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High frequency heart-rate variability predicts adolescent depressive symptoms, particularly anhedonia, across one year

Lauren Vazquez^a, Julia D. Blood^b, Jia Wu^{a,f}, Tara M. Chaplin^c, Rebecca E. Hommer^d, Helena J. V. Rutherford^{a,f}, Marc N. Potenza^{a,g,h,i,j}, Linda C. Mayes^{a,f}, and Michael J. Crowley^{a,e,f,*}

^aYale Child Study Center, Yale School of Medicine, New Haven, CT, USA

^bSchool of Medicine, University of Texas Health Science Center, San Antonio, TX, USA

^cDepartment of Psychology, George Mason University, Fairfax, VA, USA

^dEmotion and Development Branch, National Institute of Mental Health, National Institutes of Health, Bethesda, MD, USA

^eProgram for Anxiety Disorders, Yale Child Study Center, New Haven, CT, USA

^fDevelopmental Electrophysiology Laboratory, Yale Child Study Center, New Haven, CT, USA

^gDepartment of Psychiatry, Yale School of Medicine, New Haven, CT, USA

^hDepartment of Neuroscience and CASAColumbia, Yale University School of Medicine, New Haven, CT, USA

ⁱConnecticut Mental Health Center, Yale University School of Medicine, New Haven, CT, USA

^jCASAColumbia, Yale University School of Medicine, New Haven, CT, USA

Abstract

Background—Few prospective studies examine the link between lower heart rate variability (HRV) and depression symptoms in adolescents. A recent animal model specifically links HRV to anhedonia, suggesting a potential translational model for human research.

Method—We investigated the association between spectral measures of resting HRV and depressive symptoms measured one year later, among 73 adolescents, aged 11–18 years. We evaluated (1) the predictive power of relative high frequency (HF) HRV, relative low frequency (LF) and relative very low frequency (VLF) HRV for depressive symptoms; and (2) the relative strength of association between HF HRV and depressive symptomatology (anhedonia, negative mood, interpersonal problems, ineffectiveness, negative self-esteem).

Results—HF HRV significantly predicted self-reported depressive symptoms across one year, controlling for age, puberty and sex. HF HRV was most strongly associated with anhedonia one year later, after considering other facets of depressive symptomatology.

*Correspondence to: Yale Child Study Center, 230 South Frontage Rd., New Haven, CT 06520, USA. michael.crowley@yale.edu (M.J. Crowley).

Conclusions—Results provide support for the prospective relationship between relative HF HRV and depressive symptoms among adolescents across one year. Findings concur with rodent models that suggest a specific link between HF HRV and anhedonia.

Limitations—We investigated relative spectral power HF HRV and depressive symptom dimensions. We cannot make strong claims about these associations in clinical depression. Physical activity levels could be controlled in future work.

Keywords

Adolescence; Depression; Anhedonia; Heart rate variability; Time-frequency analysis

1. Introduction

A growing body of work examines the link between depression and cardiac function, particularly heart-rate variability (HRV) (Carney et al., 2005; Henje Blom et al., 2014; Yaroslavsky et al., 2014). HRV, thought to reflect the impact of the sympathetic and parasympathetic branches of the autonomic nervous system on cardiac function, refers to the measurement of beat-to-beat changes in heart rate (Berntson et al., 1997). While high HRV is associated with healthy cardiac activity—low HRV suggests inadequate parasympathetic or excessive sympathetic activity (Michels et al., 2013; Task Force, 1996). HRV is thought to index the nervous system's impact on the regulation of physiological arousal in proportionate response to an environmental challenge (Appelhans and Luecken, 2006; Thayer and Lane, 2000). HRV is consistently linked to emotion regulation ability—response to stress is associated with HRV reductions (Berntson et al., 1993; Porges et al., 1994), and low resting HRV is associated with negative emotionality and depressive symptoms (Beauchaine, 2001; Thayer and Lane, 2000).

