Western University Scholarship@Western

Electronic Thesis and Dissertation Repository

8-20-2018 9:30 AM

Pilot Study: Heart Rate Variability Analysis and Mental Health Outcomes in University Female Hockey Players

Kaitlyn Jacobs, The University of Western Ontario

Supervisor: Dr. J. Kevin Shoemaker, *The University of Western Ontario* Co-Supervisor: Dave Humphreys, *The University of Western Ontario* A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Kinesiology © Kaitlyn Jacobs 2018

Follow this and additional works at: https://ir.lib.uwo.ca/etd

Part of the Exercise Physiology Commons, Health Psychology Commons, Other Kinesiology Commons, and the Systems and Integrative Physiology Commons

Recommended Citation

Jacobs, Kaitlyn, "Pilot Study: Heart Rate Variability Analysis and Mental Health Outcomes in University Female Hockey Players" (2018). *Electronic Thesis and Dissertation Repository*. 5693. https://ir.lib.uwo.ca/etd/5693

This Dissertation/Thesis is brought to you for free and open access by Scholarship@Western. It has been accepted for inclusion in Electronic Thesis and Dissertation Repository by an authorized administrator of Scholarship@Western. For more information, please contact wlswadmin@uwo.ca.

Abstract

Exercise improves anxiety and depression, both of which are associated with impaired autonomic regulation of heart rate (HR). In turn, HR variability (HRV) is a reliable physiological indicator of external stressors. The following research tested the hypothesis that HRV is indicative of resilience towards mental stress in female varsity hockey players. 17 varsity hockey players (HOCK, age 21 \pm 1.5) and 15 healthy controls (CTRL, age 21 \pm 2.2) at Western University participated three times throughout a 7-month season. Participants completed questionnaires (brief resilience scale, BRS; generalized anxiety scale, GAD-7; mental health inventory, MHI; visual analog scale, VAS; short form 36-item health survey, SF-36). Five-minute measures of HRV, root mean square of successive R-R intervals (RMSSD), were calculated from NREM sleep. RMSSD improved alongside BRS, MHI, and GAD-7 scores in HOCK group, with no change in CTRL group. Results suggest heavy training improves HRV, resilience, and mental health outcomes.

Keywords: Heart Rate Variability, Mental Health, Anxiety, Resilience, Student-Athletes

ACKNOWLEDGEMENTS

If I were asked where I saw myself in five years at the beginning of my undergraduate career, I am not certain I would have said completing a Masters degree. However, I am forever grateful for the path I have chosen to take and for the endless support. These past two years have allowed me to grow tremendously as an individual and an academic. This growth would not have been possible without the enormous help given by so many people in so many different ways.

I first would like to thank Dr. Kevin Shoemaker. Your dedication and passion for research are an inspiration. You have allowed me to combine my two passions of athletics and mental health advocacy in the field of research and for that you have my deepest appreciation. The lessons I've learned about being a person, an academic, and a team member stretch much further than the walls of the lab. Thank you for being an incredible role model.

To Dave Humphreys, you have inspired me to pursue my passion of sports with a greater meaning. Your enthusiasm and dedication are what make you an outstanding professor and I have been honoured to work alongside you.

To my advisory and defense committees: Dr. Craig Hall, Dr. Lindsay Nagamatsu, and Dr. Thomas Overend. Thank you for your comments and contributions throughout the completion of this dissertation and degree.

To my family and friends who have been with me every step of the way, I express the deepest of gratitude. Mom and Dad, you have provided me with the foundation for success and this accomplishment is a testament to that. Greg, Derek, and Rachel, you are my biggest fans, from watching every hockey game to listening to me rant about my thesis. It is my hopes that you will finally understand what I have been talking about when you read this. Thank you for your support, love, and patience.

To my amazing lab mates, who I now call close friends. Thank you for your support and willingness to help. This dissertation would not at all be possible without your continued contributions. Getting to know each of you has been an absolute pleasure and I hope that as we continue on with our careers we stay connected. You all truly made my experience in and out of the lab special. Special thanks go out to Emilie Woehrle. This study could not have been completed without your enormous assistance with data collection in freezing hockey arenas and your enthusiasm and encouragement throughout this entire process. Your work ethic is truly inspirational, and I am sincerely grateful for the part you play in my life.

A special thanks to all the individuals who participated in this research. These individuals generously shared their time and experience for the purposes of this project. Without your help none of this would have been possible.

Tori, you truly are my rock and this dissertation could not have come to fruition if it weren't for your unwavering encouragement and support. Through good times, challenges, stresses or obstacles in both my academic and athletic career, you have been there. For this I am forever grateful.

This has truly been an amazing journey. I know my thanks is insurmountable to the actual amount of gratitude I feel towards every person who has played a part in this accomplishment. For that reason, I dedicate this to you.

TABLE OF CONTENTS

ABSTRACT	i
ACKNOWLEDGEMENTS	ii
TABLE OF CONTENTS	iv
LIST OF TABLES	vi
LIST OF FIGURES	vii
LIST OF APPENDICES	viii
LIST OF ABBREVIATIONS	ix
CHAPTER 1 – INTRODUCTION	1
CHAPTER 2 – LITERATURE REVIEW	
2.1 Heart Rate Variability	
2.2 Innervations of The Heart	
2.2.1 Parasympathetic (vagal) Innervation of The Heart	
2.2.2 Sympathetic Innervation of The Heart	
2.2.3 The Cardiac Cycle & Electrocardiogram (ECG)	
2.2.4 Autonomic Regulation of The Heartbeat	
2.2.5 Influence of Respiration on HRV	
2.2.6. Influences of Menstrual Cycle on HRV	
2.3 Basic Measurements of HRV Parameters and Interpretations	
2.4 Physical Activity, Mental Health, & HRV	
2.5 Application of HRV & Mental Health in The Sport Setting	
2.6 HRV and Sleep	
2.7 Physiological Demands of Hockey	
CHAPTER 3 – METHODS	
3.1 Participants	
3.2 Data Acquisition & Protocol	
3.3 Methods of Assessment	

3.4 Data Analysis	
CHAPTER 4 – RESULTS	
4.1 Baseline Anthropometrics and Overall Health	
4.2 The Influence of Menstrual Cycle on HRV	
4.3 HRV Parameters	
4.4 Mental Health Questionnaires and Indices	
4.5 Regression of Independent Variables on RMSSD	
4.6 HRV Responses Correlated with Mental Health Indices	
CHAPTER 5 – DISCUSSION	
5.1 Reliability of Firstbeat Artefact Correction	
5.2 Hemodynamics	
5.3 Time Domain of HRV	
5.4 Frequency Domain of HRV	
5.5 Autonomic Response to Stressors	59
5.6 Exercise and HRV	50
5.7 Mental Health Indices	
5.8 Contributing Factors to Resilience	61
5.9 Limitations	
5.10 Future Directions	
CHAPTER 6 – CONCLUSION	
REFERENCES	66
APPENDICES	
CURRICULUM VITAE	

LIST OF TABLES

Table 3.1: Activity classification for participants	. 27
Table 4.1: Baseline 36-Item Short Form Survey (SF-36) scores for participants as a measure of	of
overall health	. 35
Table 4.2: Baseline descriptive, heart rate, and blood pressure measures at each time	
point	36
Table 4.3: RMSSD based on menstrual phase (follicular or luteal) for all participants at T1, T2	2,
and T3	37
Table 4.4: Heart rate variability indices for both time and frequency domain at each time sessi	on
separated by hockey (HOCK) or control (CTRL) group	. 38
Table 4.5: Mental health indices at each time point for both the HOCK and CTRL group	. 42
Table 4.6: Subgroups within the Mental Health Inventory (MHI) at each time point for both th	ıe
HOCK and CTRL group	
Table 4.7: Summary of the regression model for predicting RMSSD	47
Table 4.8: Pearson correlations of RMSSD and mental health indices at each time point for	
CTRL group	. 49
Table 4.9: Pearson correlations of RMSSD and mental health indices at each time point for	
HOCK group	. 49
Table 4.10: Pearson correlations of RMSSD and mental health indices at each time point	
regardless of participant group	. 50
Table 4.11: Pooled data from both participant groups RMSSD and mental health indices for	
Pearson correlation of RMSSD and mental health indices, disregarding time points	. 50

LIST OF FIGURES

Figure 2.1: Brain Stem Nuclei	9
Figure 2.2: Autonomic innervations of the heart	10
Figure 2.3: Schematic of sympathetic and parasympathetic innervations of the heart	10
Figure 2.4: Electrical activity of myocardium represented on an electrocardiogram	11
Figure 2.5: Schematic representation of R-R intervals	15
Figure 2.6A: Artifact corrected R-R interval plotted against time	16
Figure 2.6B: Artifact corrected heart beat plotted against time	16
Figure 2.7: Representation of decomposed R-R intervals into a power spectral analysis	
Figure 3.1: Schematic representation of Firstbeat Technologies bodyguard 2 device	
demonstrating correct placement	30
Figure 4.1: Mean VAS scores calculated over the three time points for day one	43
Figure 4.2: Mean VAS scores calculated over the three time points for day two	44
Figure 4.3: Mean BRS scores for the HOCK and CTRL groups at each time point	45
Figure 4.4: Correlations for university students at time points 1-3 (columns), for RMSSD) and
BRS, RMSSD and MHI, and RMSSD and GAD-7	51
Figure 4.5: Global correlation of university student data for RMSSD and MHI	
Figure 4.6: Global correlation of university student data for RMSSD and BRS	53
Figure 4.7: Global correlation of university student data for RMSSD and GAD-7	54

LIST OF APPENDICES

APENDIX A – Supplementary HRV Data	71
Figure A.1. Global Correlation of all SDNN-HRV and Brief Resilience Scale (BRS) Scores	
pooled from all three time points and all participant data	71
Figure A.2. Global Correlation of all SDNN-HRV and Generalized Anxiety Disorder (GAD-7))
Scores pooled from all three time points and all participant data	72
Figure A.3. Global Correlation of all SDNN-HRV and Mental Health Inventory (MHI) Scores	
pooled from all three time points and all participant data	73
Table A.1. Average response for the 18 items of the MHI questionnaire over the seven-month	
protocol separated by participant group	74
Table A.2. Well-being scores (out of 100%) for each time point on Days one and two, separate	d
by group	75
APPENDIX B – Ethics Approval	76
APPENDIX C – Mental Health Instruments	77
C.1. Mental Health Inventory (MHI) Questionnaire	77
C.2. Brief Resilience Scale (BRS) Survey	79
C.3. Generalized Anxiety Disorder 7-Item Scale	
C.4. Visual Analog Scale for Anxiety	81
C.5. Short form 36-item Survey (SF-36)	82

LIST OF ABBREVIATIONS

ACH	Acetylcholine	
ANS	Autonomic Nervous System	
AP	Action Potential	
ATP	Adenosine Triphosphate	
ATP-PCR	Adenosine Triphosphate Phosphocreatine	
AV	Atrioventricular node	
BG-2	Bodyguard 2 Device (Firstbeat)	
BMI	Body Mass Index	
BP	Blood Pressure	
BRS	Brief Resilience Scale	
BRS	Baroreflex sensitivity	
CNS	Central Nervous System	
CTRL	Control group	
CVLM	Caudoventrolateral Medulla	
DBP	Diastolic Blood Pressure	
DMN	Dorsal Motor Nucleus	
ECG	Electrocardiogram	
GAD-7	Generalized anxiety 7-item Scale	
HF	High frequency	
НОСК	Hockey group	
HR	Heart Rate	
HRV	Heart Rate Variability	
LF	Low frequency	
MET	Metabolic equivalent	
MHI	Mental Health Inventory	
NAmb	Nucleus Ambiguous	
NTS	Nucleus Tractus Soltarius	
pNN50	Percentage of differences between successive R-R intervals	
PNS	Parasympathetic Nervous System	
REM	Rapid eye movement stage of sleep	
RMSSD	Root mean square of successive R-R intervals	
R-R INT	R-R interval	
RSA	Respiratory sinus arrhythmia	
RVLM	Rostroventrolateral Medulla	
SA	Sinoatrial node	
SBP	Systolic Blood Pressure	
SDNN	Standard deviation in normal R-R intervals	
SF-36	Short form 36-item survey	
SG	Sympathetic Ganglia	

SNC	Sympathetic nervous chain
SNS	Sympathetic nervous system
ТР	Total power
VAS	Visual Analog Scale
VLF	Very low frequency

CHAPTER 1 – Introduction

Mental health disorders among North American university students have become an epidemic. In 2015, 264 million individuals worldwide lived with an anxiety disorder, reflecting a total increase of 14.9% since 2005 (World Health Organization; WHO, 2017). Within the same period, depression increased 18.4% (WHO, 2017). WHO states that depression affects roughly 4.4% of the world's population, with most of these individuals simultaneously suffering from an anxiety disorder. These commonly dichotomized mental health disorders were considered the sixth leading cause of disability in 2010, in terms of Years Lived with a Disability (YLD) (Baxter et al., 2014) and were the leading cause of disability in 2017 (WHO, 2017). The National College Health Assessment (NCHA) survey found that during the fall 2016 period, 86.9% of university students reported having 'felt overwhelmed by all (they) had to do' and 61.9% reported having 'felt overwhelmed by all (they) had to do' and 61.9% reported having 'felt overwhelmed by all (they) had to do' and 61.9% reported having 'felt overwhelmed by all (they) had to do' and 61.9% reported having 'felt overwhelmed by all (they) had to do' and 61.9% reported having 'felt overwhelmed by all (they) had to do' and 61.9% reported having 'felt overwhelming anxiety'. Moreover, 34.4% of students reported that stress impacts their individual academic performance.

For the purpose of this study, stress is regarded as a state of mental or emotional strain or tension resulting from adverse or highly demanding circumstances (Oxford English Dictionary Online, 2017). Likewise, general anxiety will be defined as an averse emotional response to a perceivably threatening circumstance (Eysenck et al., 2007). It is noted that anxiety can be subdivided into state and trait. The classification of anxiety will depend on the duration and intensity with the former having a more immediate and short-term effect on an individual compared to the latter. Trait anxiety, like state anxiety, is a state in which an individual interprets a threatening situation which consequently hinders their ability to initiate a sound pattern of behaviour to avoid the imminent threat (Dimitriev, Saperova, & Dimitriev, 2016), the only difference being the intensity, duration and range of situations in which anxiety occurs. Miu,

Heilman, and Miclea (2009) found that individuals with trait anxiety were at greater risk of developing anxiety disorders and autonomic dysfunction. Resilience will be defined as the ability to "bounce back" or recover from stressful or adverse circumstances (Smith et al., 2008).

Chronic stress increases an individual's risk of acquiring cardiovascular disease (CVD), arteriosclerosis, hypertension, and other metabolic disorders (McEwan & Stellar, 1995; Lucini, Di Fede, Parati, & Pagani, 2005), whereas acute stressors consequently result in short-term increases in blood pressure, heart rate (HR), and decreased metabolic efficiency (Appelhans & Luecken, 2006; Friedman & Thayer, 1998). A meta-analysis by Forcier et al., (2006) confirmed that regular bouts of exercise can increase physiological capacity to cope with external stressors (e.g., anxiety) by attenuating cardiovascular reactivity and increasing resilience; these responses included lower HR response, lower systolic blood pressure (SBP), slightly lower diastolic blood pressure (DBP) responses, and improvements in HR recovery time (Forcier et al., 2006). These protective mechanisms manifested by physical activity lead to the notion that exercise can be prescribed as medicine (Huang, Webb, Zourbos, & Acevedo, 2013).

