Journal of Psychosomatic Research 110 (2018) 54–60

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Sex and family history of cardiovascular disease influence heart rate variability during stress among healthy adults

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ARTICLE INFO

Keywords:
Autonomic function
Cardiovascular disease
Family history
Heart rate variability
Parasympathetic nervous system
Sex differences

ABSTRACT

Objective: Studies of sex differences in heart rate variability (HRV) typically have not accounted for the influence of family history (FH) of cardiovascular disease (CVD). This study evaluated sex differences in HRV response to speech stress among men and women (age range 30–49 years) with and without a documented FH of CVD.
Methods: Participants were 77 adults (mean age = 39.8 ± 6.2 years; range: 30–49 years; 52% female) with positive FH (FH+, $n = 32$) and negative FH (FH−, $n = 45$) of CVD, verified with relatives of participants. Cardiac activity for all participants was recorded via electrocardiogram during a standardized speech stress task with three phases: 5-minute rest, 5-minute speech, and 5-minute recovery. Outcomes included time domain and frequency domain indicators of HRV and heart rate (HR) at rest and during stress. Data were analyzed with repeated measures analysis of variance, with sex and FH as between subject variables and time/phase as a within subject variable.
Results: Women exhibited higher HR than did men and greater HR reactivity in response to the speech stress. However, women also exhibited greater HRV in both the time and frequency domains. FH+ women generally exhibited elevated HRV, despite the elevated risk of CVD associated with FH+.
Conclusions: Although women participants exhibited higher HR at rest and during stress, women (both FH+ and FH−) also exhibited elevated HRV reactivity, reflecting greater autonomic control. Thus, enhanced autonomic function observed in prior studies of HRV among women is also evident among FH+ women during a standardized stress task.

Cardiovascular disease (CVD) remains the leading cause of death in the world [1] despite major reductions in the incidence of CVD during recent decades. Reduced CVD incidence has resulted, in part, from greater awareness of risk factors for CVD and better management of risk. Risk factors for CVD can be categorized into major non-modifiable risk factors (older age, male sex, positive family history), major modifiable risk factors (e.g., hypertension, diabetes, high cholesterol), and modifiable behavioral factors (e.g., smoking, low physical activity, poor diet, excessive alcohol consumption, stress) [2,3]. Excess physiological reactivity to stress also has been posited as a CVD risk factor. Sex is a non-modifiable factor of particular interest because women generally develop CVD approximately 10 years later in life than do men [4]. Studies of sex differences in autonomic function, both at rest and during psychological stress, reveal that premenopausal women generally exhibit higher heart rate (HR) [5] at rest and during stress, relative to men.

One explanation for the paradoxical finding of higher HR and lower CVD risk is that women also may have higher parasympathetic autonomic activity than men, thereby providing protection from at least some forms of CVD (arrhythmia, coronary heart disease). Data from a number of studies indicate greater relative vagal autonomic control among women, as reflected by several parameters in the frequency domain of HRV including lower low frequency (LF) power and lower ratio of LF to high frequency (HF), LF/HF power [6–9]. In addition, a recent meta-analysis of 172 studies evaluating sex differences in HRV confirmed that women generally exhibit higher HR than men, but that women also exhibit greater vagally-mediated autonomic control than men [10].

Whereas past studies of sex differences typically have evaluated HRV at rest or during exercise, postural manipulation, or 24-hour

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https://doi.org/10.1016/j.jpsychores.2018.04.011
Received 8 December 2017; Received in revised form 23 April 2018; Accepted 25 April 2018
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monitoring, fewer studies have addressed psychological/behavioral mechanisms in a controlled laboratory environment. Studies of HRV in the context of a laboratory stressor facilitate evaluation of psychosocial mechanisms that may contribute to any observed sex differences.

Positive family history (FH) of CVD is a significant risk factor for CVD among offspring [11]. Thus, understanding the influence of FH on CVD is critical as an indicator of genetic or family environment influences in sex-related stress responses. Middle-aged individuals with a positive FH of CVD, for example, exhibit exaggerated cardiovascular reactivity to a variety of laboratory stressors, with men having a more consistent and robust response than women [5,12]. Wright and colleagues [13] examined HRV responses to stress among young adults with (n = 75) and without (n = 16) a FH of CVD. In response to psychosocial stress (combination of Stroop task and public speaking task), positive family history of CVD (FH+) was associated with greater HR and HRV reactivity in response to stress only among women. FH did not have an influence on HRV among men. This study examined only the time domain measures of HRV, specifically the root mean square of successive differences (RMSSD); and FH of CVD was defined very broadly to incorporate parent or grandparent history of diabetes, heart disease, high cholesterol, or high blood pressure. FH+ participants did not necessarily have a family history of heart disease, but could be defined as FH+ with a history of two or more related conditions (hypertension, diabetes, high cholesterol) in either parent or a history of three or more related conditions in any grandparent, or a combination of parent and grandparent risk. The resulting sample of FH+ participants represented individuals with greater risk of cardiovascular-related disease, but this operationalization of FH+ is broader than typically used to define FH+ in identifying individuals with elevated genetic risk of heart disease. In addition, the sample was relatively young (age range 18–25).