Research investigating the high co-morbidity of adult heart disease and depression found that HRV partially mediated this link (Bhattacharyya et al., 2008; Carney et al., 2005; Celano and Huffman, 2011). Healthy adult studies generally find an association between low HRV and depressive disorders (Gorman and Sloan, 2000; Licht et al., 2008). Similar findings have emerged for youth (Forbes et al., 2006; Shannon et al., 2007; Tonhajzerova et al., 2010). However, demographic factors also need consideration when examining depression-HRV linkages in youth because changes in age and puberty are associated with changes in HRV (Tanaka et al., 2000), and sex might play a role in the link between HRV and depression (Greaves-Lord et al., 2007).

One relevant translational line of research links cardiac function, depression, anhedonia and stress within a rodent model. Depression induced by stress paradigms, (e.g. hindlimb unloading, chronic mild stress) is related to behavioral changes including decreased sucrose intake (a measure of anhedonia) and decreased spontaneous locomotor activity, suggesting reduced appetitive drive (Grippe et al., 2006, 2005; Grippe and Johnson, 2009). Stress-related physiological alterations associated with rodent models of depression include autonomic and cardiovascular changes (e.g. elevated resting heart rate, elevated sympathetic tone, decreased HRV) and immune function changes (e.g. increased pro-inflammatory cytokines, decreased immune response) (Grippe et al., 2003, 2005, 2008; Grippe and

Johnson, 2009; Moffitt et al., 2008). This work is particularly relevant because it dovetails with recent human work linking stress, anhedonia, and depression (Pizzagalli et al., 2007).

Few studies examine cardiac activity and depressive symptoms among adolescents prospectively (Yaroslavsky et al., 2014) or discuss HRV and depressive pathophysiology within a translational framework. Our study had two goals. First, we examined HRV at Time 1 [T1] as a predictor of depressive symptoms one year later [Time 2; T2]. Second, we evaluated the relative importance of T2 anhedonia and other depressive symptom facets (negative mood, interpersonal problems, ineffectiveness, negative self-esteem), reflecting the five-factor structure of the Children's Depression Inventory (CDI; Kovacs, 1992), to explain variability in T1 HF HRV.

2. Methods

2.1. Participants

Participants in this report were part of a larger study (Blood et al., 2015), about stress, reward, and risk-taking with 160 typically developing children and adolescents recruited via mass mailings to greater New Haven, CT. Here we report on 73 healthy adolescents (34 boys; $M/SD=14.82/2.12$ years) returning for a one-year follow-up. Participant ethnicities were: 76.7% Caucasian, 9.6% Hispanic or Latino, 6.8% African American, 5.5% Asian, and 1.4% Other. Included participants provided sufficient resting heart rate (HR) data for analysis, were medication free during their electrocardiogram (ECG), and reported no prior history of psychosis, autism, or bipolar disorder. Participants were not excluded based on prior history of depression. Four participants scored at or above the cutpoint for clinical levels of depressive symptoms on the T2 CDI (Costello and Angold, 1988). This research was approved by the Yale School of Medicine Human Investigation Committee.

2.2. Procedure

This study follows up on our previous report that concurrently linked depressive symptoms and HRV (Blood et al., 2015). We recorded participant resting ECG for 7 min. Informed adolescent assent and parental consent were obtained for all participants. Participants received \$60 at T1 and \$40 at T2.

2.3. Measures

2.3.1. Cardiovascular response—ECG was recorded at T1 using a 3-lead Coulbourn Instruments Holter electrocardiogram, sampled at 1000 Hz, with post processing artifact removal done in QRS tool and spectral analysis done with Kubios software. Three leads were placed – one on the left lower back, one on the right upper back and one on the left arm as a ground. Data processing steps are detailed elsewhere (Blood et al., 2015). Participants with at least six minutes of artifact free data were included (mean 6:57; $SD=00:07$). Fourier transform (FFT) was used to spectrally analyse IBI data. We extracted three HRV frequency ranges thought to reflect different physiological processes (Shaffer et al., 2014): very low frequency (VLF: .0033–.04 Hz), low frequency (LF: .04–.15 Hz) and high frequency (HF: .15–.4 Hz). Relative power was computed as the percentage of total power in each frequency band (Yeragani et al., 1997).

