FINAL PROGRESS REPORT

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List of Terms and Abbreviations

CVD – cardiovascular disease

Inflammatory mediators – a group of biomarkers, primarily cytokines, which contribute to either the promotion or inhibition of inflammation in the lining of blood vessels.

Abstract

Law enforcement officers have higher all-cause morbidity and mortality than the general population. Cardiovascular disease (CVD) accounts for a significant portion of this excess illness, with published prevalences as high as 1.7 times that of the general population. There are over 17,000 criminal justice agencies in the United States that employ over 800,000 sworn officers. Therefore, identifying job-related sources of cardiovascular disease is a crucial step towards reducing this excess prevalence. Since the prevalence of traditional CVD risk factors among law enforcement officers is similar to the general population, other mechanisms must be involved in this excessive morbidity. Cardiovascular disease is now recognized as an inflammatory disease and inflammatory mediators are an important part of this process. Stress can markedly alter the production of several pro- and anti-atherogenic inflammatory mediators. Therefore, this research project tested the hypotheses that 1) compared to the general population, law enforcement officers have higher levels of pro-atherogenic inflammatory mediators and lower levels of anti-atherogenic inflammatory mediators and 2) job-related stress is associated with these differences.

To test these hypotheses, inflammatory mediators (IL-1β, IL-6, TNF-α, C-reactive protein, fibrinogen, IL-4, IL-10), global stress, job-related stress (job strain, vital exhaustion, effort-reward imbalance) and social support were compared among 444 sworn law enforcement officers of the Iowa Department of Public Safety and 166 demographically similar non-officers. Neither global CVD risk, expressed as the Framingham Risk Score, nor the number of metabolic syndrome risk factors differed between these two groups. IL-4 did not differ significantly between the two groups while four of the remaining six mediators were higher in the officers (range = 1.8 – 5.2 fold higher). The other two were 25-33% lower. Global stress was higher in the control participants than the officers as was job strain and vital exhaustion. Effort-reward imbalance was higher in the officers, primarily due to relatively greater extrinsic effort. In both groups, social support was equally high. However, none of these stress measures explained more than 4% of the variability in any of the inflammatory mediators.

The key findings of this research project are 1) law enforcement officers may be at an increased risk for CVD due to a relatively pro-inflammatory vascular milieu but 2) this increased risk does not appear to be associated with either job-related stress or traditional CVD risk factors. The mechanisms underlying this pro-inflammatory profile remain uncertain, although candidates are shiftwork and poor sleep quality. Both are common to law enforcement and limited evidence suggests that both are associated with inflammation.

The utility of these results to the workplace is primarily recognizing that members of the law enforcement profession may have an increased risk for CVD that would not be detected with a typical medical examination. Most physicians routinely screen for traditional CVD risk factors, such as hypertension, hyperlipidemia and tobacco use. However, the risk factor of increased inflammatory mediators is novel, nontraditional, and not routinely assessed in most physicians' offices. Thus, CVD risk may be underestimated in law enforcement officers.

SECTION 1: Highlights/Significant Findings

The first specific aim of this project was to quantify the levels of pro- and anti-atherogenic inflammatory mediators in law enforcement officers. Our working hypothesis was that, compared to an otherwise similar general population cohort, law enforcement officers will have

higher levels of pro-atherogenic C-reactive protein, fibrinogen, TNF- α, IL-1β, IL-6, and IL-18 as well as lower levels of anti-atherogenic IL-4 and IL-10. This specific aim was achieved with this project and our working hypothesis was partially supported. Of the five pro-atherogenic inflammatory mediators that were ultimately assessed (it was not practicable to assess IL-18 with the final laboratory protocol), three were higher in the officers and two were higher in the comparison group. Of the two anti-atherogenic inflammatory mediators, one did not differ between the two groups while the other one was higher in the officers. However, an assessment of the effect sizes (i.e., Cohen's *d*) suggests that officers have a more pro-inflammatory vascular environment. Of the six inflammatory mediators that differed between the two groups, the three largest effect sizes were seen with the three pro-inflammatory mediators which were elevated in the officers. The smallest effect size was seen with the anti-atherogenic inflammatory mediator. Based on these data, we conclude that law enforcement officers may be at an increased risk for cardiovascular disease (CVD) due to a pro-inflammatory vascular enviornment. Our data also suggest that this increased risk cannot be explained by group differences in either traditional CVD risk factors or risk for the metabolic syndrome.