The current study was designed to evaluate sex differences in HRV response to speech stress among men and women (age range 30–49 years) with and without a documented FH of heart disease. Men and women in the fourth and fifth decade of life were selected for study because they are likely to be experiencing age-related changes in physical function and cardiovascular fitness, but not yet likely to have a diagnosis of CVD. The study utilized a more stringent definition of FH+ than the Wright et al. [13] study and incorporated both time domain and frequency domain indicators of HRV, with the goal of both replicating past findings in this area of research and further extending the work to better explicate possible mechanisms underlying observed sex differences in heart disease risk.

Parental history of myocardial infarction (MI) was used as the indicator of CVD risk to identify individuals with heightened genetic/environmental risk of CVD because both paternal and maternal history of MI increases risk of MI in adult children [14], and because parental history of MI is associated with greater magnitude of blood pressure and heart rate response to stress [15]. In addition, previous investigations have indicated that reports of parental MI by adult offspring are accurate due to the salience of the medical event [16,17], and awareness of parental MI does not require broader knowledge about parent health status or health care utilization. Thus, adults were recruited who would be considered higher risk due to parent history of MI. Based on prior studies, it was hypothesized that women would exhibit better autonomic control as reflected by lower LF and LF/HF ratio and higher vagally-mediated HRV than men. In addition, consistent with data from Wright et al. [13], it was hypothesized that HRV reactivity would be greater among women than among men, and that reactivity would be most pronounced among FH+ women.

1. Methods

1.1. Participants

This study included 77 adults (mean age = 39.8 ± 6.2 years; range: 30–49 years; 52% female) with confirmed FH [both positive (n = 32) and negative (n = 45)] of CVD. The sample included somewhat more women (56%) than men with positive FH. As shown in Table 1, there were only five self-reported smokers in the study, with most of them (n = 4) in the FH+ group.

Table 1
Demographic and health characteristics of the study participants.

Variable	Women (n = 40)		Men (n = 37)	
	FH+ (n = 18)	FH− (n = 22)	FH+ (n = 14)	FH− (n = 23)
	M (SE) or %	M (SE) or %	M (SE) or %	M (SE) or %
Age (years)	42.9 (1.4)[a]	37.5 (1.3)[a]	38.9 (1.6)	40.2 (1.3)
Ethnicity (% white)	78	82	86	70
Body mass index	29.6 (1.2)[a]	25.0 (1.1)[a,b]	28.1 (1.4)	28.8 (1.1)[b]
Cigarette smokers (%)	11	0	14	4
Baseline heart rate (bpm)	64.9 (2.6)	65.7 (2.5)	60.4 (2.9)	59.8 (2.2)
Peak HF (hz)[1]	0.27 (0.01)	0.25 (0.01)	0.28 (0.02)	0.26 (0.01)

Note: Means in a row sharing superscripts are significantly different: [a]$p < 0.01$, [b]$p < 0.05$; FH+ = positive family history of cardiovascular disease; FH− = negative family history of cardiovascular disease.
[1] Mean (SD) central frequency of the high frequency (HF) spectral peak.

Data were collected as part of the Healthy Heart Project, designed to evaluate cardiovascular, lipid, and neuroendocrine responses to stress among community-residing healthy adults with and without a FH of CVD. Prospective participants were recruited via on-line and print advertising for a study requiring three visits to the laboratory, including a screening visit, followed by two experimental visits, separated by at least one week. At the screening visit, participants completed a thorough review of medical history and had a blood draw for measurement of cholesterol, as well as a resting electrocardiogram (ECG). The two subsequent visits included an exercise stress at one visit and a speech stress at the other visit, with the order of visits randomly assigned. HRV data for this study came from the speech stress visit.

Prospective participants were screened by telephone prior to scheduling an initial screening visit. The telephone screen included questions pertaining to sex and family history of CVD, with the goal of recruiting balanced numbers of men and women with and without FH of CVD. In addition, women in the study were premenopausal with normal menstrual cycles (menstrual periods for the previous 3 months occurring every 26–34 days) as assessed by self-report. Women were excluded if they were pregnant, nursing, or using oral contraceptives during the previous 6 months. Prospective participants also were excluded if taking medication for diabetes, hypertension, or CHD; or if diagnosed with renal disease, hyperlipidemia, or any other metabolic disease. Any potential participant with a history of cancer, hepatitis, epilepsy, psychiatric illness, hypertension, or CHD was excluded. A total of 277 prospective participants passed the initial telephone screen and were scheduled for a visit, but 48 of them did not attend the screening visit and could not be contacted or were not interested in rescheduling. Of the 229 participants who completed the screening visit, 15 were excluded due to still not meeting study criteria (e.g., age limit, use of psychiatric medication, elevated resting blood pressure, difficulty with blood draw, abnormal electrocardiogram). Prospective participants completed a detailed questionnaire and interview concerning FH of CVD, and MI specifically. In addition, participants completed the Beck Depression Inventory to confirm the absence of significant depression. No participants were excluded due to depression scores. Following the screening visit, 53 participants did not complete further study visits, primarily due to time conflicts with work or other life demands, resulting in a sample of 161 participants who completed the screening visit and at least one additional visit. Eleven of those

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