In 2016, Western University had a total enrollment of 28,864 students, with roughly 2,000 student-athletes (e.g., 8% of Western University's total student enrollment) participating in over 20 varsity sports in the U Sport League (Western University Facts & Figures, 2016). Student-athletes represent a unique population as they are subjected to certain external stressors not experienced by other collegiate students, including extreme time commitments (e.g., training for 10h/week minimum), pressures to achieve both academically and athletically, injuries, and burnout (Baumert et al., 2006; Cresswell & Eklund, 2007; Gustafsson, Kenttä, & Hassmén, 2011). Nonetheless, despite the added pressures of being a full-time student-athlete, most of these individuals decide not to seek help due to the stigmatization associated with poor mental health, a

lack of mental health literacy or resources, and negatively perceived experiences of seeking help in the past (Gulliver, Griffiths, & Christensen, 2012). With this discrepancy between reporting rates of mental ill-health and the climbing prevalence in university students, there is a discernible need for alternative approaches to ensure student-athletes are not only physically but also mentally fit and fully equipped to manage these heightened stressors.

Heart rate variability (HRV) represents beat-by-beat variations in the length of each cardiac cycle. These variations reflect, primarily, the dynamic interplay of the two divisions of the autonomic nervous system which regulate the instantaneous HR (Task Force, 1996; Bilchick, Ronald, & Berger, 2006). Monitoring HRV has a primary application in the clinical settings of cardiovascular health and rehabilitation. HRV provides a window to monitor autonomic responsiveness of cardiac control (Task Force, 1996). These characteristics enable efficient tracking of training and adaptation/maladaptation to physical and psychological stress. Further, HRV reflects the sufficiency of recovery between bouts of exercise or instances of high external stressors (Dong, 2016).

CHAPTER 2 – Literature Review

2.1 Heart Rate Variability

The present research evaluated the possible role of HRV as an indicator of psychological resilience in female varsity hockey players and in healthy female students at Western University. Specifically, the root mean square of successive differences in R-R intervals (RMSSD) was evaluated for changes in this population. RMSSD is a measure of HRV in the time domain as it takes into consideration the sequence of time between each R wave of the QRS complex (see Figure 2.5) seen on an ECG (R-R intervals) over short and longer time scales (e.g., ten seconds to

five minutes). There are many different techniques within the time domain of HRV which take into account longer duration temporal fragments of R-R intervals, such as the standard deviation of normal R-R intervals (SDNN). The SDNN is applied for measures of HRV lasting five minutes or longer (Spiers, Silke, McDermott, Shanks, & Harron, 1993). Previous literature has indicated that decreases in RMSSD, a reflection of vagal tone, is associated with greater mental strain and anxiety inducing situations such as exams in university students (Dimitriev, Dimitriev, Karpenko, & Saperova, 2008). Therefore, high RMSSD values should reflect increased vagal activity and good recovery from stress (Tiesala et al., 2014). Further, metrics of HRV are known to be associated with physical health, self-regulatory capacity, and adaptability or resilience (Shaffer & Ginsberg, 2017). This trend has yet to be examined in university athletes.

2.2 Innervations of the Heart

The autonomic nervous system is integrated through both the central (CNS) and peripheral nervous systems (PNS) to regulate visceral functions of the body. The CNS is comprised of the brain, brainstem, and its associated nuclei and bundles of visceral fibres in the spinal cord, while the PNS is represented by all nerve fibres and ganglia outside of the CNS (Battipaglia & Lanza, 2015). The central division receives, integrates and distributes commands through efferent (motor) nerves based on the feedback received from afferent (sensory) impulses. Motor nerves that service autonomic control are organized in a sequential neuron pathway for transmission of information: a pre-ganglionic neuron, which starts in the CNS and exists along a cranial or spinal nerve, and a post-ganglionic neuron which exists entirely outside the CNS (Tortora & Nielson, 2012; Shaffer, McCraty, & Zerr, 2014; Battipaglia & Lanza, 2015). Autonomic outflow to the heart and vasculature originates, and is primarily regulated, in the medulla, a structure found within the

brainstem. A site located within the medulla known as the nucleus tractus solitarius (NTS) receives afferent input from baroreceptors regarding environmental and physiological demands. Baroreceptors are a negative feedback system that are sensitive to changes in blood pressure (monitored by stretch in vascular walls) and are located in both the carotid sinus and the aortic arch. The NTS in turn, integrates the afferent information and distributes motor information which stimulates the appropriate cardiovascular responses in the divisions of the autonomic nervous system (ANS) (e.g., increasing/decreasing HR, blood pressures, and contractility of coronary vessels) to meet physiological demands and maintain homeostasis in the smooth muscle, cardiac muscle, and glands (Carnevali & Sgoifo, 2014). The two main divisions of the ANS include the sympathetic nervous system (SNS) and the parasympathetic (vagal) nervous system (PNS) (Triposkiadis, Karayannis, Giamouzis, Skoularigis, Louridas, & Butler, 2009). The SNS mobilises body systems and causes the 'fight or flight' response by increasing alertness and metabolic activity (e.g., increases HR, causes vasoconstriction, and increases BP), whereas the PNS conserves energy and is known for 'rest and digest' control of the body (e.g., slows HR, causes vasodilation, and decreases BP).

2.2.1 Parasympathetic (vagal) innervation of the heart

Cardiac vagal pre-ganglionic neurons generate activity through nuclei located deep within the medullary reticular formation called the dorsal motor nuclei (DMN) and the nucleus ambiguous (NAmb), as seen in Figure 2.1 (Carnevali & Sgoifo, 2014). The vagal nerve (cranial nerve X) exits the brain stem as a long pre-ganglionic efferent fibre forming a synapse with a short postganglionic fibre within the heart. Specifically, the right and left vagal nerves innervate the sinoatrial (SA) and atrioventricular (AV) nodes, respectively (Battipaglia & Lanza, 2015). The vagal efferents also innervate the atria and sparsely innervate the ventricular myocardium (Makivić et al., 2013).

2.2.2 Sympathetic innervation of the heart

Sympathetic influence on cardiac activity can be traced back to the rostral ventrolateral medulla (RVLM) (Fig. 2.1). The NTS stimulates sympathetic outflow through a network of nuclei. Specifically, projections from the NTS synapse in the caudoventrolateral medulla (CVLM) (Ross, Ruggiero, & Reis, 1985). These neurons project inhibitory signals to the RVLM, the source of efferent sympathetic nerve activity that descend the spinal cord. These nerves synapse with 'short' pre-ganglionic neurons in the thoracolumbar spinal cord which travel to, and synapse within, the sympathetic nerve chain (SNC) or sympathetic ganglia (SG) that run parallel to the spinal cord on either side of the anterior face of vertebral bodies (see Figures 2.1 & 2.2) (Battipaglia & Lanza, 2015). Post-ganglionic efferent fibres in the paravertebral cervical and thoracic SG give origin to cardiac cervical nerves (superior, medium, and inferior), and cardiac thoracic nerves (1°, 2°, 3°, and 4°). These in turn travel to the heart and vascular tissue where they synapse at their target sites: sinoatrial (SA) node, atrioventricular (AV) node, atria, and ventricles (see Figures 2.1, 2.2, and 2.3).

2.2.3 The cardiac cycle & the electrocardiogram (ECG)

The SA and AV nodes (seen in Figure 2.2 & 2.3) are critical to optimal function of the heart as they are internal pacemakers (e.g., responsible for intitiating the heartbeat). The electrical conduction and contractile actions of these two nodes are what is recognized on an electrocardiogram (ECG) (see Figure 2.4). The SA node intitates each cardiac cycle through

spontaneous depolarization of its autorhythmic fibres, resulting in an electrical impulse (action potentials; AP) that travels from the atria to the AV node, subsequently leading to its depolarization. The depolarization of both the SA and AV nodes results in muscular contraction of the atria, known as atrial systole (see Figure 2.4). Following in sequence, the depolarization flows through the heart muscles of the atria to the top of the septum (which separates the heart into left and right) where the bundle of His sends the AP down the septum and into the ventricles (see Figure 2.4). The depolarization of the septum is the beginning of the QRS complex representing the Q-wave. Depolarization reaches the bottom of the myocardium where the Purkinje fibres distribute the AP evenly across the ventricles for a synchronized contraction (representing the R-wave). Ventricular contraction (ventricular systole) is represented by the QRS complex on the ECG, proceeded by the repolarization and relaxation of the ventricles (ventricular diastole), represented by the S-T-segment (Tortora & Nielson, 2012; Shaffer, McCraty, & Zerr, 2014).

2.2.4 Autonomic regulation of the heartbeat

The variation seen in the time between each heartbeat reflects the perpetual ebb and flow of sympathetic and vagal nerve activity on the SA node. Even in normal ECG recordings from healthy individuals recorded under resting conditions, there are temporal variations in R-wave initiation (R-R intervals; Figure 2.5). The average resting heart rate of a healthy individual is 70 bpm, but can be as low as 45 bpm in a highly trained athlete (Tortora & Nielson, 2012). The intrinsic firing rate of the SA node is 100 bpm meaning that, at rest, vaga activity must dominate sympathetic activity in order to keep the HR low. This constant input from the PNS to the heart's pacemaker is known as vagal tone (Levy, 1984) and with training, the vagal tone can increase. The PNS slows down the firing rate of the SA node by causing the release of the neurotransmitter aceytlcholine (Ach) which has a rapid inhibitory impact on calcium ion (Ca^{2+}) channels. Calcium ions are critical in many physiologic processes such as depolarization of tissues, conduction of APs, and muscle contraction (Tortora & Nielson, 2012). Therefore, vagal activation results in fewer Ca^{2+} ions flowing into the cardiac contractile cells indirectly causing less contractability, and a slower HR. The SNS, however, speeds up the firing rate of the SA node by causing the release of the neurotransmitter norephinephrine (NE), and the hormone epinephrine from the adrenal glands. Norepinephrine opens Ca^{2+} channels allowing a greater number of ions to flow into the cardiac contractile cells causing an increasing in contractibility and a faster bpm (Tortora & Nielson, 2012). The two branches of the ANS work competitively in regulating HR. In order for HR to increase there must be a reciprocal change in the sympathovagal balance, meaning there must be a withdrawal of the predominant vagal tone and an increase of sympathetic nerve activity on the SA node (e.g., in times of external stressor such as physical activity or mental stress).

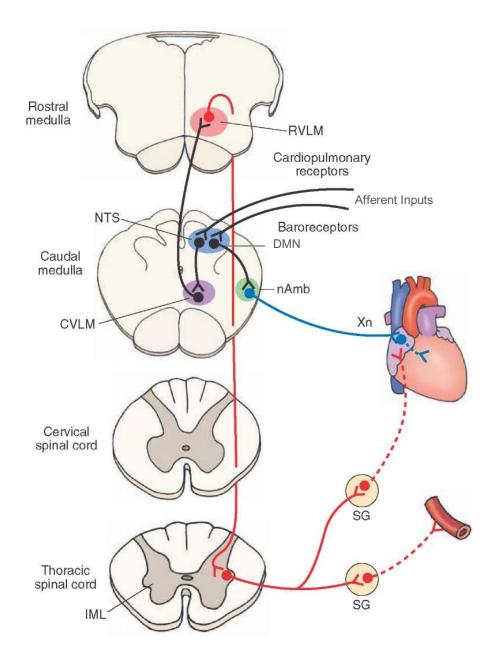


Figure 2.1. Brainstem nuclei – Simplification of key nuclei located within the medulla, such as the nucleus tractus solitarius (NTS), nucleus ambiguous (nAmb), rostroventrolateral medulla (RVLM), Dorsal Motor Nucleus (DMN), caudoventrolateral medulla (CVLM), vagus nerve (Xn), Sympathetic ganglia (SG), and intermediolateral cell column of the spinal cord (IML). Diagram shows their orientation and the interaction of their pathways within the spinal cord.

Image has been modified from: http://what-when-how.com/neuroscience/the-autonomicnervous-system-integrative-systems-part-5

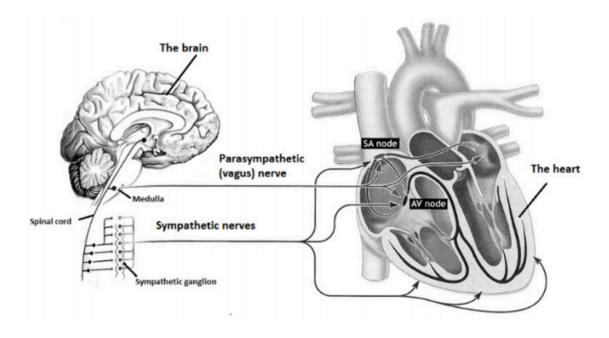


Figure 2.2. Autonomic Innervations of the heart. Schematic representation of the cardiorespiratory centers in the brain stem (medulla) and the innervations of the heart and associated cardiac vasculature. (Firstbeat Sports Technology, Stress and Recovery Analysis Method Based on 24-hour HRV)

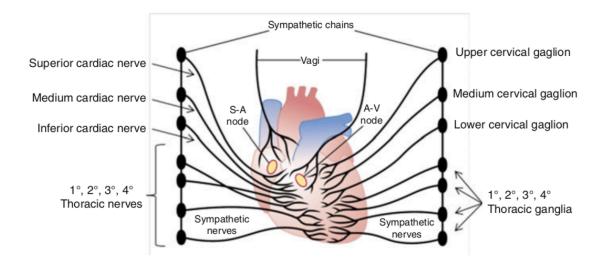


Figure 2.3. Schematic of sympathetic innervations of the heart. Displaying the superior, medium, and inferior cardiac nerves, as well as, the cardiac thoracic ganglia (Battiplagia & Lanza, 2015).

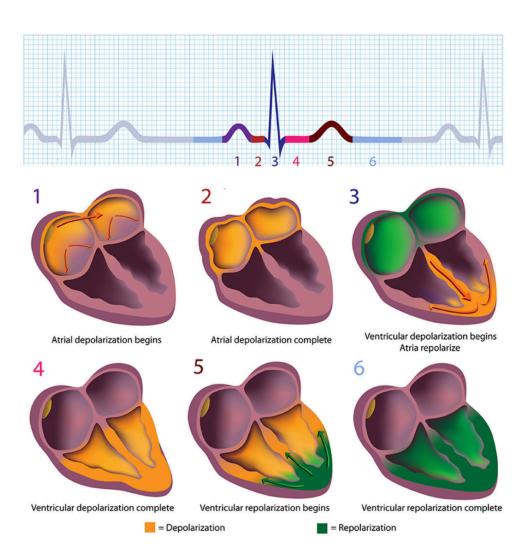


Figure 2.4. Electrical activity of the myocardium represented on an electrocardiogram. Displays how the heart conductions action potentials through the myocardium and its resultant influence on ECG recordings (Shaffer et al., 2014).

