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Psychological hardiness predicts cardiovascular health

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ABSTRACT

Many, but not all people experience diminished health, performance and well-being as a function of exposure to stress. However, the underlying neurophysiological processes which characterize hardy or resilient people are not well understood. This study examines psychological hardiness and several indicators of cardiovascular health, including body mass index (BMI) and blood cholesterol markers in a sample of 338 middle-aged adults enrolled in a national security education program. Hierarchical regression analyses reveal that after controlling for the influence of age and sex, high hardiness is related to higher HDL – high density lipoprotein and less body fat (BMI). Lower hardiness is associated with greater total cholesterol to HDL ratio, a cardiovascular disease risk factor. These results suggest that psychological hardiness confers resilience in part through an influence on cholesterol production and metabolism.

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Stress; hardiness; cardiovascular health; cholesterol; resilience

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When exposed to stress, many people experience degraded health and performance while others show more resilient, healthy response patterns. Despite a growing recognition of the value of resilience, underlying processes are still poorly understood. Additional research is needed to develop our understanding of what influences resilient response patterns, and to clarify underlying mechanisms.

One characteristic that distinguishes healthy, resilient people from unhealthy ones is psychological hardiness (Kobasa, 1979; Maddi & Kobasa, 1984). Hardiness was originally described as a personality trait with three interrelated dimensions: *commitment,* tendency to regard life as interesting and meaningful; *control,* belief that one can influence outcomes by taking action; and *challenge*, an adventurous, exploring approach to living. Recently, hardiness is seen as part trait and part state, amenable to change based upon contextual factors (Bartone, 2012). Many studies have found that hardiness protects against the ill-effects of stress on health and performance (Bartone, 1999; Britt, Adler, & Bartone, 2001; Eschleman, Bowling, & Alarcon, 2010; Kobasa & Puccetti, 1983). But despite considerable evidence for a health-protective function of the hardy-resilient response pattern, scant research has

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explored the underlying neurophysiological processes. The present study examines possible links between hardiness and blood cholesterol markers of cardiovascular health.

Cardiovascular disease remains the leading cause of death worldwide (Barter, 2005). Among recognized risk factors for cardiovascular disease are psychosocial stress (National Heart, Lung and Blood Institute Working Group [NHLBI], 2004) and high cholesterol levels (Singh & Mori, 1992). While low density lipoprotein (LDL) is generally seen as the primary culprit in coronary heart disease (CHD), recent attention has focused on the possible protective role of high density lipoprotein (HDL; Barter, 2005). Several early studies found evidence that hardiness is linked to cardiovascular health, as indexed by blood pressure reactivity to stress (Contrada, 1989) and elevated triglycerides (Howard, Cunningham, & Rechnitzer, 1986). The present study goes further by examining hardiness against a full lipid profile including HDL. Theoretically, since HDL appears to function as a protective factor at the biochemical level (Ridker, Stampfer, & Rifai, 2001), it may also be linked to hardiness.

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Methods

This study was approved by the institutional review boards of the National Defense University, and US Department of Defense, Edgewood Chemical Biological Center. Adult students at a major national defense college completed a voluntary hardiness scale (Dispositional Resilience Scale-15) during standard in-processing, and gave informed consent to access their cholesterol screening results. Of approximately 400 students available, 373 (93%) completed surveys. Participants were 67% military and 33% civilian, 22% women and 78% men. Age ranged from 31 to 62 years (mean = 44.07; *SD* = 4.49). Two weeks later, 338 (91%) participants completed a voluntary cholesterol screen, yielding measures of total cholesterol, HDL-C, LDL-C, triglycerides and total cholesterol/HDL ratio. Height and weight were assessed using standard instruments. Body mass index (BMI) provided a measure of total body fat (Harvard School of Public Health, 2015). BMI was calculated as (weight – kilograms divided by height – meters) divided by height – meters.

Hardiness was measured with the Dispositional Resilience Scale (DRS)-15 (Bartone, 1989, 1995; Bartone et al., 2007). The DRS-15 has been used extensively in military and civilian populations with good results (e.g. Bartone, 2006; Bartone, Ursano, Wright, & Ingraham, 1989; Hystad, Eid, Laberg, Johnsen, & Bartone, 2009). The DRS-15 was further revised in 2006 to be more balanced in terms of positive and negative items, and to eliminate idiomatic expressions that are difficult to translate (Bartone et al., 2007). The DRS-15 shows excellent psychometric properties and factorial validity, with three factors (commitment, control and challenge) nested hierarchically under a more general higher order factor (hardiness) (Hystad, Eid, Johnsen, Laberg, & Bartone, 2010). Cronbach's alpha coefficient in the present sample was .75.