The second specific aim was to determine the forms of job-related stress most associated with changes in these inflammatory mediators. Our working hypothesis was that vital exhaustion, job strain, and effort-reward imbalance will affect these mediators. This specific aim was also achieved but our hypothesis was not supported. None of the measures of job-related stress were associated with any of the inflammatory mediators. This finding was true whether the two subject groups were assessed separately or collectively. We therefore conclude that job-related stress does not appear to be associated with changes in inflammatory mediators. A caveat to this conclusion is that, as a group, these subjects were not greatly stressed. In other words, this hypothesis may have been supported if a broader range of stress levels had been found among the 610 subjects.

Translation of Findings

The long-range goal of our research is to reduce the incidence of cardiovascular disease-related illness and death among law enforcement officers. The objective of this proposal was to test the hypothesis that officers have a higher risk of cardiovascular disease due to alterations in pro- and anti-atherogenic inflammatory mediators. We found evidence supporting this hypothesis. However, as with all research, these findings need to be replicated by other researchers studying other law enforcement populations before a clear "law enforcement and inflammation" connection can be made with confidence. Moreover, the proposed mechanism for this increase, job-related stress, does not appear to be an important contributor. We therefore remain uncertain of the specific mechanism(s) underlying the pro-inflammatory vascular environment seen in law enforcement officers. Thus, it is premature to make specific recommendations regarding how to reduce the occupational hazards contributing to this specific risk factor for cardiovascular disease in law enforcement.

Our findings reinforce the need for law enforcement officers to be regularly screened, and treated, for traditional cardiovascular disease risk factors. Despite the uniqueness of the law enforcement profession relative to the general population, the standard recommendations for the latter remain true for the former: modifying traditional risk factors remains the single best way

of reducing cardiovascular disease in law enforcement. For example, the officers in the present study had a 10-year risk for cardiovascular disease ~50% above the desired risk.

On the other hand, a recent American Heart Association/Centers for Disease Control and Prevention consensus report (Pearson et al., 2003) recommended measuring C-reactive protein (one of the biomarkers assessed here) in asymptomatic subjects at intermediate risk for developing clinically significant cardiovascular disease. We do not feel that the added expense of assessing these biomarkers is a cost-effective measure for most law enforcement departments desiring to reduce cardiovascular disease risk in their officers. In his review of biomarkers, Koenig (2007) also concluded that the currently available biomarkers do not appreciably improve risk prediction.

Future investigative activities should focus on determining the mechanisms underlying the pro-inflammatory vascular environment found in the present study. The most likely mechanisms are shiftwork, or irregular and unpredictable work hours, and poor sleep habits – both can affect the local vascular environment. More than 77% of the officers in the present study reported either shiftwork or irregular work hours while only 27% of the control group did. Moreover, the majority of the officers in this study changed their work shift every 2 weeks, which is more frequent than commonly recommended. Shiftwork can also affect sleep quality but poor sleep is independently associated with inflammation and cardiovascular disease mortality. In assessing law enforcement officers, we recently found poor sleep (i.e., poor sleep quality, altered sleep duration) to be associated with increased risk for cardiovascular disease and increased self-reported stress, burnout, and depression scores. Unfortunately, we did not have the funding to assess inflammatory mediators in this pilot study.

Outcomes/Relevance Impact

The outcomes of this study can be used to guide future investigations and research in the following ways. First, this study should be replicated in larger and more diverse law enforcement groups. If the findings of those studies are consistent with the present study, then a mechanism contributing to the increase prevalence of cardiovascular disease seen in the law enforcement occupation will have been identified with reasonable certainty. Second, the mechanisms contributing to the altered inflammatory profile in the officers need to be identified. If these mechanisms are associated with the occupation (e.g., inappropriate shiftwork practices), then recommendations can be made regarding how to attenuate these contributory mechanisms.

