakhsh A, Mancini GB. Carotid intima-media thickness measurements: What defines abnormality? A Systematic Review. Clin Inves Med. 1999;22:149–157.

61. Toikka JO, Laine H, Ahotupa M, Haapanen A, Jorma SA, Viikari JJ, Olli H, Raitakari T. Increased arterial intima-media thickness and in vivo LDL oxidation in young men with borderline hypertension. Hypertension. 2000;36:929–933.

62. Howard G, Sharrett AR, Heiss G, Evans GW, Chambless LE, Riley WA, Burke GL. Carotid artery intimal-medial thickness distribution in general populations as evaluated by b-mode ultrasound. ARIC investigators. Stroke. 1993;24:1297–1304.

63. Yehuda R. Biology of posttraumatic stress disorder. J Clin Psych. 2001;62(Suppl):41–46.

64. Yehuda R. Current status of cortisol findings in posttraumatic stress disorder. Psych Clin N Amer. 2002;25:341–368.

65. Yehuda R. Clinical relevance of biological findings in PTSD. Psych Quart. 2002;73:123–133.

66. Kellner M, Yehuda R, Arlt J, Wiedemann K. Longitudinal course of salivary cortisol in posttraumatic stress disorder. Acta Psych. Sc. 2002; 105:153–155.

67. Yehuda R, Golier JA, Halligan SL, Meaney M, Bierer LM. The ACTH response to dexamethasone in PTSD. Am J Psych. 2004;161: 1397–1403.

68. Clow A, Thorn L, Evans P, Hucklebridge F. The awakening cortisol response: Methodolgical issues and significance. Stress. 2004;7:29–37.

69. Reaves BA. Census of state and local law enforcement agencies, 2002. Bur Justice Stat Bull, NCJ. 2004;2:2015–2030.

70. Theorell T. Biological stress markers and misconceptions about them. Stress and Health. 2003;19:59–60.

71. Kumiko H, Kadirvelu A, Raja E, Takeshi H, Junichi Y, Shunichi H, Chim C. Measurement of coronary vasomotor function: Getting to the heart of the matter in cardiovascular research. Clinical Science. 2004; 107:449–460.

72. Widlansky ME, Gokce N, Keaney JF Jr, Vita JA. The clinical implications of endothelial dysfunction. J Am Coll Cardiol. 2003;42: 149–1160.

73. Anderson TJ. Assessment and treatment of endothelial dysfunction in humans. J Am Coll Cardiol. 1999;34:631–638.

74. Kuvin JT, Karas RH. Clinical utility of endothelial function testing: Ready for prime time? Circulation. 2003;107:3243–3247.

75. Chambless LE, Folsom AR, Clegg LX, Sharrett AR, Shahar E, Nieto FJ, Rosamond WD, Evans G. Carotid wall thickness is predictive of incident clinical stroke: The Atherosclerosis Risk in Communities (ARIC) Study. Am J Epidemiol. 2000;151:478–487.