2.2.5 Influence of respiration on HRV

The interface of sympathetic and parasympathetic nerve activity on cardiac activity is perpetually confounded by the mechanistic influence of respiration, referred to as the respiratory sinus arrhythmia (RSA) (Billman, 2013). Cardiac acceleration and deceleration mimics inhalation and exhalation, respectively (Dong, 2016). This occurs through two mechanisms: (1) direct projections from the central respiratory pattern generator, and (2) indirectly through ascending afferents from lung stretch receptors that are activated during inspiration (Carnevali & Sgoifo, 2014). This afferent input from lung stretch receptors is received by the NTS and results in the inhibition of vagal motorneurons in the NAmb (see Figure 2.1). The RSA is represented by rhythmic oscillations in HR mimicking inhalation and exhalation. HR will increase during inspiration due to the momentary suppression of vagal influence. During expiration, HR will decrease with the return of vagal influence (Carnevali & Sgoifo, 2014).

2.2.6. Influences of Menstrual Cycle on HRV

Most women, with the exception of those on contraceptives, experience cyclical changes in hormones which correspond to the two phases of the ovarian cycle: the follicular phase (days 1 to 14) and the luteal phase (days 14 to 28) (Tortora & Nielson, 2012). The two key hormones in menstruation are estrogen and progesterone. Estrogen is known to progressively increase during the follicular phase, whereas progesterone remains low throughout (Tortora & Nielson, 2012; Brar, Singh, & Kumar, 2015). The luteal phase is dominated by estrogen and progesterone. One study found that women in the secretory, or luteal phase, display a significantly higher mean RR and mean HR, as well as a significantly lower RMSSD, SDNN, and pNN50 (Brar, Singh, & Kumar, 2015; Yazar & Yazici, 2016). Therefore, menstrual cycle and related hormones are potential modifiers of HRV observations and have the potential to alter the magnitude of sympathovagal balance. Future studies are required to determine the specificity of their influence.

2.3 Basic measurement of HRV parameters and interpretations

Measuring HRV proves to be a viable, inexpensive, and non-invasive tool to evaluate the intricacies of the autonomic nervous system (Task Force, 1996; Dong, 2016). There are several ways in which HRV may be calculated and analyzed from a series of inter-beat intervals (R-R intervals) taken from either an electrocardiogram (ECG) or technology capable of monitoring HR and detecting R-R intervals (e.g., Firstbeat Bodyguard 2 device). Once the R-R intervals have been obtained and examined for necessary artefact correction, the data can be formatted and partitioned into three different classes of HRV analyses; time-domain, frequency-domain, and non-linear indices. The most frequently used technique to evaluate HRV examines the time-domain (Task Force, 1996). This method examines fluctuations of normal R-R intervals over time, as depicted in Figures 2.5 and 2.6a. Multiple indices exist within the time domain approach to analyze R-R intervals such as, standard deviation of normal to normal R-R intervals (SDNN), root mean square of successive differences in R-R intervals (RMSSD), and the percentage of differences between successive R-R intervals over 24-hours that are greater than 50ms (pNN50) (Kleiger, Stein, & Bigger, 2005; Bilchick & Berger, 2006). The RMSSD is computed by selecting each successive time difference between heart beats (R-waves) in milliseconds (ms). Then, each value is squared, and the result is averaged before the square-root of the total is obtained (Shaffer, McCraty, & Zerr, 2014). When analyzing short-term R-R interval data (e.g., 5 minutes or under), RMSSD shows to be the most accurate and preferred method when compared to its time-domain counterparts (Munoz et al., 2015; Bilchick & Berger 2006) as it is an established measure of HRV due to its reliability, reproducibility and functionality in short-term settings (Pitzalis et al., 1996). It is also sensitive to high-frequency fluctuations in the respiratory range, effectively capturing the RSA, as well as lower frequency fluctuations for a versatile measure. RMSSD is a reliable index of cardiac parasympathetic influences and is recommended as a measure of vagally-mediated HRV for its simple interpretation (Task Force Guidelines, 1996; Shaffer, McCraty, & Zerr, 2014). For the aforementioned reasons, RMSSD was selected as the primary measure of HRV for this study. Accurately quantifying HRV provides a deeper understanding of an individuals' capacity for adaptation and resilience to internal and external stressors (Teisala et al., 2014).

Another parameter for analyzing HRV uses spectral analysis of R-R intervals to produce the frequencies, or indices in the frequency domain. Typically using Fast Fourier Transform (FFT) analysis, this technique probes series of numbers (along the time axis) from the ECG data, and quantifies the variation occurring at different frequencies, expressed in Hertz (Hz) or oscillations per second (Kleiger, Stein, & Bigger, 2005; Appelhans & Luecken, 2006). These numbers are computed to yield a total power spectrum which produces three distinct bands representing the frequencies that exist within this domain: very low frequency (VLF) band (<0.04Hz), low frequency (LF) band (0.04-0.15Hz), and high frequency (HF) band (0.15-0.40Hz), as seen in Figure 2.7. The quantification of this variability in HR caused by the dynamic interplay of SNS and PNS provides a window to observe the state and integrity of the autonomic nervous system and requires greater than three minutes of steady-state R-R interval data (Bilchick, et al., 2006). Monitoring HR response to external stressors (e.g., physical activity, emotional strain) provides the most informative parameter of changes in the functional state of the body (Dimitriev et al., 2008). Therefore, monitoring the body's stress reaction and its influence on the cardiac system can reveal adaptive responses and an overall capacity for modulation of the ANS.

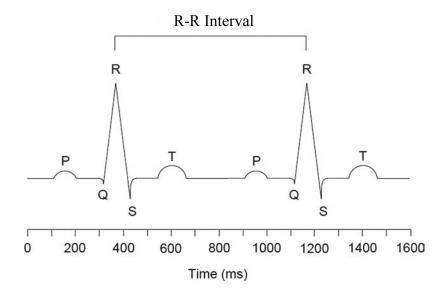
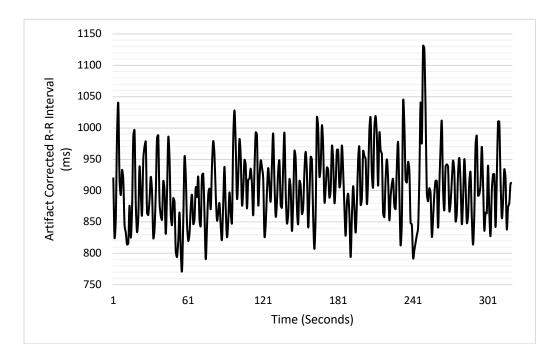


Figure 2.5. Schematic representation of one R-R interval showing the time (ms) between consecutive heartbeats (R-R intervals) which exists due to the dynamic interplay of sympathovagal activity on the internal pacemaker of the cardiac tissue known as the sinoatrial node.

Modified from Appelhans & Luecken, 2006.



В.

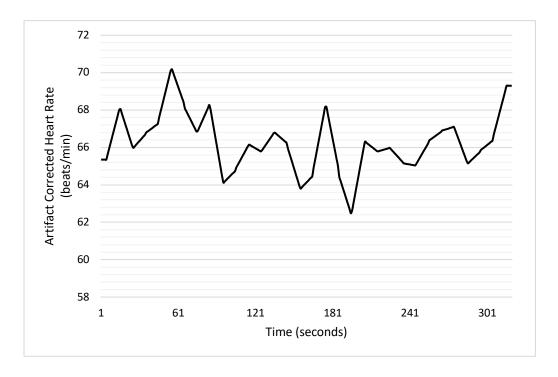


Figure 2.6. A. Displays the artifact corrected R-R intervals (ms) computed from Firstbeat technologies presented in the time-domain over a 301 second (or five-minute) measure of sleep. **B**. Shows the artifact corrected heart beat (beats/min) over a period of 301 seconds (or five minutes). There is an observable inverse relationship between the R-R interval and the HR.

Both graphs are series of raw HR data obtained from a Firstbeat recording.

Α.

The VLF and LF bands have controversially been associated with the complex interplay of both sympathetic and parasympathetic activity (Pumprla, Howorka, Groves, Chester, & Nolan, 2002), whereas the HF band is suggested to be solely influenced by parasympathetic activity due to respiratory sinus arrhythmia (Appelhans & Luecken, 2006). The respiratory sinus arrhythmia reflects the rhythmic oscillation of HR resulting from inhaling a breath which temporarily negates parasympathetic influence on HR causing it to increase, and exhaling a breath which reinstates parasympathetic influence which reduces HR. Due to the attributable ambiguity of the LF component, researchers will report the ratio of LF to HF power (LF/HF ratio) as an index of sympathovagal balance of cardiovascular autonomic regulation, where increases in LF/HF ratio are suggested to reflect a shift to sympathetic dominance, and decreases in this index correspond to parasympathetic dominance (Bilchick, Ronald, & Berger, 2006; Billman, 2013). This theory relies on the assumption that physiological interventions always elicit reciprocal changes in parasympathetic and sympathetic nerve activity (Berntson et al., 1997; Eckberg, 1997; Parati et al., 2006; Billman, 2009, 2011). However, the interface of sympathetic and parasympathetic nerve activity is perpetually confounded by the RSA (Billman, 2013). For this reason, the LF/HF ratio values are not reported in this study and were not investigated or analysed. Instead, total power and the respective percentages of VLF, LF, and HF are reported.

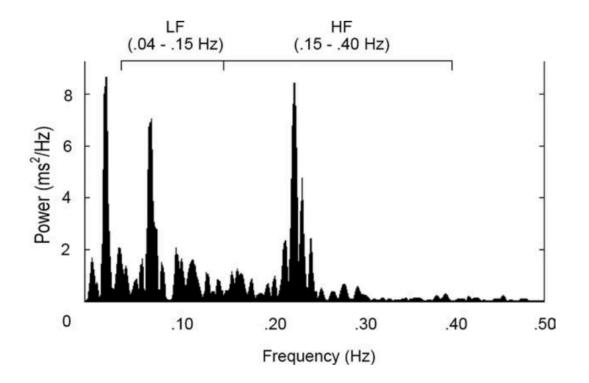


Figure 2.7. A representation of heart rate variability power spectrum computed via the fast Fourier transform (FTT) on a 5-min recording obtained from a resting subject in supine position. The low-frequency (LF) component occurs between .04 and .15 Hz and the high-frequency (HF) component occurs between .15 and .40 Hz. Hz cycles per second (Appelhans & Luecken, 2006).

2.4 Physical activity, mental health & HRV

A recent 2017 study states that mental illness is expected to account for roughly 15% of the global burden of disease by 2020 (Biddle & Asare, 2017). Anxiety, depression, and stress are all known to negatively influence the cardiac autonomic nervous system's vagal withdrawal and subsequent sympathetic activation (Friedman, 1998; Schwartz et al., 2003). Investigations into alternative interventions to pharmaceutical approaches are required to attenuate the increasing prevalence of mental illness among adolescents and young adults. Numerous studies state that exercise has equivocal, if not superior, remedial benefits to pharmaceuticals by mitigating feelings of pain, anxiety, workplace stress, depressive symptoms, and improving both physical and mental health outcomes (Dimitriev et al., 2008; Chu et al., 2014; Hearing et al., 2016; Biddle & Aare, 2017).

In direct juxtaposition to exercise and physical activity, research on sedentary behaviour has revealed a strong negative correlation with quality of mental health and prevalence of mental ill-health symptoms (Carney et al., 2017; Griffiths et al., 2014; Lachytova et al., 2017). Literature examining adolescents' self-rated health via the 36-item Short Form Health Survey (SF-36) found that an individual's self-rated health strongly correlates with mental ill-health (Lachytova et al., 2017). Moreover, the study found that physical activity and sedentary behaviour are crucial factors associated to one's self-rated health. Physical activity and mental health display an inverse dose-response relationship further supporting the protective mechanisms of exercise on the body as a whole (Griffiths et al., 2014).

Of particular interest is the potential modifying factor of sex on adaptation of physical fitness and mental health. For example, women who reported increases in physical activity by more than 2 metabolic equivalent (MET) hours per week demonstrated a reduced risk of later mental ill-health compared to their sedentary female counterparts (Griffiths et al., 2014). However, whether these responses differ from males is not clear. Specifically, a study examining the influence of trait anxiety and perceived stress on HRV in physically fit men and women discovered an inverse relationship, suggesting a lower vagal mediated cardiac response to stressful situations (Dishman et al., 2000). This relationship was independent of age, sex, physical fitness, and trait anxiety. Yet, a study of competitive collegiate student athletes found that the prevalence and risk factors associated with developing symptoms of depression are 21% (N = 257; 167 males, 90 females) (Yang et al., 2007) and that female athletes, as well as freshmen athletes, were more likely

to experience symptoms of depression with 1.32 greater odds (95% CI, 1.01 to 1.73) and 3.27 greater odds (95% CI, 1.63 to 6.59), respectively. Students competing at the collegiate level experience intense mental and physical demands that are unique to the student athlete population and have the potential to raise their susceptibility to mental ill-health and/or risk-taking behaviours (Hughes & Leavey, 2012).

2.5 Application of HRV & Mental health in the Sport Setting

In sport competition, HRV is largely understudied, especially in the moments prior to competition (D'Ascenzi et al., 2014). Current research regarding HRV in sport training has established that during physical and/or mental states of stress (i.e., exercise or anxiety) HRV will be significantly reduced in comparison to baseline measures, suggesting an altered autonomic state favouring the sympathetic reflex (Baurmet, et al., 2006; Morales, et al., 2012; Dimitrev, et al., 2016). HR and HRV have an inverse relationship, inferring that when the ANS experiences excitation, the SNS will be activated and HR will increase, thus decreasing the variability between subsequent heart beats.

An examination of state anxiety and RMSSD in a competitive swimming club during training and competition found that self-rated levels of anxiety significantly increased from training to competitive conditions as seen through the Competitive State Anxiety Inventory 2 (CSAI-2). Further, RMSSD was significantly reduced suggesting increased sympathetic dominance as a result of vagal withdrawal in response to stress (Cervantes Blásquez, Rodas Font, & Capdevila Ortís, 2009). RMSSD was measured during a 2-week track-and-field training camp that included triathlon athletes and found that HRV is significantly reduced during this period accompanied by a decline in baroreflex sensitivity (BRS) (Baumert et al., 2006). BRS modulates the intensity of baroreceptor feedback to the CNS. These findings suggest that during periods of training there is altered ANS activity favoring sympathetic nerve activity and a reduction of vagal influence on cardiac function. In contrast, a study monitoring HRV in elite female volleyball players showed only a slight change in HRV, with no pronounced variation in ANS activity prior to a decisive competition (D'Ascenzi et al., 2014).

Sport psychology often focuses on competition-induced stress, and less often on the stressors experienced outside of direct competition. An early study in the field of athlete's mental health examined the psychosocial stressors that go beyond an athlete's heavy training regime, (e.g., external variables that address the environment, the personality of the athlete and the impact of stressful life events) and found that demanding life events such as school or work, financial problems, dysfunctional relationships and social conflict all influence an athlete's training tolerance (Miller, Vaughn, & Miller, 1990). These stressors may be a valid conduit to increase an individual's risk of underperformance and autonomic maladaptation. Athlete burnout is at the forefront of sport research and is defined as a psychophysiological syndrome characterized by emotional and physical exhaustion, as well as a reduced sense of accomplishment, accompanied by distress and sport devaluation (Gustofsson, Kenttä, & Hassmén, 2011). Research that examines the entirety of the sporting experience, has identified that persistently having to perform well, fear of failure, balancing sport and school commitments, lack of feedback from coaches, and having an overall reduced sense of autonomy, are all antecedent to mental ill-health and athlete burnout (Noblet & Gifford, 2002). Therefore, monitoring HRV and indices of mental health may provide useful markers of maladaptation during training which enable early detection of overtraining syndrome in young athletes (Baumert et al., 2006).