2.3.2. Depressive symptoms—Adolescents completed the CDI (Kovacs, 1992) at T1 and T2. The CDI contains 27 items scored from 0 to 2. The CDI measures clinical depression and provides a total score and five subscales (anhedonia, negative mood, interpersonal problems, ineffectiveness, negative self-esteem), and was recently replicated in a large sample of children and adolescents ($n=4,707$) (Garcia et al., 2008).

2.3.3. Pubertal status—Adolescents completed the Pubertal Development Scale (PDS) Self Report and their primary caregiver completed a Parent Report (Petersen et al., 1988) at T1. The 5-item PDS estimates pubertal status based on the presence or absence of developmental features (4-point format). We used the mean of both informants.

2.4. Data analysis

Main study variables included the CDI (T1 and T2), T2 CDI subscales, and T1 spectral power in VLF, LF and HF bands. Analysis included linear regression, (1) using the T2 CDI as the criterion variable with covariates (sex, age, puberty) and T1 HRV spectral relative power measures as predictor variables, (2) then with T1 CDI added to the model, and (3) using T2 CDI subscales to account for variability in T1 HF HRV. Because age, sex, and pubertal status influence HRV, we considered these variables in our correlation and regression analyses.

3. Results

Means and SDs for study variables are presented in Table 1. Correlation analyses (Table 1) revealed that relative T1 and T2 HF HRV was negatively associated with several T2 CDI subscales. T1 LF HRV was positively related to T2 anhedonia, but not related to T2 depression overall. Contrary to our previous report, T1 VLF HRV was not related to T2 depressive symptoms. As expected, sex, age, and pubertal status were significantly associated with HRV (Table 1). We controlled for these variables in our models. Females and males were comparable on depressive symptoms, ($t(71) = -1.48, p=.14$).

To evaluate the predictive power of relative T1 HF HRV in explaining variability in T2 depressive symptoms, we used hierarchical linear regression, first entering age, sex and pubertal development (see Table 2). While HF has the most support in the literature as a correlate of depressive symptomatology (Carney et al., 2001; Celano and Huffman, 2011), other reports found that VLF predicted concurrent depressive symptoms (Blood et al., 2015; Vaccarino et al., 2008). Thus we considered the predictive power of relative HF, LF, and VLF. Age, puberty and sex were first entered into the model $F(3, 72)=1.41, p=.25, R^2=.045$. Then, HF, LF, and VLF HRV were added with forward selection, yielding a significant model, $F(4, 72)=3.79, p=.002, R^2=.165, R^2=.12$. Only sex and HF contributed significantly to the model ($ps<.05$, see Table 2). Next, with T1 depressive symptoms entered, the overall model was significant in predicting T2 depressive symptoms, $F(5, 72)=11.96, p<.001, R^2=.47$, but HF HRV was no longer significant, $p=.108$ (Table 2, Model 3). Because both T1 and T2 depressive symptoms significantly relate to T1 HF HRV, this suggests a stable linkage between HF HRV and depressive symptoms.

Next, building on a translational animal model linking anhedonia to decreased HRV, we considered the relative significance of the five **T2** CDI depression subscales for explaining variability in T1 HF HRV. We used hierarchical linear regression, entering the five CDI subscales with a forward selection procedure (Table 3). Only T2 anhedonia continued to account for significant variance in T1 relative HF HRV, $F(5,72)=10.39$, $p=.002$, $R^2=.12$, $R^2=.12$, suggesting anhedonia is the specific depressive symptomatology that shows the strongest longitudinal link with variance in HF HRV.