SECTION 2: Scientific Report

Background

Law enforcement officers have higher cardiovascular disease-related morbidity and mortality than the general population (Dubrow et al. 1988; Franke et al., 1998; Calvert et al., 1999; 1-3). This increased prevalence is often attributed to an increased prevalence of traditional CVD risk factors such as physical inactivity, hypercholesterolemia, hypertension, tobacco use, obesity, and hyperinsulinemia (Williams et al., 1987; Pyörälä et al., 2000). However, cross-sectional and longitudinal comparisons of risk factor prevalence in law enforcement officers and general population cohorts suggest that, in general, both groups have an equal risk for CVD due

to traditional risk factors (Franke et al., 1997). Thus, other factor(s) likely contribute to this increased CVD-related morbidity in law enforcement officers.

Job-related stress appears to be a likely culprit. A causal relationship likely exists between psychosocial stress and CVD (Hemingway and Harmot, 1999; Krantz and McCeney, 2002) and police work is often considered to be uniquely stressful (Finn and Tomz, 1997; Agdollahi, 2002). What is underappreciated is that chronic stressors, often due to organizational factors, are more injurious than acute stressors, such as responding to critical incidents, in law enforcement. The latter are typically short-term stressors, relatively infrequent, and mechanisms often exist to attenuate the effects of this stress (e.g., stress debriefings). On the other hand, chronic, organizationally-based stressors are often ignored since they are sub-acute.

Specific Aims

Stress may affect CVD risk due to its effects on inflammatory mediators. Psychological stress can affect the balance of pro- and anti-inflammatory mediators (Miller and O'Callaghan, 2002; Steptoe et al., 2007) and many of these mediators contribute to the development of atherogenic plaques. Thus, law enforcement officers may have an increased risk for CVD due to job-related stress leading to a pro-inflammatory vascular environment. With this study, this hypothesis was tested by pursuing the following specific aims:

1. Quantify the levels of pro- and anti-atherogenic inflammatory mediators in law enforcement officers.
2. Determine the forms of job-related stress most associated with changes in these inflammatory mediators.

Methods

Participants

Sworn officers of the Iowa Department of Public Safety were invited to participate in this study as they underwent their annual medical evaluation; 466 of these 531 officers (88%) agreed to do so. Most were members of the State Patrol (n=322) and the remainder were members of either the Division of Criminal Investigation, Division of Narcotics Enforcement, or the State Fire Marshal's office. The comparison group (n=177) consisted of civilians who were demographically and socioeconomically similar to the officers.

Exclusion criteria for this study were the following: a history of cardiovascular disease, recent surgery, or any acute illness; self-reported ingestion of large quantities of either nonsteroidal anti-inflammatory medications or any glucocorticoids, having ever been treated for an autoimmune disease, or if they provided incomplete data. 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 and weight was assessed to the nearest 0.1 kg while wearing light clothing but no shoes. After 5 minutes seated rest, blood pressure was measured twice with a mercury sphygmomanometer. A 30 ml fasting blood sample was acquired with half the blood used for assessment of lipids and glucose by a large commercial laboratory (Quest Diagnostics, Wood Dale, IL) and the other half used for assessment of inflammatory mediators. The latter blood was allowed to clot for 2 h and the serum frozen at -80° C for later analysis. After all the blood samples had been acquired, high-sensitivity ELISA

kits (Millipore, Billerica, MA) were used to quantify the presence of pro-atherogenic IL-1β, IL-6, TNF-α, C-reactive protein and fibrinogen, and anti-atherogenic IL-4 and IL-10. All samples were run in duplicate.

Psychological measures.

Global stress was assessed using the Perceived Stress Scale (Cohen et al., 1983). Three other forms of stress, germane to law enforcement were assessed. They were job strain (Bosma et al., 1997), vital exhaustion (Kopp et al., 1998), and effort-reward imbalance (Peter and Siegrist, 1999). Since social support can moderate these effects, it was assessed using the Social Provisions Scale (Cutrona and Russell, 1987).

Data Analyses.