2.6 HRV and Sleep

Rest and recovery from psychological and physiological stress occurs most commonly during sleep (Hynynen et al., 2006). Autonomic balance during rapid eye movement (REM) sleep has comparable SNS activity to that of wakefulness (Trinder et al., 2001) and is characterized by rapid eye movements, dreaming, bodily movement, and faster HR and respiration. In comparison, non-rapid eye movement (NREM) sleep in humans appears to be associated with an increase in cardiac vagal activity (Burgess et al., 1997). This period of sleep offers a window to monitor resting baseline measures, exempt of any environmental influences.

Perceived external stressors (i.e., anxiety) are associated with a higher resting HR and a resultant lower HRV during the waking period. The influence of stress extends into nocturnal sleep, resulting in a heightened state of arousal, restlessness, and perceived poor quality of sleep (Brosschot et al., 2007; Rice, Purcell, De Silva, Mawren, McGorry, & Parker, 2016). Individuals who experience greater reductions in HRV due to increases in worry or anxiety have larger increases in stress-related sleep disturbances over time, compared to their less anxious counterparts who showed little changes in HRV (MacNeil et al., 2007). Stress-related thoughts and negative affect influence an individual's subjective perceptions of their quality of sleep and have a negative impact on HRV during sleep (Hall et al., 2004). Consequently, recovery and energy levels are therefore impacted as well. In the current study, negative affect, a personality trait counter to positive affect, will be conceptualized as an individual experiencing negative emotions, such as anger and nervousness, and poor self-concept (Watson & Clark, 1984) which will be measured using the positive affect (MHP) subscale of the mental health inventory (MHI). Low scores in MHP suggest high negative affect.

The HRV response which occurs during the awakening period may be influenced by athletic stress (e.g., training) leading to decreased variability (Hynynen et al., 2011). Monitoring HRV during the awakening period (between 07:00h and 08:00h) in elite female volleyball players during play-offs revealed that, contrary to Hynynen et al., (2011), athletes' HRV showed no significant decline in variation of mean RR interval, RMSSD and pNN50 during the awakening period (D'Ascenzi et al., 2014). In addition, Myllymäki, Rusko, and Syväoja (2011) found that moderately active men showed elevations in nocturnal HR with increased exercise intensity without changes in HRV. These discrepancies in HRV could be explained by the level of perceived stress by the individuals rather than the impact of training and physiologic adaptation.

Literature on mental health (e.g., external stress, worry, and anxiety) and HRV remains limited, as no concrete conclusions have been drawn regarding this relationship. It is unknown whether higher subjective stress is directly associated with lower objective recovery during sleep as measured through HR. However, research suggests that daily emotions at work are directly related with HRV measures during sleep (Kageyama et al., 1988). Opportunity to investigate the potential association between subjective stress and resting HRV during the sleep period should be examined in future research.

2.7 Physiological demands of hockey

Participating in a varsity sport at the university level requires above average skill and a high level of physical fitness. Varsity hockey in particular, requires its athletes to uphold highly developed aerobic and anaerobic capacities, as well as sustaining a greater overall body strength, power, agility and flexibility (Twist & Rhodes, 1993; Burr, Baker, Macpherson, Gledhill, & McGuire, 2008). The game of hockey consists of intermittent short-duration maximal power

outputs (30 to 60 seconds) referred to as "shifts" (Burr, Baker, Macpherson, Gledhill, & McGuire, 2008). These bursts require sufficient bioenergetics to supplement the rapid muscle contractions and respiratory demand. The primary source of energy for short duration high intensity exercise is adenosine triphosphate-phosphocreatine, also known as the ATP-PCr system, responsible for breaking down and using ATP anaerobically (Green, Bishop, Houston, McKillop, Norman, & Stothart, 1976; Twist & Rhodes, 1993; Green, Pivarnik, Carrier, & Womack, 2006). This energy reserve dominates energy provision up to 15 seconds in an all-out maximal effort performance, and thus, a second energy system is required to continue optimal performance during a hockey shift. Anaerobic glycolysis begins to dominate the energy supply roughly 10 to 12 seconds after exercise begins, to supply ATP to the body's working muscles, therefore making it the main source of energy for a single shift (Summanen & Westerlund, 2001). During only moderate intensity exercise, the aerobic system contributes approximately 60 to 70 percent of the body's energy requirements. This is subject to change depending on variations between games as well as changes throughout the game in intensity, level of competition, position and the player's level of conditioning (Rhodes & Twist, 1990). NHL players have an average aerobic capacity (maximum oxygen consumption; VO₂max) of 54.6 to 59.2 ml/kg/min (Montgomery, 2006), compared to the average adult male (20-29 years) with a capacity of 43 to 52 and adult female of 33 to 42 ml/kg/min (Wilmore & Costill, 2005). This aerobic capacity is necessary to support recovery between shifts.

The average working HR for hockey players varies between 170 and 174 bpm with a recovery time of 3 to 5 minutes between shifts, where the HR falls back down to roughly 110 to 120 bpm (Summanen & Westerlund, 2001). On average, these athletes reach 87-92% of their maximal HR value computed during their baseline maximal oxygen consumption (VO₂ max) test. On-ice performance of varsity male hockey players at the University of Waterloo found that the average

total playing time, regardless of position, was 24.5 minutes during which the athlete covered an average of 5553m on the ice surface (Green, Bishop, Houston, McKillop, Normal, & Stothart, 1976). According to WHO, recommended levels of activity for the average adult (aged 18-64) over the course of a week is 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity exercise (WHO, 2018 – accessed online). Including practices (roughly 4 to 5 times a week), training (2 to 6 hours) and games (2 per week), elite hockey players surpass this recommendation two-fold. Hockey has evolved and developed into a faster paced game, only increasing the emphasis of physical fitness and aerobic development. Between 1992 and 2003, aerobic fitness of ice hockey players was found to increase from 54.6 to 59.2 ml/kg/min (Montgomery, 2006). Participating in the sport of hockey at elite levels will evidently result in adaptations to fitness levels which will not only impact performance, but the functionality and efficiency of the cardiovascular system and energy usage. It is possible that these adaptations in fitness level act as a substrate in mental resilience. In fact, Skirka (2000) reported that varsity athletes display higher levels of 'hardiness' (one's ability to endure difficult situations) and coherence and lower perceived stress than their nonathlete counterparts. Deuster and Silverman (2013) found a positive association between aerobic capacity and hardiness. This supports the association of physical fitness and one's ability to bounce back or come through difficult times.

The aim of this study was to evaluate and interpret subjective measures of mental health indices (e.g., state and trait anxiety, stress, depression, perceived behavioural control, and positive affect), as well as measures of mental resilience, and directly compare the impact of these parameters on HRV. Therefore, the present study tested the hypothesis that an increase in mental health resilience to external stressors among varsity athletes would be accompanied by an increase in RMSSD, reflected as a positive correlation between resilience and HRV. An objective marker of mental ill-

health would allow for the identification of at-risk individuals, provide greater insight for addressing mental health standards in sport, and allow for better management of mental ill-health individuals in university sports.

CHAPTER 3 – Methods

3.1 Participants

21 healthy elite female hockey players (HOCK) aged 17-23 years of age ($M_{age} = 21$, SD = 1.5), and 15 healthy female students (CTRL) aged 18-25 years of age ($M_{age} = 21$, SD = 2.2), volunteered and were enrolled in this study. All participants in the HOCK group were members of the Women's varsity hockey team at Western University, playing in the U Sports league and enrolled in full-time studies. All control participants were enrolled in full time studies at Western University and were not a part of a varsity team or league, nor did they play any elite controlled sports. The built-in fitness classification by Firstbeat SPORTS software (Figure 3.1) quantified the participant's fitness level. CTRL participants were moderately active (e.g., engaged in regular to light exercise training), exercising roughly one to three times a week for no longer than 45 minutes (e.g., activity classification of 3 to 4). HOCK participants were considered extremely active (e.g., training daily) exercising roughly 7 to 13 hours per week (e.g., an activity classification of 8.5 to 9). The fitness classification is based on a 10-point scale which is determined by training frequency and duration per week over the last two to three months.

Typical Training Description	Typical Training Frequency	Time Spent in Week Training	Activity Class		
No exercise	-	-	0		
	Once every two weeks	Less than 15min	1		
Light exercise occasionally	Once every two weeks	Less than 30min	2		
	Once a week	~ 30min	3		
		~45min	4		
	2 to 3 times a week	45min to 1hr	Training Activity class - 0 Less than 15min 1 Less than 30min 2 ~ 30min 3 ~ 45min 4 45min to 1hr 5 1 to 2hr 6 3 to 5hr 7 5 to 7hr 7.5 7 to 9hr 8 9 to 11hr 8.5 11 to 13hr 9 13 to 15hr 9.5		
Regular training		1 to 2hr	6		
	3 to 5 times a week	3 to 5hr	6 7		
	5 to 5 times a week	5 to 7hr	7.5		
	A lucast deiler	7 to 9hr	8		
	Almost daily	9 to 11hr	8.5		
Training daily		11 to 13hr	9		
	Daily	13 to 15hr	9.5		
		More than 15hr	10		

Table 3.1. Activity classification according to Firstbeat SPORTS software. The activity class is selected based on which best describes the general level of training (aerobic training and heavy physical workload) during the past two to three months.

Source: modified from Firstbeat SPORTS software.

This study was conducted over the duration of a two-semester hockey season (seven months total ranging from September to March). Anthropometric measurements were obtained upon participants' first session in a lab setting. Both the CTRL and HOCK group completed three sessions throughout their academic year (referred to as T1, T2, and T3). The final data analysis included 17 HOCK participants who met the study's inclusion criteria. The inclusion criteria for the HOCK group in this study was participating on the women's varsity hockey team, playing in the games at all three time points, having complete data for HRV analysis as well as complete questionnaire data for all three time points and acquired no injuries during the season. Participants

missing HRV or questionnaire data were excluded from the final analysis, as well as individuals who sustained injuries during the season. The inclusion criteria for participants in the CTRL group required healthy full-time students who were not participating in any type of competitive sport, nor did they do any vigorous or strenuous exercise. All 15 CTRL participants met the inclusion criteria of HRV data and questionnaires. This research was conducted in accordance with the standards of the Declaration of Helsinki for medical research involving human subjects and was approved by the Western University Research Ethics Board (REB) and all participants provided informed consent.

3.2 Data Acquisition & Protocol

Each of the 32 participants were required to wear a Firstbeat Bodyguard 2 device (BG2; Firstbeat Technologies Ltd, Jyväskylä, Finland) during each of the three time points (beginning, mid, and end of season). This was done to build an account of participants' experiences at different points throughout the academic year which, in the case of the HOCK group, coincided with their competitive season. For each time point, the BG2 device was worn for a total of 24 to 48 hours in order to record and evaluate an accurate representation of heart rate and HRV at baseline and daily living.

The BG2 is a small transportable device that connects to the participant by clipping onto two single use, adhesive electrodes; one placed below the right collar bone and the other electrode on the left side midway down the participant's rib cage (Fig. 3.1) (Firstbeat Bodyguard 2 Guide, 2017). Once the device is attached, the measurement of heart beat data begins automatically, as confirmed by a flashing green light to show good connectivity. The BG2 device is equipped with a built-in USB device for easy connection and off-loading of R-R interval data into the Firstbeat SPORTS software (version 4.7.3.1; Firstbeat Technologies Ltd). The Firstbeat SPORTS software is designed to analyse HRV data and provide comprehensive physiologic measures.

In the case of the HOCK group, the length of time was chosen to encompass a day of rest (pre-game day) and an entire game day (second day). In addition, this approach allowed for the capture of two solid measures of sleep (e.g., before and after the game). The baseline day was included to ensure a sleep measure exclusive of any prior strenuous physical or mental activity. For the HOCK group, the BG2 device was administered one day prior to the game, at roughly 18:00h. The protocol for the CTRL group was similar in that the BG2 device was worn for 24-48 hours, but it differed in the sense that there was no "active" day, but rather two "control" or "baseline" days. The CTRL group was allowed to exercise but not to the extent that it became strenuous intense exercise.

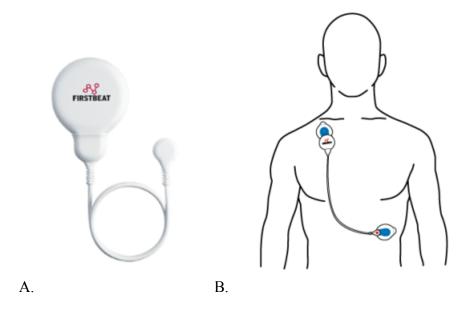


Figure 3.1. Schematic placement of Firstbeat Technologies demonstrates the correct placement of electrodes and subsequent attachments for the Firstbeat Bodyguard 2 device. One electrode is placed under the participants' right clavicle and the other midway down the left side of the ribs (Firstbeat Bodyguard 2 Guide).

To evaluate HRV, the root-mean-square of successive differences (RMSSD) was evaluated during sleep. Specifically, a 5-minute recording of steady state sleep was taken 30 minutes after the participant recorded sleep onset. This was done in order to account for the sleep onset latency (SOL) period, defined as the length of time it takes to accomplish the transition from full wakefulness to the lightest non-REM (NREM) sleep stage (Hall et al., 2004). This provides a stable and repeatable condition in which the influence of all external factors is kept to a minimum.

3.3 Methods of Assessment

Although objective physiological measures play a significant role in behavioural assessments, subjective measures provide supplementary alternative means to assess psychological functioning. The following questionnaires were employed on the first day of each data collection time point to get a measure of participants' mental states: The Generalized Anxiety Disorder 7-item (GAD-7) Scale, Visual Analog Scale for Anxiety (VAS), Brief Resilience Scale (BRS), Mental Health Inventory (MHI), and 36-item short form (SF-36) questionnaire (see Appendix C), as well as a general sleep log to track quality and duration of sleep.

The VAS was also used on the second day for both groups, specifically for the purpose of gaining a sense of the HOCK participants' precompetitive states of anxiety. The VAS requires participants to record their current state of general anxiety on a visual scale scored from 0-100 (in milimetres). A score of 0 on the scale being "not anxious at all," and a score of 100 being "extremely anxious" (Abend, Dan, Maoz, Raz & Bar-Haim, 2014).

The GAD-7 evaluates an individual's general anxiety relating to the past 4 weeks and is assessed by assigning scores of 0, 1, 2, or 3, to the response categories of 'not at all', 'several days', 'more than half the days', and 'nearly every day', respectively. A sum of the scores for each of the seven items computes a total score. The "bench marks" for mild, moderate, and severe anxiety are 5-9, 10-14, and 15-21, respectively (Kertz, Bigda-Peyton & Bjorgvinsson, 2013).

The brief resilience scale (BRS) defines resilience as "the capacity to recover quickly from difficulties; toughness" (Smith et al., 2008). The BRS is comprised of six items, each rated on the participants' level of agreement (e.g., 'strongly disagree', 'disagree', 'neutral', 'agree', and 'strongly agree') to each of the six statements in the questionnaire and corresponds to a numerical value of 1, 2, 3, 4 or 5, respectively. Items 1, 3, and 5 of the questionnaire are positively worded,

32

and items 2, 4, and 6 are negatively worded. Therefore, the BRS is scored by reverse coding items 2, 4, and 6 and finding the mean of the six items. Total BRS scores range from 1-6: the higher the score the more resilient the individual (Smith et al., 2008).