4. Discussion

This study yielded three important findings. First, we observed a negative association between T1 relative HF HRV and T1 and T2 depressive symptoms, with reduced relative HF associated with greater depressive symptoms. This finding is consistent with previous research linking decreased HF HRV with depressive symptoms among adolescents (Henje Blom et al., 2010; Michels et al., 2013; Tonhajzerova et al., 2010). We also found a positive association between T2 depressive symptoms and T1 relative LF HRV. The literature on LF HRV and internalizing symptoms is mixed (Greaves-Lord et al., 2007; Schmitz et al., 2013) suggesting need for replication. Our findings suggest that youth with greater depressive symptoms may have greater relative sympathetic influence on their HRV. In contrast with previous reports based on concurrently assessed HRV and depression (Blood et al., 2015; Vaccarino et al., 2008), we did not find an association between T1 relative VLF HRV and T2 depressive symptoms. Given the dearth of previous longitudinal research linking depression and VLF HRV, we speculate this may reflect the different neurophysiological mechanisms underlying VLF, LF, and HF HRV and their respective connections to psychological functioning over time (Shaffer et al., 2014), although our modest sample size could have also been a factor.

Second, after considering demographic covariates, we found that only T1 HF HRV significantly predicted variability in T2 depressive symptoms. This finding adds to previous literature documenting a prospective link between autonomic functioning and depressive symptoms among adolescents (Henje Blom et al., 2010; Yaroslavsky et al., 2014). Moreover, consideration of T1 depressive symptoms suggests that the association between depressive symptoms and HF HRV largely reflects a more trait like association, persistent across one year.

Finally, we observed that after considering anhedonia, negative mood, interpersonal problems, ineffectiveness, negative self-esteem, only anhedonia one year later significantly accounted for the variance in relative HF HRV. This finding aligns with rodent models of depression concurrently linking stressor-induced behavioral indicators of anhedonia with decreased HRV, elevated sympathetic tone, and sympathovagal imbalance (Grippe et al., 2006, 2008; Moffitt et al., 2008). Although our study has important differences from the rodent studies, i.e. self-report versus behavioral assessment of anhedonia and longitudinal versus concurrent assessment, our results provide the first preliminary evidence for the translational link between anhedonic features of depression and cardiac function among adolescents.

5. Limitations and future directions

We studied depressive symptoms dimensionally. Thus we cannot make strong claims about HRV and clinical depression. Further, although no participants were taking anti-hypertensive medications, we did not exclude youth for hypertension. This and other relevant influences on HRV including physical activity/exercise behavior, should be considered in future research. Finally, our sample size was modest, warranting replication with a larger sample.

The high comorbidity of depression and cardiovascular disease elucidates the need for further research investigating early markers of depression risk, cardiovascular risk and their interplay. Animal models of depression have revealed common mechanisms between depression and cardiovascular function including autonomic function, baroreflex receptor function, and immune function with linkages to stress and anhedonia (Grippe and Johnson, 2009). Future studies should focus on these interrelated factors (i.e. HRV, blood pressure, pro-inflammatory cytokines) in children and adolescents to further examine the association between cardiac function and depressive pathophysiology, particularly anhedonia.

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Abbreviations

HRV	Heart rate variability
HF	High frequency
LF	Low frequency
VLF	Very low frequency
ANS	Autonomic nervous system
RSA	Respiratory Sinus Arrhythmia
MDD	Major Depressive Disorder
HR	Heart rate
ECG	Electrocardiogram
IBI	Inter-beat interval
FFT	Fast Fourier Transform
CDI	Children's Depression Inventory
EATQ DM	Early Adolescent Temperament Questionnaire Depressive Mood Scale
PDS	Pubertal Developmental Scale

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Table 1

Means, standard deviations and correlations for major study variables.