Body mass index (BMI, $kg \cdot m^2$) was determined using height and weight. Global risk for CVD, based on the traditional risk factors of gender, age, LDL-cholesterol, HDL-Cholesterol, blood pressure, smoking status and presence of diabetes, was estimated using the Framingham Risk Score (Wilson et al., 1998). Risk factors for the metabolic syndrome were identified according to the National Cholesterol Education Program Adult Treatment Panel III criteria (NIH, 2001). A BMI >30 $kg \cdot m^2$ was used instead of waist circumference as the criterion for abdominal obesity (Farin et al., 2006). Thus, the risk factors were BMI ≥30 $kg \cdot m^2$, triglycerides ≥150 $mg \cdot dl^{-1}$, HDL- cholesterol <40 $mg \cdot dl^{-1}$, blood pressure ≥130 mmHg systolic and/or ≥85 mmHg diastolic or currently using antihypertensive medications, and fasting plasma glucose ≥110 $mg \cdot dl^{-1}$ 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. In order to determine the extent to which psychosocial factors were most associated with changes in these inflammatory mediators, the measures of stress (i.e., 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, shiftwork 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 (i.e., officers and non-police participants). This procedure permitted testing for interactions between group membership and the predictor variables. All data are presented as mean ± SD with statistical significance set at $p < 0.05$.

Results

Cardiovascular disease risk characteristics are presented in Table 1. While there were group differences in some of some of the traditional CVD risk factors, neither the Framingham Risk Score nor the number of metabolic syndrome risk factors differed between the two groups. Surprisingly, global stress was higher in the control participants than the officers (Table 2) as was job strain and vital exhaustion. Effort-reward imbalance was higher in the officers, primarily due to relatively greater extrinsic effort. Both groups had similarly high social support.

Specific Aim 1: Quantify the levels of pro- and anti-atherogenic inflammatory mediators in law enforcement officers.

With the exception of IL-4, the inflammatory mediators differed significantly between the two groups (Table 1). Concentrations of four of the remaining six mediators were approximately two to three times higher in the officers (range = 1.8 – 5.2). Fibrinogen and C-reactive protein were lower (25 and 33%, respectively) in the officers.

Specific Aim 2: Determine the forms of job-related stress most associated with changes in these 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 whether the data were analyzed within the 2 groups or across the entire cohort.

Discussion

Specific Aim 1: Quantify the levels of pro- and anti-atherogenic inflammatory mediators in law enforcement officers.

Of the seven inflammatory mediators assessed here, five are generally considered pro-atherogenic (i.e., IL-1β, IL-6, TNF-α, C-reactive protein, fibrinogen) while two are anti-atherogenic (i.e., IL-4, IL-10). Three pro-atherogenic mediators were higher in the officers while 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.

Because of the relatively large sample sizes used here, we took steps to ascertain whether some of these statistically significant differences may be spurious. In order to elucidate the relative importance of these mediators on CVD risk, the effect sizes of these differences were determined via Cohen's *d* (34; Table 1). Of the six inflammatory mediators which differed between groups, the largest effect sizes were found in the three pro-atherogenic inflammatory mediators that were higher in the officers than the non-officers. Consequently, we interpret these data as suggesting the officer group had a more pronounced pro-atherogenic inflammatory milieu than the comparison group. Thus, the hypothesis associated with this specific aim was supported.

Specific Aim 2: Determine the forms of job-related stress most associated with changes in these inflammatory mediators.

None of the stress measures were statistically associated with any of the inflammatory mediators. Surprisingly, the comparison group was more stressed than the officers. Besides having a higher mean global stress score, a disproportionately high number of controls were in the highest quartile of stress (i.e., 45% of the control group vs 17% of the officers). Nevertheless, none of the inflammatory mediators differed between the highest quartile of subjects and the lowest quartile. Moreover, while three of the four stress measures were higher in the control group, none of the stress measures were predictive of the inflammatory variables. Based on these data, chronic job-related stress does not appear to contribute to CVD in law enforcement officers by altering their inflammatory profile Thus, the hypothesis associated with this specific aim was not supported.