Data were analyzed using Statistical Package for the Social Sciences (SPSS). Bivariate relations were assessed with Pearson correlations. Hierarchical regressions tested associations between hardiness and cardiovascular health indicators, controlling for age and sex.

Results

Table 1 displays descriptive statistics and intercorrelations among study variables. Age shows no correlation with other variables, although there is a trend toward higher triglycerides

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Table 1. Correlations and descriptive statistics for key study variables (probability level is shown in italics).

	Mean (*N*)	SD	Age	Sex	BMI	Chol	Trig	HDL	LDL	Chol/HDL	Hardy
Age	44.07 (373)	4.486	1	.094	.068	.078	.098	.046	.012	.042	−.06
				.071	*ns*	*ns*	*.073*	*ns*	*ns*	*ns*	*ns*
Sex	1.22 (373)	.413		1	**−.267**	.087	**−.116**	**.399**	−.087	**−.270**	**.106**
					.0001	*.109*	***.032***	***.0001***	*.109*	***.0001***	***.040***
BMI	26.01 (200)	3.322			1	.072	**.248**	**−.372**	**.165**	**.389**	**−.149**
						ns	***.001***	***.0001***	***.025***	***.0001***	***.035***
Cholesterol	189.93 (338)	31.374				1	**.314**	**.287**	**.824**	**.436**	.010
							.0001	***.0001***	***.0001***	***.0001***	*ns*
Triglycerides	94.89 (338)	67.011					1	**−.366**	.084	**.653**	−.062
								.0001	*ns*	***.0001***	*ns*
HDL	68.99 (338)	16.081						1	−.086	**−.684**	**.157**
									ns	***.0001***	***.004***
LDL	102.28 (338)	26.162							1	**.584**	−.049
										.0001	*ns*
Chol/HDL	2.8665 (338)	.715								1	**−.124**
											.022
Hardy	32.43 (373)	4.769									1

Notes: Age range = 31–62; Sex, 1 = Male, 2 = female; BMI = Body Mass Index; HDL = High Density Lipoprotein; LDL = Low Density Lipoprotein; Chol/HDL = Ratio of Total Cholesterol to HDL; *N*'s are shown in parentheses; Significant correlations are in bold; probability levels shown in italics; ns = not significant.

Table 2. Hierarchical regression analysis for age, sex and hardiness predicting BMI ($N = 199$).

	Model 1			Model 2		
Variable	*B*	*SE B*	*β*	*B*	*SE B*	*β*
Age	.09	.05	.12	.09	.05	.13
Sex	−2.22	.53	−.29***	−2.18	.53	−.28***
Hardiness				−.10	.05	−.14*
R^2			.09		.11	
F for change in R^2			9.17***		4.39*	

Notes: Final Model $F(196, 3) = 7.68$, $p < .001$.
*$p < .05$; **$p < .01$; ***$p < .001$.

Table 3. Hierarchical regression analysis for age, sex and hardiness predicting HDL ($N = 337$).

	Model 1			Model 2		
Variable	*B*	*SE B*	*β*	*B*	*SE B*	*β*
Age	−.001	.18	.00	.02	.18	.01
Sex	15.35	1.94	.40***	14.89	1.94	.39***
Hardiness				.40	.17	.12**
R^2			.16			.17
F for change in R^2			31.75***			5.92**

Notes: Final Model $F(334, 3) = 23.45$, $p < .001$.
*$p < .05$; **$p < .01$; ***$p < .001$.

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with increasing age. Hardiness correlates with sex ($r = .11$, $p < .04$), with women being more hardy and with HDL ($r = .16$, $p < .01$). Hardiness correlates negatively with BMI ($r = -.15$, $p < .03$) and cholesterol/HDL ratio ($r = -.12$, $p < .02$).

The hierarchical regression predicting BMI (Table 2) reveals that female sex and hardiness are associated with lower BMI body fat ($F(196, 3) = 7.68$, $p < .001$; $R^2 = .11$). Next, looking at HDL (Table 3), both female sex and hardiness are associated with higher HDL levels, a healthy pattern ($F(334, 3) = 23.45$, $p < .001$; $R^2 = .17$). Finally, both female sex and low hardiness scores are associated with lower cholesterol/HDL ratios ($F(334, 3) = 10.68$, $p < .001$; $R^2 = .09$). Here, the contribution of hardiness was marginally significant at $p < .06$. These results are summarized in Table 4.