The MHI entails 18 questions all evaluated on a 6-point Likert-scale response. The MHI provides a method for evaluating various mental health indices by partitioning each of the 18 items into four subgroups: anxiety (MHA), depression (MHD), behavioural control (MHC), positive effect (MHP), and general distress (overall emotional functioning; total MHI score). MHA is calculated by taking items 4, 6, 10, 11, and 18, which correspond to the following questions: 'have you been a very nervous person', 'have you felt tense or high-strung', 'were you able to relax without difficulty', 'have you felt restless, fidgety, or impatient', and 'have you been anxious or worried, respectively. MHD is calculated by using items 2, 9, 12, and 14, which correspond to the following questions: 'did you feel depressed', 'have you felt downhearted and blue', 'have you been moody and brooded about things', and 'have you been in low or very low spirits', respectively. MHC is calculated by using items 5, 8, 16, and 17, which correspond to the following questions: 'have you been in firm control of your behaviours, thoughts, emotions, feelings', 'have you felt emotionally stable', 'did you feel you had nothing to look forward to', and 'have you felt so down in the dumps that nothing could cheer you up', respectively. Lastly, the MHP is calculated by taking the remaining items; 1, 7, 13, 15, which correspond to the following questions: 'has your daily life been full of things that were interesting to you', 'have you felt calm and peaceful', 'have you felt cheerful, light-hearted', and 'were you a happy person', respectively. Once each section has been partitioned and summed, the total score as well as the total subscale scores will range from 0-100, with higher scores indicating better mental health (Meybodi et al., 2011).

The SF-36 is a self-administered questionnaire consisting of 36 questions measuring eight dimensions of health: physical functioning, social functioning, role limitations due to physical problems, role limitations due to emotional problems, mental health, pain, general health and health change (Brazier et al., 1992; Garratt et al., 1994). Lastly, the sleep logs, which were to be filled out following each night's sleep, recorded the participants' duration (e.g., sleep start and sleep end) and quality of sleep, as well as rating how they felt on a scale of 0-100%, 100% being 'normal'. Participants were not prompted with any keywords, but rather asked to give a brief explanation of, if applicable, why they were not feeling 100%.

3.4 Data Analysis

A mixed two-way ANOVA with Bonferroni correction (to avoid type I error) was employed to assess the effect of Group and Time on all questionnaire data, as well as for the 5minute HR and HRV data (including RMSSD and BP measures). In the event of a group x time interaction, a two-tailed unpaired *t*-tests at each time point examined the between group (CTRL vs. HOCK) differences among questionnaire data as well as discrepancies in HRV data. Levene's test for equality of variances tests the null hypothesis that the population variances are equal. This test calculates an F-statistic and an associated p-value to indicate evidence against the null hypothesis. A significant difference indicates that the alternative hypothesis will be accepted and the variance between the populations is not equal. In the case of unequal variance, SPSS has a built-in feature which employs the Welch *t*-test (1974) to modify and compute reliable results for these unequal variances.

Pearson correlations were used to determine whether RMSSD was significantly associated with the targeted mental health indices (MHI, BRS, GAD-7, and VAS). Correlations were

computed for both group's data at each time point, as well as all data pooled for each time point (e.g., N = 32 participants). This was done to get a greater account of the association between HRV and mental health for all university students, regardless of an active lifestyle. Data were pooled for global correlations. A Pearson correlation was also computed to examine whether or not menstrual phase was associated with measures of RMSSD, as well as independent *t* tests to determine any differences in RMSSD between the two menstrual phases (e.g., follicular and luteal). The α value for all correlations was set at 0.05. A multivariate regression was employed in attempt to explain part of the variability seen in the dependent variable, RMSSD. All data were analyzed using SPSS (v.22, IBM SPSS Inc., USA).

CHAPTER 4 – Results

4.1 Baseline anthropometrics and overall health

Several significant differences were detected between the two group's general health (Table 4.1 presented as mean \pm standard deviation (SD)) as assessed by the SF-36. The HOCK group consistently scored higher on the SF-36, revealing a significant main group effect and several significant differences (see Table 4.1). The HOCK group scored significantly higher for energy/fatigue (p < 0.05, d = 0.93), emotional well-being (p < 0.05, d = 0.81), social functioning (p < 0.05, d = 0.93), and general health (p < 0.05, d = 0.95). Table 4.2 displays participants' baseline hemodynamics. By a mixed two-way ANOVA, no significant differences were observed among the two groups for age, height, weight, and BMI over the 7-month protocol. The HOCK group showed no significant differences in baseline HR or diastolic blood pressure (DBP). However, a main effect of time revealed a significant difference in systolic blood pressure (SBP) for the HOCK group, F(2, 60) = 3.722, p < 0.05, $\eta^2 = 0.110$. This finding was especially prominent

in the fluctuations between T2 to T3 (121 ± 11.18mmHg to 115 ± 9.41 mmHg; p = 0.008, d = 0.59). No significant group effect was detected for differences in SBP, F(1, 30) = 3.737, p = 0.063, partial $\eta^2 = 0.111$. However, a higher SBP was measured in the HOCK group at T2 (122 ± 11mmHg) compared to the CTRL group (112 ± 11mmHg; p = 0.023, d = 0.909). The CTRL group only showed a significant difference in DBP between T1 to T2 (67 ± 7.96mmHg to 76 ± 6.77mmHg, p = 0.003, d = 1.24).

Table 4.1. 36-Item Short Form Survey (SF-36) scores from both HOCK and CTRL groups as a measure to assess of baseline overall health.

	НОСК	CTRL	<i>p</i> value
Physical functioning	96.18 ± 12.19	95.00 ± 8.02	0.753
Role limitation due to physical health	100.00 ± 0.00	90.00 ± 26.39	0.164
Role limitation due to emotional problems	78.41 ± 37.20	75.53 ± 29.57	0.812
Energy/fatigue *	69.12 ± 12.28	57.00 ± 13.72	0.013
Emotional well-being *	79.29 ± 9.41	70.93 ± 11.05	0.028
Social functioning *	93.47 ± 9.94	80.20 ± 17.44	0.012
Pain	87.18 ± 12.75	80.13 ± 19.17	0.226
General health *	79.12 ± 13.37	65.33 ± 15.52	0.011

Data were analysed using an unpaired *t*-test. Values are reported as mean \pm SD. *, denotes significances at the p < 0.05 level (2-tailed).

	Т	1	Т	2	Т	3
	НОСК	CTRL	НОСК	CTRL	НОСК	CTRL
Age, years	21 ± 1.5	21 ± 2.2	-	-	-	-
Height, cm	166.7 ± 6.8	165.3 ± 6.2	-	-	-	-
Weight, kg	65.2 ± 6.1	62.8 ± 8.8	-	-	-	-
BMI , kg/m^2	23.4 ± 1.6	22.9 ± 2.5	-	-	-	-
HR, bpm	60 ± 6	62 ± 8	62 ± 7	60 ± 6	60 ± 7	62 ± 7
SBP, mmHg	115 ± 8	113 ± 9	$123 \pm 11*$	112 ± 11	115 ± 10	110 ± 10
DBP , mmHg	74 ± 7	67 ± 8	74 ± 6	$76 \pm 7^{**}$	76 ± 8	74 ± 9

Table 4.2. Baseline descriptive, heart rate, and blood pressure measures at each session separated by hockey (HOCK) or control (CTRL) group.

Values are mean \pm SD. BMI, body mass index; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure.

*, denotes significance from T1 within HOCK group (p < 0.05).

**, denotes significance from T1 within CTRL group (p < 0.05).

4.2 The influence of menstrual cycle on HRV

No significant differences were detected between the RMSSD for participants in luteal or follicular phases at T1, p = 0.153, T2, p = 0.828, and T3, p = 0.792 (see table 4.3). Further, there were no significantly correlations between menstrual phase and RMSSD.

	Т	1	Т	2	Т	3
	Follicular N = 16	Luteal N = 15	Follicular N = 17	Luteal N = 14	Follicular N = 13	Luteal N = 18
RMSSD, ms	53 ± 15	62 ± 20	65 ± 23	63 ± 19	67 ± 27	65 ± 22

Table 4.3. RMSSD based on menstrual phase (follicular or luteal) for all participants at T1, T2, and T3.

Values are mean \pm SD. RMSSD, root mean square of successive differences. No significant differences were detected, alpha set at 0.05, 2-tailed.

4.3 Heart rate variability parameters

The HOCK group showed no significant differences in the time domain HRV index referred to as the standard deviation of the average normal-to-normal (NN) intervals (SDNN), nor the frequency domain referred to as total power (TP), and percentages of very low frequency power (VLF%), low frequency power (LF%) and high frequency power (HF%) (see table 4.4). A main effect of time was observed in RMSSD (F(2, 60) = 3.393, p < .05, partial $\eta^2 = 0.102$) where RMSSD increased from 59 ± 16.47ms to 71 ± 22.14ms from T1 to T3 (p = 0.01, d = 0.89) in the HOCK group. No further significant effects or interactions were detected over time for HRV metrics (see Table 4.4).

	T1	1	L	T2	T3	3
	НОСК	CTRL	НОСК	CTRL	HOCK	CTRL
RMSSD, ms	59 ± 16	54 ± 20	66 ± 19	58 ± 25	71 ± 23 *	57 ± 24
SDNN, ms	71 ± 26	52 ± 21	69 ± 24	56 ± 22	78 ± 37	58 ± 24
Total Power, ms ²	6367 ± 4427	4467 ± 3147	6572 ± 3760	5100 ± 4082	<i>7577</i> ± <i>6</i> 270	5670 ± 4325
VLF%	2.8 ± 1.5	2.1 ± 1.5	2.5 ± 1.4	2.3 ± 0.9	2.5 ± 1.0	2.3 ± 1.2
LF%	41.1 ± 14.2	29.8 ± 13.6	38.2 ± 12.1	33.9 ± 8.4	39.0 ± 10.8	31.3 ± 12.4
HF%	57.1 ± 15.3	68.0 ± 14.3	59.3 ± 13.4	64.0 ± 8.6	58.5 ± 11.3	66.5 ± 13.0

AUUN -1 1 . 1. 4:-4 • Ξ ç ÷ . 111111 Ĩ VV Tabla 38

4.4 Mental health questionnaires and indices

No significant interactions were detected for group and for MHI and GAD-7 scores (see Table 4.5). The HOCK group did, however, display consistently higher scores on the MHI compared to the CTRL group (proportional to a scale of 0 to 100) and consistently lower on the GAD-7 when compared to CTRL at all three time points (see Table 4.5). Despite this pattern, there was no main effect of group on these scores. There was a statistically significant group x time interaction on BRS scores, F(2, 60) = 6.468, p < 0.05, partial $\eta^2 = 0.177$. Subsequent contrasts indicated a significantly greater (p < 0.05) BRS scores for HOCK compared to CTRL at T3 but not for T1 or T2 (see Table 4.5; Figure 4.3). A main effect of group showed that there was a significant difference in state anxiety, as assessed by the VAS, between the two groups F(1, 30) = 11.537, p = .002, partial $\eta^2 = .278$. Post-hoc analysis of VAS scores revealed that the level of state anxiety experienced by the CTRL group was significantly higher (p < 0.05) on "rest days," or day one in comparison to the HOCK group a T1, T2, and T3 (see Table 4.5). However, on "game days," or day two of data collection, this discrepancy dissolved as the HOCK group's state anxiety increased at all three time points (see Table 4.5).

No significant interaction was detected between the two groups and time on the subscale of anxiety (MHA), F(2, 60) = 0.011, p = 0.973, partial $\eta^2 = 0.001$. Further, there was no main effect of time on anxiety levels at the different time points, F(2, 60) = 1.156, p = 0.321, partial η^2 = .037, nor main effect of group F(1, 30) = 1.599, p = 0.216, partial $\eta^2 = 0.051$. No significant interaction was detected between the groups and time on the subscale of depression (MHD), F(2, 60) = 0.867, p = 0.426, $\eta^2 = 0.028$. However, the main effect of time showed a significant difference in levels of depression at the different time points, F(2, 60) = 4.052, p < 0.05, partial η^2 = 0.119. Specially, the HOCK group scored higher than the CTRL group at T2 for MHD (HOCK: 80.30 ± 14.60 vs. CTRL: 67.80 ± 12.50, p = 0.015) and MHC (HOCK: 85.90 ± 9.20 vs. CTRL: 76.30 ± 14.20, p = 0.030). The main effect of group showed no significant differences in depression levels between the two groups F(1, 30) = 3.609, p = 0.067, partial $\eta^2 = 0.107$. No significant interaction was detected between the two groups or time on the subscale of behavioural and emotional control (MHC), F(2, 60) = 1.558, p = 0.219, partial $\eta^2 = 0.049$. Further, there was no main effect of time on behavioural or emotional control at the different time points, F(2, 60) =0.195, p = 0.824, partial $\eta^2 = 0.006$, nor main effect of group F(1, 30) = 2.055, p = 0.162, partial $\eta^2 = 0.064$. No significant interaction was detected between the two groups and time on the subscale of positive affect (MHP), F(2, 60) = 0.258, p = 0.774, partial $\eta^2 = 0.009$. Further, the main effect of time on behavioural control levels at the different time points was significant, F(1, 30) = 4.543, p = 0.041, partial $\eta^2 = 0.132$. The main effect of group was not found to be significant F(1, 30) = 0.002, p = 0.961, partial $\eta^2 = 0.001$.

Independent sample *t*-tests were used to analyse the Group effect for each question within the MHI subgroups (anxiety, MHA; depression, MHD; behavioural control, MHC; positive affect, MHP). All data from all questions were pooled to get an average regardless of time point and separated by participant group leading to sample sizes of HOCK (n = 51) and CTRL (n = 44) for each question (see Table 4.6 & Appendix A; Table A.1). The assumption of homogeneity of variances was violated, as assessed by Levene's test for equality of variances, in questions 5, 8, and 13 (p = 0.038, p = 0.046, and p = 0.044) of the MHI but not for the remainder of the questions (p < 0.05). MHA is comprised of questions 4, 6, 10, 1, and 18. No differences were detected between the two groups for questions 4 (p = 0.207), 6 (p = 0.164), 11 (p = 0.960) or 10 (p = 0.062). A significant difference was detected for question 18 (p = 0.012, d = 0.517). The overall difference in MHA scores between groups was not significant, despite the HOCK group scoring consistently higher (see Table 4.6). MHD is comprised of questions 2, 9, 12, and 14. Questions 2 (p = 0.069) and 12 (p = 0.337) showed no significant difference between the two groups over the averaged time points, whereas question 9 (p = 0.030) and 14 (p = 0.038) did show a significant difference. The overall difference in MHD scores between groups was significant (p = 0.031, d = 0.419; Table 4.6). MHC is comprised of questions 5, 8, 16, and 17. Question 5 (p = 0.014, d = 0.511) and 8 (p = 0.011, d = 0.528) showed statistical significance. However, questions 16 (p = 0.419) and 17 (p = 0.694) showed no significant differences between the two groups. The overall difference in MHC scores between groups was significant (p = 0.041, d = 0.408; see Table 4.6). MHP is comprised of questions 1 (p = 0.968), 7 (p = 0.131), 13 (p = 0.098), and 15 (p = 0.467) all showed no significant differences between the two groups. The overall difference in MHP scores between groups also showed no significant difference (see Table 4.6).