Clearly, stress is not a mechanism underlying the pro-inflammatory profile of the officers is uncertain. Of course, the question remains: what is? One candidate is shiftwork. Shiftwork is associated with CVD and this association often remains after traditional CVD risk factors are accounted for (Sookian et al., 2007; Puttonen et al., 2009). In the present study, over 77% of the officers reported having either shiftwork or working irregular, unscheduled hours compared to just 27% of the comparison group. Many of the officers, members of the State Patrol, have a rotating shift schedule such that they work different shifts every 2 weeks. Other officers, largely in the Division of Criminal Investigation, have irregular work hours in that they have to respond to major crimes promptly, regardless of when they occur.

It is unclear how shiftwork may contribute to increased CVD, although Sookian and colleagues (2007) recently reported that leukocyte count, a marker of inflammation, was associated with shiftwork. Relatively young shiftworkers have evidence of early atherosclerosis via a 2.2-fold higher risk of carotid plaques and higher carotid artery intima-media thickness (Joseph et al., 2009). Of relevance to the present 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 (Joseph et al., 2009). Unfortunately, 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. Collectively, these studies provide evidence supporting the notion that differences in shiftwork, rather than work-related stress, may affect inflammatory markers associated with an increased CVD risk.

Sleep quality, either in concert with or independent of irregular work hours, may play a role. Poor sleep quality is associated with inflammation (McNicholas, 2009) and CVD mortality (Mallon et al., 2002). It is also more common in law enforcement officers than non-officers (Vila, 2006). While we did not assess sleep quality in the present study, anecdotal evidence suggested that many officers routinely experienced various markers of poor sleep, such as difficulty falling asleep, fitful sleep, or waking up tired. We have begun exploring these mechanisms and recently found poor sleep (i.e., poor sleep quality, altered sleep duration) to be associated with increased risk for cardiovascular disease and increased self-reported stress, burnout, and depression scores. Unfortunately, we did not have the funding to assess inflammatory mediators in this pilot study.

Conclusions

The purpose of the present investigation was to determine the extent to which law enforcement officers have a higher risk of cardiovascular disease 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. However, group differences in job-related stress were neither associated with, nor predictive of, group differences in the inflammatory mediators. The present 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, since neither the Framingham Risk Score nor the prevalence of Metabolic Syndrome risk factors differed between the two groups.

Table 1. Cardiovascular disease risk factors and inflammatory mediators of this risk.

Variable	Law Enforcement	Control		Cohen's *d*
CVD Risk Factors				
Age (y)	37.4 ± 9.2	42.0 ± 11.3	†	.45
Body Mass Index ($kg \cdot m^2$)	28.6 ± 4.0	28.3 ± 5.9		
Total Cholesterol ($mg \cdot dl^{-1}$)	193.3 ± 35.2	192.7 ± 36.3		
LDL-Cholesterol ($mg \cdot dl^{-1}$)	119.4 ± 30.3	117.7 ± 28.9		
HDL-Cholesterol ($mg \cdot dl^{-1}$)	47.4 ± 11.2	49.7 ± 13.9	*	.21
LDL/HDL ratio	2.6 ± 0.9	2.5 ± 0.8		
Triglycerides ($mg \cdot dl^{-1}$)	134.7 ± 87.2	133.3 ± 84.4		
Glucose ($mg \cdot dl^{-1}$)	92.5 ± 9.7	93.3 ± 22.0		.15
Mean Arterial Pressure (mmHg)	99.3 ± 7.8	95.9 ± 8.6	†	.30
Framingham Risk Score (%)	5.7 ± 3.1	5.1 ± 4.2		
Metabolic Syndrome Risk (# factors)	1.4 ± 1.2	1.3 ± 1.4		
Pro-atherogenic Inflammatory Mediators				
IL-1β ($pg \cdot ml^{-1}$)	6.3 ± 14.9	1.1 ± 2.0	†	.49
IL-6 ($pg \cdot ml^{-1}$)	29.8 ± 40.7	15.1 ± 20.3	†	.45
TNF-α ($pg \cdot ml^{-1}$)	10.5 ± 18.2	5.8 ± 5.6	†	.35
C-reactive protein ($pg \cdot ml^{-1}$)	7.2 ± 11.7	10.6 ± 18.0	†	.27
Fibrinogen ($pg \cdot ml^{-1}$)	0.28 ± 0.14	0.34 ± 0.24	†	.32
Anti-atherogenic Inflammatory Mediators				
IL-4 ($pg \cdot ml^{-1}$)	176.1 ± 223.4	147.6 ± 235.4		
IL-10 ($pg \cdot ml^{-1}$)	46.5 ± 113.4	20.5 ± 73.2	†	.25