Discussion

This study examines psychological hardiness against several indicators of cardiovascular health, including body fat (BMI) and standard serum cholesterol markers. Results show that hardiness is negatively related to body fat, and positively related to HDL. HDL plays a critical role in removal of excess cholesterol from the bloodstream, thus reducing the risk of atherosclerotic and cardiovascular disease (Tung & Yeo, 2010). In the present study, subjects high in hardiness were also more likely to be high in HDL. Additionally, psychological hardiness is negatively related to total cholesterol/HDL ratio, a strong predictor of cardiovascular disease risk (Lemieux et al., 2001).

One possible explanation for these findings lies in the positive appraisals and coping expectations typically formed by high-hardy persons. These mental activities occur in the pre-frontal cortex, the seat of executive functioning. This area of the brain is engaged in threat appraisal, consideration of response options and the decision to respond in certain ways based upon context, past experience and long-term goals and expectations. More

Table 4. Hierarchical regression analysis for age, sex and hardiness predicting total cholesterol/HDL ratio ($N = 337$).

	Model 1			Model 2		
Variable	*B*	*SE B*	*β*	*B*	*SE B*	*β*
Age	.01	.01	.07	.01	.01	.07
Sex	−.48	.09	−.28***	−.46	.09	−.27***
Hardiness				−.01	.01	−.10*
R^2		.08			.09	
F for change in R^2		14.24***			3.37*	

Notes: Final Model $F(334, 3) = 10.68$, $p < .001$.
*$p < .06$; **$p < .01$; ***$p < .001$.

specifically, the appraisal and judgment process involves the superomedial prefrontal cortex including the anterior cingulate gyrus (Suchy, 2009), and the anterior prefrontal cortex (Koechlin, Basso, Pietrini, Panzer, & Grafman, 1999). These brain areas have abundant bidirectional pathways to a variety of limbic structures including the amygdala and hypothalamus (Thayer & Lane, 2000, 2007). The hypothalamus in turn is known to play a critical role in cholesterol regulation, through the hypothalamic melanocortin signaling system, which directly regulates synthesis and re-uptake of cholesterol (HDL-C) in the liver (Perez-Tilve et al., 2010).

The positive stress appraisals made by high-hardy persons will tend to maintain the inhibitory control exercised by prefrontal cortical executive function over more primitive subcortical structures and functions, notably the amygdala-regulated fear response. In contrast, more pessimistic (non-hardy) threat appraisals will lead to rapid relinquishing of executive control functions in favor of fear-based responses. The hypothalamic melanocortin signaling system may respond variably to different stimuli received through these central autonomic neural pathways. Thus, increased activity of the hypothalamic melanocortin system associated with enhanced prefrontal cortical control provides one possible explanation for higher HDL levels in high-hardy persons, since the melanocortin system regulates HDL synthesis (Perez-Tilve et al., 2010). This is an intriguing possibility for future research to explore.

Another possibility is that higher levels of HDL observed in high-hardy persons stand out in contrast to increased cholesterol production in low-hardy persons, who respond with less executive control and greater autonomic nervous system activation in reacting to stress. This would lead to stronger and more prolonged stress reactions, including release of classic stress hormones, cortisol and adrenaline. This could signal the body to produce more cholesterol, the primary building block of steroid hormones. The low-hardy person who is chronically hyperreactive to changing conditions may then overproduce cholesterol, leading to a depletion of HDL-C and an excess of LDL-C relative to HDL-C.

Another, indirect explanation for the present findings may be that high-hardy people tend to make positive lifestyle choices, including exercising and healthy dieting, and it is this that influences HDL cholesterol levels. A related possibility is that exercise or diet influences HDL cholesterol, while also leading to an increased sense of hardiness as one sees positive results of these activities.

Some limitations of this study should be noted. First, the present results are correlational and should not be taken as definite evidence of causality. Also, since the sample consisted of adult students at a US national defense college, results may not be generalizable to the

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broader civilian population. Another limitation is that several variables were not measured that may influence the relation between hardiness and cholesterol, to include exercise, diet, medications and family history. Future studies should attempt to control for these variables.

The present study has demonstrated that psychological hardiness is associated with cardiovascular health, as indexed by HDL, cholesterol/HDL ratio and body fat. Additional research is merited to assess possible explanations for this effect, and to specify the underlying mechanisms and causal sequences involved. A better understanding of these processes should lead to more effective strategies for reducing stress-related health problems, particularly cardiovascular disease.

Disclosure statement

No potential conflict of interest was reported by the authors.

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