Responses for overall well-being rated on a scale of 0% to 100%, where 100% is defined as feeling 'normal', were analyzed between groups. The HOCK recorded an average of $88.94 \pm 13.35\%$ on day one and $86.61 \pm 19.82\%$ on day two across the three time points (See Appendix A; Table A.2.). The CTRL group reported an average of $89.56 \pm 7.94\%$ on day one and $85.86 \pm 11.05\%$ on day two across the three time points. These averages were produced from pooled data over the span of the seven-month protocol. Of the 192 total responses across the academic school year, 80 responses reported the cause of their distress was due to "lack of sleep" or "feeling tired" (HOCK: 52 vs CTRL: 28). A total of 17 responses said the attenuated overall well-being score was attributed to "being sick" or "having an illness" (HOCK: 2 vs. CTRL 15). A total of 5 responses were due to "anxiety" or "feeling overwhelmed" (HOCK: 1 vs. CTRL: 4), and 19 reported "feeling stressed" (HOCK: 0 vs. CTRL: 19).

Table 4.5. Mental health indices at each time point for both the HOCK and CTRL group.

		IL			T2			T3	
	НОСК	CTRL	p value	HOCK	CTRL	<i>p</i> value	носк	CTRL	<i>p</i> value
ШМ	75.03 ± 15.11	70.51 ± 12.98	0.370	76.34 ± 11.50	72.46 ± 11.95	0.359	77.77 ± 11.25	71.93 ± 14.38	0.216
BRS	3.56 ± 0.42	3.37 ± 0.68	0.326	3.81 ± 0.61	3.56 ± 0.47	0.203	4.21 ± 0.58	3.58 ± 0.57	0.004*
GAD-7	4.35 ± 4.37	5.53 ± 3.80	0.420	3.76 ± 4.74	4.93 ± 3.69	0.440	3.12 ± 4.12	4.73 ± 4.08	0.275
VAS 1	20.88 ± 21.45	41.33 ± 22.32	0.013*	24.12 ± 13.72	36.67 ± 17.59	0.035*	15.88 ± 10.64	30.00 ± 22.36	0.027*
VAS 2	35.00 ± 25.00	42.00 ± 26.78	0.453	41.18 ± 21.76	43.33 ± 26.37	0.804	37.06 ± 18.63	34.00 ± 22.30	0.679
 -	 		-		- - -	-			

Values are reported as mean \pm SD. MHI, mental health inventory; BRS, brief resilience scale; GAD-7, general anxiety 7-item scale; VAS, visual analog scale. *, denotes significance at the 0.05 level (2-tailed).

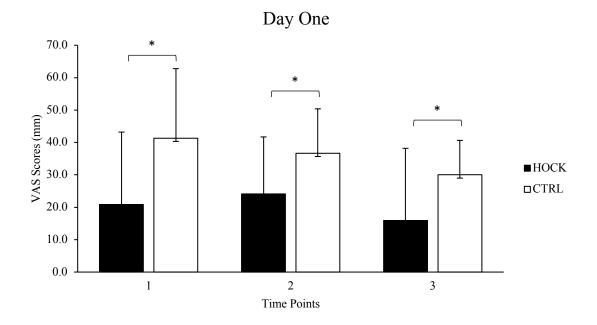


Figure 4.1. Displays the mean VAS scores calculated over the three time points between the CTRL and HOCK groups from all day one data. *, significant difference between groups, p < 0.05

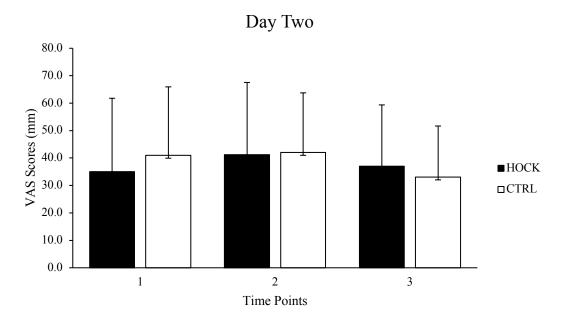


Figure 4.2. Displays the mean VAS scores calculated over the three time points between the CTRL and HOCK groups from all day two data. No significant differences were detected between groups.

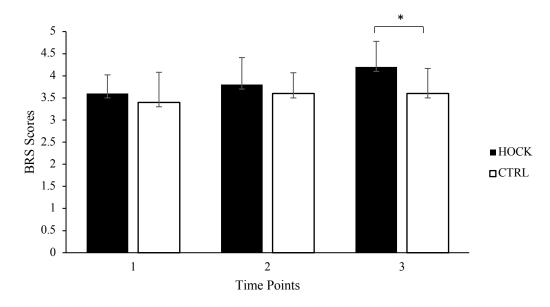


Figure 4.3. Displays the mean and standard deviation of BRS scores for the HOCK and CTRL groups at each time point. *, denotes significant difference (p < 0.05).

4.5 Regression of independent variables on RMSSD

A multivariate regression was conducted using age, BMI, menstrual cycle phase (e.g., follicular or luteal), activity level (e.g., HOCK or CTRL), all mental health indices (e.g., MHA, MHD, MHP, GAD-7, and BRS), as well as the eight categories found within the SF-36. MHC scores were excluded as the assumption of independent residuals was violated. R² for the proposed overall model was 36.0% with an adjusted R² of 22.1%, a small to moderate size effect according to Cohen (1988). The regression revealed that age, BMI, activity level, cycle phase, BRS, GAD-7, MHA, MHD, MHP and the eight items from the SF-36 statistically significantly predicted RMSSD, F(17, 78) = 2.584, p = 0.002 (see Table 4.7).

HOCKCTRLHOCKCTRLHOCMHA 65.65 ± 22.30 58.20 ± 24.55 67.53 ± 17.43 60.53 ± 24.55 $68.64 \pm$ MHD 82.65 ± 16.40 75.00 ± 15.58 80.30 ± 14.63 $67.80 \pm 12.51*$ $83.82 \pm$ MHC 83.53 ± 17.57 81.33 ± 15.29 85.88 ± 10.96 $76.33 \pm 14.20*$ $85.30 \pm$	HOCK 65.65 ± 22.30 82.65 ± 16.40	CTRL 58.20 ± 24.55	HOCK			
65.65 ± 22.30 58.20 ± 24.55 67.53 ± 17.43 60.53 ± 24.55 82.65 ± 16.40 75.00 ± 15.58 80.30 ± 14.63 $67.80 \pm 12.51*$ 83.53 ± 17.57 81.33 ± 15.29 85.88 ± 10.96 $76.33 \pm 14.20*$	65.65 ± 22.30 82.65 ± 16.40	58.20 ± 24.55		CTRL	HOCK	CTRL
$82.65 \pm 16.40 75.00 \pm 15.58 80.30 \pm 14.63 67.80 \pm 12.51 *$ $83.53 \pm 17.57 81.33 \pm 15.29 85.88 \pm 10.96 76.33 \pm 14.20 *$	82.65 ± 16.40		67.53 ± 17.43	60.53 ± 24.55	68.64 ± 17.60	62.40 ± 13.34
83.53 ± 17.57 81.33 ± 15.29 85.88 ± 10.96 $76.33 \pm 14.20*$		75.00 ± 15.58	80.30 ± 14.63	$67.80 \pm 12.51^*$	83.82 ± 12.93	77.00 ± 19.35
	83.53 ± 17.57	81.33 ± 15.29	85.88 ± 10.96	$76.33 \pm 14.20*$	85.30 ± 10.96	78.00 ± 17.10
MHP 67.94 ± 14.26 68.67 ± 15.30 71.47 ± 72.94 68.67 ± 15.30 $72.94 \pm 12.94 \pm 12.30$	67.94 ± 14.26	68.67 ± 15.30	71.47 ± 72.94	68.67 ± 15.30	72.94 ± 15.82	71.33 ± 13.70

Table 4.6. Subgroups within the Mental Health Inventory (MHI) at each time point, as well as the pooled average, for both the HOCK and CTRL group.

Variable	В	SE _B	β	<i>p</i> value
Intercept	0.705	83.753		
Fitness	1.433	6.001	0.034	0.812
Age	-0.531	1.488	-0.046	0.722
BMI	-0.561	1.522	-0.052	0.714
Menstrual phase	6.714	4.122	0.176	0.107
BRS	2.167	5.089	0.056	0.671
GAD	-0.821	0.756	-0.158	0.281
MHA	-0.392	0.198	-0.320	0.051
MHD	0.258	0.214	0.189	0.232
MHP	0.218	0.216	0.144	0.316
Physical Functioning	0.536	0.251	0.256	0.036 *
Role limitation due to physical health	-0.153	0.159	-0.130	0.340
Role limitation due to emotional problems	-0.172	0.074	-0.266	0.023 *
Energy/fatigue	0.536	0.242	0.351	0.030 *
Emotional well-being	-0.070	0.377	-0.035	0.853
Social functioning	0.048	0.179	-0.034	0.790
Pain	-0.109	0.177	-0.082	0.541
General health	0.265	0.189	0.194	0.166

Table 4.7. Summary of the regression model for predicting RMSSD.

Note. *B*, unstandardized regression coefficient; SE_B, standardized error of the coefficient; β , standardized coefficient. *, denotes significance, p < 0.05

4.6 HRV Responses Correlated with Mental Health Indices

RMSSD correlated with MHI at T2 for the CTRL group (r = 0.640, p = 0.010), but not T1, or T3 (see Table 4.8). No additional significant correlations were detected for each time point between HRV and mental health indices (see Table 4.8). The HOCK group showed no significant correlations between HRV and mental health indices over the 7-month span of three time points (see Table 4.9).

The two participant groups were pooled to investigate the general university student population over the three time points, thereby increasing the diversity of the sample as well as increasing N for correlation of data (N = 32). Subsequently, RMSSD showed a significant correlation with MHI scores at T2 (r = 0.453, p = 0.009), but at no other time points (see Table 4.10). RMSSD also showed a significant correlation with BRS scores at T3 (r = 0.358, p = 0.035), but at no other time points. Lastly, an overall global correlation was conducted between HRV and mental health indices, pooling all data points from participants regardless of group and disregarding time points (n = 96). Global correlations revealed a weak though significant positive association (r = 0.311, p = 0.002) between RMSSD and BRS (Table 4.11; Figure 4.6). Furthermore, there was a negative association between and GAD-7 (r = -0.216, p = 0.035; Figure 4.7), as well as a positive association with MHI (r = 0.373, p = 0.001; Table 4.10; Figure 4.5).

	Time	MHI	BRS	GAD-7	VAS 1	VAS 2
	1	0.338	0.310	-0.258	0.125	-0.076
RMSSD, ms	2	0.640*	0.271	-0.425	0.066	-0.265
	3	0.477	0.098	-0.244	-0.256	-0.096

Table 4.8. Pearson correlations of RMSSD and mental health indices at each time point for the CTRL group (N = 15).

Values are reported as Pearson correlations. RMSSD, root mean square of successive differences; MHI, mental health inventory; BRS, brief resilience scale; GAD-7, general anxiety 7-item scale; VAS, visual analog scale. *, significant at the 0.05 level (2-tailed).

Table 4.9. Pearson correlations of RMSSD and mental health indices at each time point for the HOCK group (N = 17).

	Time	MHI	BRS	GAD-7	VAS 1	VAS 2
	1	0.083	0.260	-0.161	-0.077	0.086
RMSSD, ms	2	0.195	0.197	0.012	0.209	0.278
	3	0.058	0.401	0.031	-0.099	0.127

Values are reported as Pearson correlations. RMSSD, root mean square of successive differences; MHI, mental health inventory; BRS, brief resilience scale; GAD-7, general anxiety 7-item scale; VAS, visual analog scale. No significant associations were detected.

	Time	MHI	BRS	GAD-7	VAS 1	VAS 2
	1	0.217	0.303	-0.218	-0.024	-0.017
RMSSD, ms	2	0.453**	0.257	-0.210	0.039	-0.046
	3	0.336	0.358*	-0.155	-0.287	0.030

Table 4.10. Pearson correlations of RMSSD-HRV and mental health indices at each time point, regardless of participant group (N = 32).

Values are reported as Pearson correlations. RMSSD, root mean square of successive differences; MHI, mental health inventory; BRS, brief resilience scale; GAD-7, general anxiety 7-item scale; VAS, visual analog scale. *, significant at the 0.01 level (2-tailed); **, significant at the 0.05 level (2-tailed).

Table 4.11. Pearson correlations for pooled data from both HOCK and CTRL groups for global Pearson correlations of RMSSD-HRV and mental health indices, disregarding time points (N = 96).

	MHI	BRS	GAD-7	VAS 1	VAS 2
RMSSD, ms	0.341**	0.311**	-0.216*	-0.088	-0.010

MHI, mental health inventory; BRS, brief resilience scale; GAD-7, general anxiety 7-item scale; VAS, visual analog scale. *, significant at the 0.01 level (2-tailed); **, significant at the 0.05 level (2-tailed).

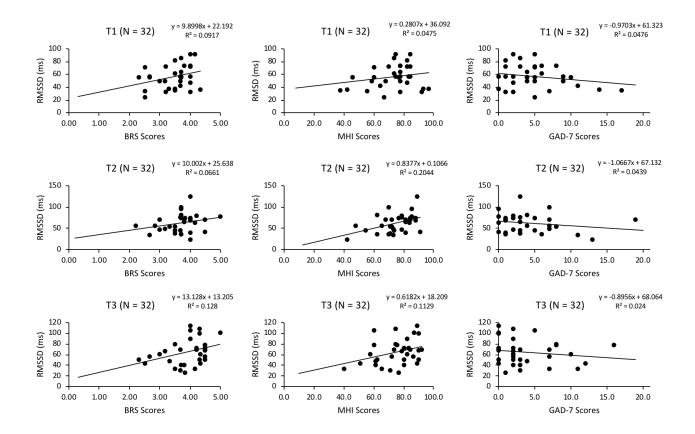


Figure 4.4. A. Displays the correlation for female university students at time points 1-3 (columns) for root mean square of successive differences (RMSSD) and Brief Resilience Scale (BRS) scores (T1 p = 0.092, T2 p = 0.156, T3 p = 0.044). B. Displays the correlation for university students at time points 1-3 (columns) for RMSSD and Mental Health Inventory (MHI) scores (T1 p = 0.232, T2 p = 0.009, T3 p = 0.060). C. Shows the correlations for university students at time points 1-3 (columns) for RMSSD and Generalized Anxiety Disorder 7-item scale (GAD-7) scores (T1 p = 0.230, T2 p = 0.250, T3 p = 0.397).

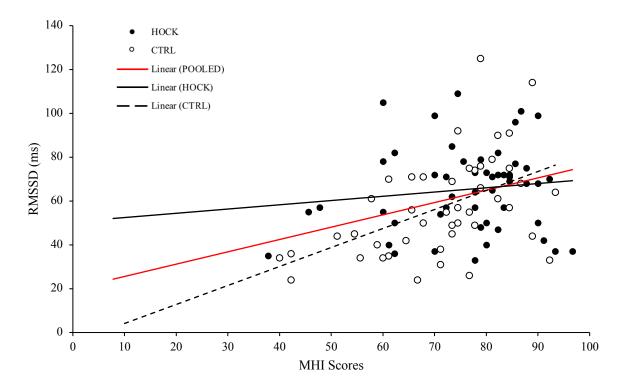


Figure 4.5. Global correlation of female university student data showing the positive association between the root mean square of successive differences (RMSSD) and mental health inventory (MHI) scores for all data (N = 96): r = 0.341, p = 0.001, 2-tailed.