Data adjusted for age. * $P < 0.05$, † $P < 0.01$

Table 2. Stress markers.

Variable (possible range)	Law Enforcement	Control		Cohen's *d*
Perceived Stress (0 – 56)	16.7 ± 6.1	21.5 ± 6.7	†	.79
Job Strain (1 – 16)	3.4 ± 0.7	3.6 ± 0.9	†	.24
Job Demands (4 – 16)	10.4 ± 1.8	11.1 ± 2.7	†	.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	†	.36
Sense of Exhaustion (1 – 3)	1.3 ± 0.7	1.6 ± 0.9	†	.37
Effort-Reward Imbalance (0.25 – 4.0)	0.88 ± 0.14	0.79 ± 0.20	†	.54
Extrinsic Effort (5 – 20)	15.5 ± 1.8	13.2 ± 2.7	†	.96
Intrinsic Effort (4 – 16)	9.3 ± 2.2	9.7 ± 2.3	*	.18
Rewards (8 – 32)	28.5 ± 2.7	27.2 ± 2.8	†	.47
Social Support (20 – 96)	81.3 ± 9.9	80.7 ± 9.8		

Data adjusted for age. * $P < 0.05$, † $P < 0.01$

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SECTION 2: Publications

Franke WD, Kohut ML, Russell DW, Yoo HL, Ekkekakis P, Ramey SP: [2010] Is job-related stress the link between cardiovascular disease and the law enforcement profession? Journal of Occupational and Environmental Medicine 52:561-565.

Franke WD, Kohut ML, Russell DW, Yoo H, Ekkekakis P: [2010] Is job-related stress the link between cardiovascular disease and the law enforcement profession? Annual Meeting of the American College of Sports Medicine, Baltimore, MD, June 2-5. (Abstract published in Medicine and Science in Sports and Exercise 42:S414).

SECTION 2: Inclusion of gender and minority study subjects

At the end of this document.

SECTION 2: Materials available for other investigators

Data, in the form of Excel spreadsheets and de-identified, are available to other investigators upon request made to the Principle Investigator.

Inclusion Enrollment Report

This report format should NOT be used for data collection from study participants.

Study Title: Concept: Stress, cytokines, and heart disease in police

Total Enrollment: 610 **Protocol Number:**

Grant Number: R21 OH008270

PART A. TOTAL ENROLLMENT REPORT: Number of Subjects Enrolled to Date (Cumulative) by Ethnicity and Race				
Ethnic Category	**Females**	**Males**	**Sex/Gender Unknown or Not Reported**	**Total**
Hispanic or Latino		6		6 **
Not Hispanic or Latino	55	549		604
Unknown (individuals not reporting ethnicity)				
Ethnic Category: Total of All Subjects*	55	555		610 *
Racial Categories				
American Indian/Alaska Native				
Asian				
Native Hawaiian or Other Pacific Islander				
Black or African American		7		7
White	54	544		598
More Than One Race				
Unknown or Not Reported	1	4		5
Racial Categories: Total of All Subjects*	55	555		610 *

PART B. HISPANIC ENROLLMENT REPORT: Number of Hispanics or Latinos Enrolled to Date (Cumulative)				
Racial Categories	**Females**	**Males**	**Sex/Gender Unknown or Not Reported**	**Total**
American Indian or Alaska Native				
Asian				
Native Hawaiian or Other Pacific Islander				
Black or African American				
White		6		6
More Than One Race				
Unknown or Not Reported				
Racial Categories: Total of Hispanics or Latinos**		6		6 **

* These totals must agree.
** These totals must agree.