Sex	Age	PDS-S	PDS-P	Mean-PDS	CDI T1	CDI T2	CDI A-T2	CDI NM-T2	CDI IP-T2	CDI I-T2	CDI NS-T2	VLF	LF	HF
Female	13.91 (2.17)	2.66 (.46)	2.60 (.46)	2.64 (.39)	7.52 (5.83)	7.87 (6.57)	2.69 (2.34)	2.08 (1.88)	.51 (.82)	1.38 (1.65)	1.20 (1.51)	21.66 (9.47)	27.95 (10.79)	45.57 (18.46)
Male	13.73 (2.07)	2.41 (.77)	2.28 (.78)	2.34 (.75)	6.15 (5.52)	5.91 (4.57)	2.35 (2.14)	1.23 (1.41)	.44 (.79)	1.06 (1.30)	.82 (1.14)	29.18 (13.47)	31.93 (12.25)	32.92 (18.21)
Total	13.83 (2.12)	2.50 (.60)	2.44 (.65)	2.50 (.60)	6.88 (5.69)	6.96 (5.77)	2.53 (2.24)	1.68 (1.72)	.48 (.80)	1.23 (1.50)	1.03 (1.35)	25.16 (12.03)	29.80 (11.59)	39.68 (19.29)
Age	1													
PDS-S	.675**	1												
PDS-P	.666**	.820**	1											
Mean-PDS	.707**	.952**	.959**	1										
CDI T1	.37**	.129	.044	.103	1									
CDI T2	.094	.007	.033	.103	.639**	1								
CDI A-T2	.106	-.010	.031	.043	.605**	.833**	1							
CDI NM-T2	.13	.026	.093	.082	.462**	.786**	.454**	1						
CDI IP-T2	-.014	-.025	.003	-.012	.458**	.674**	.590**	.433**	1					
CDI I-T2	.086	.045	.068	.069	.497**	.770**	.612**	.471**	.416**	1				
CDI NS-T2	-.028	-.02	-.104	-.043	.314**	.636**	.292*	.552**	.295*	.319**	1			
VLF	.12	.08	-.013	.013	.201	.192	.227	.067	.18	.158	.075	1		
LF	.296*	.256*	.221	.249*	.239*	.228	.360**	.053	.217	.197	-.038	.138	1	
HF				-.302**	-.217	-.125	-.165	-.324**	-.276*	-.374**	-.111	-.241*	-.223	-.03
Sex	.041	.205	.245*	.249*	.121	.17	.076	.245*	.045	.109	.142			-.314**
Age				.329**										

PDS – Pubertal Development Scale; CDI – Child Depression Inventory; A – anhedonia, NM – negative mood; IP – interpersonal problems, I – ineffectiveness; NS – negative self-esteem; VLF – very low frequency relative spectral power HRV; LF – low frequency relative spectral power HRV; HF – high frequency relative spectral power HRV.

**
p .01.

*
p .05, uncorrected.

Table 2

One-year prospective association between total depressive symptoms and HRV.

	B	Std. Error	β	t	Sig.	R	R ²
Model 1							
(Constant)	-.108	4.995		-.022	.983	.212	.045
Age	2.258	1.425	.196	1.585	.117		
Sex	.493	.463	.181	1.064	.291		
Puberty	-1.288	1.682	-.134	-.766	.446		
Model 2							
(Constant)	6.687	5.179		1.291	.201	.407	.165
Age	3.852	1.435	.073	.447	.656		
Sex	.200	.446	.335	2.685	.009		
Puberty	-1.506	1.585	-.157	-.950	.345		
HF	-.117	.037	-.391	-3.133	.003		
β in							
Excluded							
LF	-.059	-.312	.756	-.038	.348		
VLF	.059	.373	.711	.045	.489		
Model 3							
(Constant)	10.33	4.192		2.464	.016	.687	.472
Age	-.809	.393	-.297	-2.061	.043		
Sex	1.478	1.211	.129	1.220	.227		
Puberty	1.184	1.342	.123	.882	.381		
HF	-.052	.032	-.172	-1.630	.108		
T1 CDI	.675	.108	.665	6.233	.000		

p .01 (n=73).

Table 3

One-year prospective association between HF HRV and depression subscales.

	B	Std. Error	β	t	Sig.	R	R ²
Model 1							
(Constant)	47.826	3.195		14.968	<.001 ***	.374	.14
Anhedonia	-3.215	.947	-.374	-3.395	.001 ***		
Excluded Variables			β in				
Negative mood			.074	.600	.551		
Interpersonal problems			-.031	-.229	.819		
Ineffectiveness			.009	.067	.947		
Negative self-esteem			.086	.747	.458		

Model 1 regresses CDI subscales onto HF HRV.

p .001 (n=73).