HOCK (n = 51): y = 0.194x + 50.517, R² = 0.0155; CTRL (n = 45): y = 0.8667x - 4.5324, R² = 0.2463; POOLED: (n = 96): y = 0.5622x + 19.952, R² = 0.116.

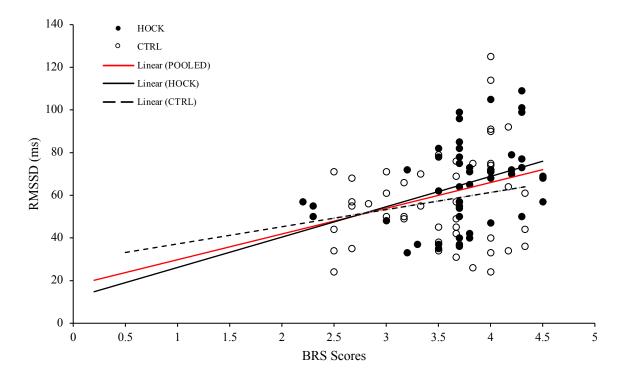


Figure 4.6. Global correlation of female university student data showing the positive association between the root mean square of successive differences (RMSSD) and brief resilience scale (BRS) scores for all data (N = 96), r = 0.311, p = 0.002, 2-tailed.

HOCK (n = 51): 14.231x + 11.924, R² = 0.1365; CTRL (n = 45): y = 8.043x + 29.155, R² = 0.041; POOLED (n = 96): y = 12.063 + 17.728, R² = 0.0967

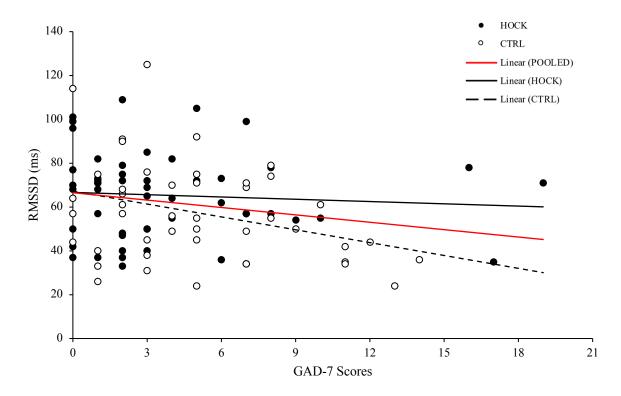


Figure 4.7. Global correlation of female university student data showing the negative association between the root mean square of successive differences (RMSSD) and generalized anxiety disorder 7-item (GAD-7) scores for all data (N = 96), r = -0.216, p = 0.035, 2-tailed. HOCK (n = 51): y = -0.3456x + 66.658, R² = 0.0058; CTRL (n = 45): y = -1.9561x + 67.222, R² = 0.1061; POOLED: y = -1.9561x + 67.222, R² = 0.1061.

CHAPTER 5 – Discussion

In this study, there were three important findings. First, the HOCK group displayed greater resilience, as seen through their BRS scores, throughout the seven-month period in comparison to their non-varsity counterparts. Second, the CTRL group reported greater levels of both state and trait anxiety, as seen through VAS scores and GAD-7 scores, respectively. Finally, the vagally mediated measure of HRV (RMSSD) of the HOCK players showed a progressive increase throughout the 7-month protocol, which was not observed in the CTRL group. This improvement

in RMSSD coupled with the improvements seen in BRS scores for the HOCK group suggest that there were physiologic adaptations which may have allowed for greater autonomic responsivity and mental resilience. There is no distinct correlation between RMSSD-HRV and mental health indices at each individual time point as seen through Pearson correlations. When data from both groups were combined, BRS and MHI correlated with RMSSD although the *r* values were low, accounting for roughly 10% of variation in RMSSD each. Therefore, the original hypothesis that RMSSD may be used as a predictive indicator of psychological resilience is not supported by this data.

5.1 Reliability of Firstbeat Artefact Correction

The recognition and correction of artefacts in HR time series has a significant impact on the accuracy of HRV recording and analysis (Porges & Bryne, 1992). The Firstbeat SPORTS software provided a reliable method to analyze the HRV data as it has the capability to parse various physiological states based on the variability seen in ambulatory recordings of R-R intervals (Parak & Korhonen, 2013). The BG2 device detects on average 99.95% of heartbeats (0.05% of beats missed) and has a 0.16% extra beat detection rate, with 2.96ms (0.54%) mean absolute error (MAE) in RRI as compared to the standard ECG (Parak & Korhonen, 2013). After the application of artefact correction, 99.98% of all heartbeats were correctly detected with MAE of 2.27% and a mean difference in RMSSD of 1.30ms in comparison to ECG recordings (Parak & Korhonen, 2013).

Artefacts in heart rate time series are common in both healthy and clinical individuals and are caused by several sources seen in both laboratory and field monitoring (Sami, Mikko, & Antti, 2004). It is important to note that there are superficial factors such as contact difficulty, moisture

with electrodes, and sudden body movements or jolts, that cause errors in monitoring which may create artefacts (Sami, Mikko, & Antti, 2004). Internal factors initiated by the body such as arrhythmias are not technically considered artefacts although they may lead to abnormal recordings (e.g., tachycardia and bradycardia) and alter computations and therefore are considered artefact by the Firstbeat software when producing HR and HRV data. These arrhythmias may produce extra (EB) or missing beats (MB). Missing beats are due to unrecognized QRS-waves, while extra beats originate from false detection of QRS-waves which causes the corresponding RRI to be split in to several RRI (Sami, Mikko, & Antti, 2004). For this reason, and the dynamic nature of this study's protocol, the Firstbeat SPORTS software and methodology for artefact correction was accepted as a reliable means to produce and analyze HRV data.

5.2 Hemodynamics

The dramatic fluctuation in systolic blood pressure (SBP) seen from T2 to T3 (-8mmHg) in the HOCK group was unexpected and no clear explanation can be determined from available data. Although speculative, this SBP response at T2 may have been influenced by their competitive situation. T2 was collected in January, following their winter break. This could also be considered a crucial time in the season before playoffs begin. It could be postulated that the fluctuation was influenced by the increased workload experienced following the winter break. However, a study done on cardiovascular control during an intensified training protocol in track and field athletes found that there were no significant changes in blood pressure variability (BPV) over time (Baumert et al., 2006). Further analysis showed that 53% of HOCK participants (9 of 17) had a percent change greater than 5% in SBP for T2 which regressed at T3. This fluctuation seen in SBP cannot be explained for by the current data and should not be strongly considered as an adaptation

due to stress or training.

The CTRL group experienced a significant increase in diastolic blood pressure (DBP) from T1 to T2 (+ 9 mmHg), which remained slightly elevated through T3 (+ 7mmHg from T1). The reason behind this change is not known, though it may be speculated that this increase in DBP may be due to external stressors such as school workload. It could also be that T2 was more stressful than T1 as the academic load would be greater as the term progressed. However, no definitive conclusions may be drawn due to insufficient data.

5.3 Time domain of heart rate variability

Most studies examining athletes and HRV look at the pre-competitive and competitive states. However, the current study was concerned with the effects of stress associated with being a full-time varsity athlete on HRV. The increase in RMSSD from T1 to T3 (+ 12ms) in the HOCK group could be explained by training adaptations. The CTRL group did not show any improvements in RMSSD but rather a plateau throughout the 7-months. This, coupled with the heightened stress seen in the CTRL, suggests that the training and exercise done by the HOCK group resulted in the alterations found in RMSSD.

5.4 Frequency domain of heart rate variability

The HOCK group appeared to have a greater total power (ms²) in R-R interval spectral analysis than the CTRL group at all three time points. Total power corresponds to the sum of energy found within the very low frequency band (VLF), low frequency band (LF), and HF band during short-term recordings (e.g., less than 24 hours). This would imply that the HOCK group

had greater autonomic influence over HR. Furthermore, when examining the frequency domain of HRV, the CTRL group showed consistently higher percentages of high frequency power than the HOCK group at all three time points. The high frequency band (0.15 to 0.40Hz) reflects parasympathetic activity and is closely linked with RSA (Bilchik & Berger, 2006; Shaffer & Ginsberg, 2017). This is an interesting and somewhat contradictory finding as exercise training and adaption should lead to greater inhibitory input from parasympathetic mechanisms which would be seen in the HOCK group (e.g., similar to that of their increased RMSSD which is known to be mediated by vagal activity). However, despite the greater levels of stress and anxiety seen in the CTRL group, their parasympathetic activity appears to be greater for this measure. This discrepancy may be explained by the HOCK group level of excitation following a hockey game leading to greater input from the sympathetic nervous system and greater vagal withdrawal. It may also suggest that there are external mechanisms behind the increases in RMSSD seen in the HOCK group that are not due to training.

One study examined the influence of sleep and circadian systems on cardiac autonomic activity by omitting sleep in one of the two groups and found that the respiratory sinus arrhythmia (RSA) showed a 24-h rhythm independent of sleep, whereas sympathetic activity, in this case measured by the pre-ejection period and low frequency (LF; 0.15 to 0.4Hz) spectral analysis, only showed a 24-h rhythm if sleep occurred (Burgess, Trinder, Kim, & Luke, 1997). This study suggests that circadian rhythms have a great influence over RSA and thus parasympathetic activity, whereas sleep impacts sympathetic activity on the heart. Also, increased daily levels of stress lead to increased stress-induced sleep disturbances (Gouin et al., 2015). This would imply that if the CTRL group was experiencing a greater number of daily stressors, their sleep quality and duration would be heavily impacted. Further, according to Burgess (1997), the LF component is heavily

influenced by sleep and therefore would appear to be lower with less sleep. It could be hypothesized that the duration and quality of sleep could have influenced the CTRL group's HF%, an indicator of parasympathetic activity, during sleep.

5.5 Autonomic response to stressors

The responsivity of the autonomic nervous system dictates the rate at which physical or emotional modulations can be made to adapt to situational demands. Depending on the situation, the increase in heart rate may be the result of either augmented sympathetic activity or attenuated parasympathetic inhibition, known as vagal withdrawal (Appelhans et al., 2006). According to the neurovisceral integration model proposed by Thayer et al., (2009), self-regulation, or one's ability to control emotions, feelings and thoughts, is closely linked to heart rate activity. Specifically, the inhibitory circuits found within the central autonomic network (CAN), which are active during self-regulation, influence the heart through vagally mediated activity (Williams, Cash, Rankin, Bernadi, Koenig, & Thayer, 2015). A study examining the influence of prolonged worry on cardiovascular control found that heightened stressors were associated with high HR and low HRV (Brosschot, 2007). This suggests that the CTRL group may have had poor self-regulation throughout the 7-month protocol and thus showed a lower RMSSD when compared to their HOCK counterparts. Literature has suggested that females who have a lower RMSSD also exhibit submissive behaviour during interpersonal stressors, as well as demonstrate defensive coping strategies (Pauls & Stemmler, 2003; Sgoifo et al., 2003). It is possible that the CTRL group did not employ efficient coping mechanisms when faced with stress causing the increase in anxieties and plateau in RMSSD. These two theories would suggest that the discrepancies seen in RMSSD and mental health indices between the CTRL and HOCK groups were primarily due to the inability to cope and self-regulate emotion. Unfortunately, these coping data were not collected, and therefore this idea remains speculation.

5.6 Exercise and heart rate variability

Another possible explanation for the increase in RMSSD seen in the HOCK group is adaptation due to their training regime. These improvements correspond to the current literature on training and adaptation (Dixon, Kamath, McCartney, & Fallen, 1992; Kiss et al., 2016). Exercise is known to play a large role in the adaption of the autonomic nervous system and its responsivity to adverse environments. Therefore, it is inferred that the improvements in autonomic functionality and increased mental health are tantamount adaptions to sustained exercise regimes such as those adopted by female varsity hockey players. Training influences autonomic responsiveness by increasing parasympathetic output, which leads to bradycardia and increased HRV. This study did not quantify the extent of the energic expenditure or metabolic rates resulting from playing varsity hockey. It is possible that these adaptations in HRV and mental health could be accounted for by the workload placed on these athletes over the seven-month span.

5.7 Mental health indices

The CTRL group showed no improvements in any of the mental health indices collected (e.g., GAD-7, BRS, or MHI), whereas the HOCK group consistently improved throughout the seven-month protocol. The SF-36 results for each group allowed for a greater understanding of the general health of each participant group in a baseline setting. Compared to the CTRL group, the HOCK group scored significantly higher on 4 of the 8 categories. Specifically, energy/fatigue, emotional well-being, social functioning, and general health. These findings were anticipated due

to the HOCK group's team environment. It is also predicted that the CTRL group would report lower levels of mental health (e.g., MHI, GAD-7, BRS) as a result of the deficiency in social function and heightened fatigue leading to poor emotional well-being and an overall lower general health reported as a baseline measure.

A striking outcome was the discrepancy in VAS scores between the two groups. The VAS illustrates the participants' state anxiety experienced on days one and two for each time point. At each of the three time points, the CTRL group consistently scored higher on day one. This implies that this group had a greater level of "baseline" state anxiety compared to the HOCK group. However, the findings for VAS on day two revealed that the two groups had similar state anxieties. The HOCK group's increase in state anxiety was anticipated and can be explained by precompetitive anxiety and stress preceding a hockey game. Blasquez et al. (2009) examined precompetitive anxiety in swimmers and found that they had greater somatic anxiety, which supports the current findings of this study. However, the CTRL group's state anxiety did not differ greatly from their day one values. This is an important finding as it solidifies the intensity of the CTRL group's state anxiety that is matched by the HOCK group pre-competitive anxiety.

5.8 Contributing Factors to Resilience

There are many personal, environmental, and socio-cultural factors which influence an individual's resilience by means of either negative or protective mechanisms. Protective mechanisms are factors which lessen the impact of adverse environments, whereas negative mechanisms are those which exacerbate or weaken an individual's ability to cope with or bounce back from difficult situations. The term "connectedness" has been used in conjunction with psychological resilience showing that adolescents with higher levels of social connectedness report

lower levels of psychological distress (Dang 2014; Abukarar & Dimitrova, 2016). This idea remains speculation as sufficient data was not collected to gain a definitive sense of the team environment experienced in either participant group. Future research should examine the level of connectedness in both the athlete and control group.

5.9 Limitations

This study acknowledges the discernible limitations of sex and sport specificity. The participant group consisted of only females making it difficult to draw inferences and generalizations for the entire student population. Further, all athletic participants were members of the same varsity hockey team, making broad generalizations to the entire student-athlete population difficult. Future directions for similar studies should examine both male and female athletes, as well as recruiting athletes from a variety of sports. This would allow for broader inferences with regard to the student, and student-athlete population and account for greater variance in participants.

Along with the limitation of sex comes the impact of the menstrual cycle on HRV. Although results suggest that sympathetic activity is highest in the secretory phase with restored vagal influence in the proliferative phase, no significant differences in RMSSD between participants in the follicular or luteal phases were detected in the current data found within this study.

A third limitation in this study, which was not controlled for, was the influence of circadian rhythms on HRV. The five-minute RMSSD data was obtained from the period of participants' sleep in order to avoid the influence of any external factors and to provide a stable, repeatable condition. A study by Burgess et al., (1997) found that the RSA and thus parasympathetic activity exhibits a 24h rhythm regardless of sleep occurrence, whereas LF-HRV was associated with sleep.

This study suggests that an individual's sleep/wake cycle has a great impact on the level of autonomic activity on the heart.

5.10 Future directions

A future research direction to gain insight into pre-competitive anxiety for varsity athletes would be to acquire subjective feelings towards personal game play and performance and directly compare these outcomes with measures of HRV. This could allow the researcher to compare and contrast the influence of positive or negative self-feedback on autonomic functionality. This type of study could be further dissected into the influence on recovery times following a positive or negative outcome in a game. The implementation of a biofeedback group in a competitive setting such as in these varsity student-athletes, or further, in the general student population, could offer interesting findings regarding HR and HRV. Prinsloo, Rauch, and Derman (2014) have shown that HRV biofeedback can effectively increase HRV, cardiac vagal tone, and decrease stress. It would be worthwhile to replicate similar biofeedback studies in the present population to observe the influence of conscious efforts to modify stress levels and HRV.

Another possible direction would be to separate participants by school year (e.g., freshman, sophomore, junior, and senior). This would allow for a more in-depth analysis on resilience by year. It is possible that participants from upper years are more resilient to external stressors based on exposure over the years and thus have gained adequate coping strategies. It is also possible that school years may not play a factor, and participants' anxiety levels are independent of school year. In this sense, gathering a baseline account of trait anxiety and stratifying the participants into low, medium, and high anxiety groups may allow for the detection of patterns in their overall mental health which could be associated with their HRV.

An alternative to the daily log employed in the present study would be a questionnaire which breaks down all of the possible external stressors such as time commitments, lack of autonomy, relationships outside their sport, and school work load to try to pin-point which factors are playing the largest role in these university student's mental health outcomes. Additionally, it would be worthwhile adding a specific question regarding how or what is helping them get through the school year when times are tough, or when they are feeling stressed or overwhelmed. For the HOCK group, it is unclear whether or not it was the comradery of the team environment, the training and conditioning for their sport, or the sense of connectedness that was helping them. For the CTRL group, it would be interesting to note the resources available to them and which resources the students actually utilized. Gathering accounts of how university students cope and get through difficult times could provide a useful marker for mental health.

CHAPTER 6 – Conclusion

The present study explored the possible relationship between short-duration HRV (rootmean squared of successive differences in R-R intervals; RMSSD) and mental health outcomes. Specifically, this study aimed to show that RMSSD could be an indicator of psychological resilience in both varsity and non-varsity university students. RMSSD is sensitive to vagal activity as it is predominantly mediated by parasympathetic outputs. Therefore, it was hypothesized that a lower RMSSD would be associated with lower mental health outcomes and thus a weakened resilience. The results of this study indicate that, over time, female varsity hockey players develop greater mental resilience, as seen through the increased BRS scores, resulting in an increased ability to bounce back from difficult times or external stressors. Furthermore, it is suggested that these varsity athletes have improved overall mental health in comparison to their non-varsity counterparts. This was indicated by the increase in MHI scores and the decline in GAD-7 scores over time for the HOCK group, which is not seen in the CTRL group. Despite these apparent patterns in HRV and mental health outcomes, there was no correlation at each time point to signify the proposed relationship between RMSSD and mental resilience. However, when the data were considered globally, regardless of time points, RMSSD was correlated, albeit weakly, with BRS, as well as GAD-7 and MHI. This lack of significance in correlations may be a result of low sample size, which was partially resolved by pooling the data. Further research into this relationship is warranted.

REFERENCES

Appelhans, B., & Luecken, L. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, 10(3), 229.

Battipaglia, I., & Lanza, G. A. (2015). The Autonomic Nervous System of the Heart. In *Autonomic Innervation of the Heart* (pp. 1-12). Springer, Berlin, Heidelberg.

Baumert, M., Brechtel, L., Lock, J., Hermsdorf, M., Wolff, R., Baier, V., et al. (2006). Heart rate variability, blood pressure variability, and baroreflex sensitivity in overtrained athletes. *Clinical Journal of Sport Medicine*, 16(5), 412-417.

Bilchick, K., & Berger, R. (2006). Heart rate variability. *Journal of Cardiovascular Electrophysiology*, 17(6), 691.

Billman, G. E. (2013). The LF/HF ratio does not accurately measure cardiac sympatho-vagal balance. *Frontiers in Physiology*, 4, 26.

Brar, T., Singh, K., & Kumar, A. (2015). Effect of different phases of menstrual cycle on heart rate variability (HRV). *Journal of Clinical and Diagnostic Research*, 9(10), CC01.

Brosschot, J., Van Dijk, E., & Thayer, J. (2007). Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *International Journal of Psychophysiology*, 63(1), 39-47.

Burgess, H. J., Trinder, J., Kim, Y., & Luke, D. (1997). Sleep and circadian influences on cardiac autonomic nervous system activity. *American Journal of Physiology-Heart and Circulatory Physiology*, 273(4), H1761-H1768.

Burr, J., Jamnik, R., Baker, J, Macpherson, A., Gledhill, N., & McGuire, E. (2008). Relationship of physical fitness test results and hockey playing potential in elite-level ice hockey players. *The Journal of Strength & Conditioning Research*, 22(5), 1535-1543.

Carnevali, L., & Sgoifo, A. (2014). Vagal modulation of resting heart rate in rats: the role of stress, psychosocial factors, and physical exercise. *Frontiers in Physiology*, *5*, 118.

Cervantes Blásquez, J., Rodas Font, G., & Capdevila Ortís, L. (2009). Heart-rate variability and precompetitive anxiety in swimmers. *Psicothema*, 21(4).

Cresswell, S., & Eklund, R. (2007). Athlete burnout: A longitudinal qualitative study. *The Sport Psychologist*, *21*(1), 1-20.

D'ascenzi, F., Alvino, F., Natali, B., Cameli, M., Palmitesta, P., Boschetti, G., et al. (2014). Precompetitive assessment of heart rate variability in elite female athletes during play offs. *Clinical Physiology and Functional Imaging*, *34*(3), 230-236.

Deuster, P. A., & Silverman, M. N. (2013). Physical fitness: a pathway to health and resilience. US Army Medical Department Journal.

Dimitriev, D. A., Dimitriev, A. D., Karpenko, Y. D., & Saperova, E. V. (2008). Influence of examination stress and psychoemotional characteristics on the blood pressure and heart rate regulation in female students. *Human Physiology*, *34*(5), 617-624.

Dimitriev, D. A., Saperova, E. V., & Dimitriev, A. D. (2016). State anxiety and nonlinear dynamics of heart rate variability in students. *PloS One*, *11*(1), e0146131.

Dishman, R. K., Nakamura, Y., Garcia, M. E., Thompson, R. W., Dunn, A. L., & Blair, S. N. (2000). Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *International Journal of Psychophysiology*, *37*(2), 121-133.

Dixon, E. M., Kamath, M. V., McCartney, N., & Fallen, E. L. (1992). Neural regulation of heart rate variability in endurance athletes and sedentary controls. *Cardiovascular Research*, 26(7), 713-719.

Dong, J. G. (2016). The role of heart rate variability in sports physiology. *Experimental and Therapeutic Medicine*, 11(5), 1531-1536.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: attentional control theory. *Emotion*, 7(2), 336.

Firstbeat SPORTS. (2017). Firstbeat Bodyguard 2 Guide. Jyväskylä, Finland.

Forcier, K., Stroud, L. R., Papandonatos, G. D., Hitsman, B., Reiches, M., Krishnamoorthy, J., & Niaura, R. (2006). Links between physical fitness and cardiovascular reactivity and recovery to psychological stressors: A meta-analysis. *Health Psychology*, *25*(6), 723.

Friedman, B. H., & Thayer, J. F. (1998). Anxiety and autonomic flexibility: a cardiovascular approach1. *Biological Psychology*, 47(3), 243-263.

Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, 44(1), 133-151.

Gouin, J. P., Wenzel, K., Boucetta, S., O'Byrne, J., Salimi, A., & Dang-Vu, T. T. (2015). High-frequency heart rate variability during worry predicts stress-related increases in sleep disturbances. *Sleep Medicine*, *16*(5), 659-664.

Green, H., Bishop, P., Houston, M., McKillop, R., Norman, R., & Stothart, P. (1976). Timemotion and physiological assessments of ice hockey performance. *Journal of Applied Physiology*, 40(2), 159-163. Green, M. R., Pivarnik, J. M., Carrier, D. P., & Womack, C. J. (2006). Relationship between physiological profiles and on-ice performance of a National Collegiate Athletic Association Division I hockey team. *Journal of Strength and Conditioning Research*, *20*(1), 43.

Griffiths, A., Kouvonen, A., Pentti, J., Oksanen, T., Virtanen, M., Salo, P., ... & Vahtera, J. (2014). Association of physical activity with future mental health in older, mid-life and younger women. *The European Journal of Public Health*, *24*(5), 813-818.

Gulliver, A., Griffiths, K. M., & Christensen, H. (2012). Barriers and facilitators to mental health help-seeking for young elite athletes: a qualitative study. *BMC Psychiatry*, *12*(1), 157.

Gustafsson, H., Kenttä, G., & Hassmén, P. (2011). Athlete burnout: An integrated model and future research directions. *International Review of Sport and Exercise Psychology*, 4(1), 3-24.

Hall, M., Vasko, R., Buysse, D., Ombao, H., Chen, Q., Cashmere, J. D., ... & Thayer, J. F. (2004). Acute stress affects heart rate variability during sleep. *Psychosomatic Medicine*, *66*(1), 56-62.

Huang, C. J., Webb, H. E., Zourdos, M. C., & Acevedo, E. O. (2013). Cardiovascular reactivity, stress, and physical activity. *Frontiers in Physiology*, *4*, 314.

Hughes, L., & Leavey, G. (2012). Setting the bar: athletes and vulnerability to mental illness. *The British Journal of Psychiartry*, 200, 95-96.

Hynynen, E. S. A., Uusitalo, A., Konttinen, N., & Rusko, H. (2006). Heart rate variability during night sleep and after awakening in overtrained athletes. *Medicine & Science in Sports & Exercise*, *38*(2), 313-317.

Hynynen, E., Konttinen, N., Kinnunen, U., Kyröläinen, H., & Rusko, H. (2011). The incidence of stress symptoms and heart rate variability during sleep and orthostatic test. *European Journal of Applied Physiology*, *111*(5), 733-741.

Kageyama, T., Nishikido, N., Kobayashi, T., Kurokawa, Y., Kaneko, T., & Kabuto, M. (1998). Self-reported sleep quality, job stress, and daytime autonomic activities assessed in terms of short-term heart rate variability among male white-collar workers. *Industrial Health*, *36*(3), 263-272.

Kiss, O., Sydó, N., Vargha, P., Vágó, H., Czimbalmos, C., Édes, E., ... & Becker, D. (2016). Detailed heart rate variability analysis in athletes. *Clinical Autonomic Research*, *26*(4), 245-252.

Lachytova, M., Katreniakova, Z., Mikula, P., Jendrichovsky, M., & Nagyova, I. (2017). Associations between self-rated health, mental health problems and physical inactivity among urban adolescents. *The European Journal of Public Health*, *27*(6), 984-989.

Levy, M. N. (1984, August). Cardiac sympathetic-parasympathetic interactions. In *Federation Proceedings* (Vol. 43, No. 11, pp. 2598-2602).

Lucini, D., Di Fede, G., Parati, G., & Pagani, M. (2005). Impact of chronic psychosocial stress on autonomic cardiovascular regulation in otherwise healthy subjects. *Hypertension*, *46*(5), 1201-1206.

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: mechanisms leading to disease. *Archives of Internal Medicine*, 153(18), 2093-2101.

Miller, T. W., Vaughn, M. P., & Miller, J. M. (1990). Clinical issues and treatment strategies in stress-oriented athletes. *Sports Medicine*, *9*(6), 370-379.

Miu, A. C., Heilman, R. M., & Miclea, M. (2009). Reduced heart rate variability and vagal tone in anxiety: trait versus state, and the effects of autogenic training. *Autonomic Neuroscience: Basic and Clinical*, *145*(1), 99-103.

Montgomery, D. L. (2006). Physiological profile of professional hockey players-a longitudinal comparison. *Applied Physiology, Nutrition, and Metabolism, 31*(3), 181-185.

Morales, J., Garcia, V., García-Massó, X., Salvá, P., & Escobar, R. (2013). The use of heart rate variability in assessing precompetitive stress in high-standard judo athletes. *International Journal of Sports Medicine*, *34*(02), 144-151.

Munoz, M. L., van Roon, A., Riese, H., Thio, C., Oostenbroek, E., Westrik, I., ... & Snieder, H. (2015). Validity of (ultra-) short recordings for heart rate variability measurements. *PLoS One*, *10*(9), e0138921.

Myllymäki, T., Rusko, H., Syväoja, H., Juuti, T., Kinnunen, M. L., & Kyröläinen, H. (2012). Effects of exercise intensity and duration on nocturnal heart rate variability and sleep quality. *European Journal of Applied Physiology*, *112*(3), 801-809.

Noblet, A. J., & Gifford, S. M. (2002). The sources of stress experienced by professional Australian footballers. *Journal of Applied Sport Psychology*, 14(1), 1-13.

Parak, J., & Korhonen, I. (2013). Accuracy of Firstbeat Bodyguard 2 beat-to-beat heart rate monitor. *White paper by Firstbeat Technologies Ltd.*

Pauls, C. A., & Stemmler, G. (2003). Repressive and defensive coping during fear and anger. *Emotion*, 3(3), 284.

Pitzalis, M. V., Mastropasqua, F., Massari, F., Forleo, C., Di Maggio, M., Passantino, A., ... & Rizzon, P. (1996). Short-and long-term reproducibility of time and frequency domain heart rate variability measurements in normal subjects. *Cardiovascular Research*, *32*(2), 226-233.

Porges, S. W., & Byrne, E. A. (1992). Research methods for measurement of heart rate and respiration. *Biological Psychology*, *34*(2-3), 93-130.

Prinsloo, G. E., Rauch, H. L., & Derman, W. E. (2014). A brief review and clinical application of heart rate variability biofeedback in sports, exercise, and rehabilitation medicine. *The Physician and Sports Medicine*, 42(2), 88-99.

Pumprla, J., Howorka, K., Groves, D., Chester, M., & Nolan, J. (2002). Functional assessment of heart rate variability: physiological basis and practical applications. *International Journal of Cardiology*, *84*(1), 1-14.

Rice, S. M., Purcell, R., De Silva, S., Mawren, D., McGorry, P. D., & Parker, A. G. (2016). The mental health of elite athletes: a narrative systematic review. *Sports Medicine*, *46*(9), 1333-1353.

Ross, C. A., Ruggiero, D. A., & Reis, D. J. (1985). Projections from the nucleus tractus solitarii to the rostral ventrolateral medulla. *Journal of Comparative Neurology*, 242(4), 511-534.

Sami, S., Mikko, S., & Antti, K. (2004). Artefact correction for heart beat interval data. *1st Probisi 2004 Proceedings. Jyväskylä, Finland: University of Jyväskylä.*

Shaffer, F., McCraty, R., & Zerr, C. L. (2014). A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. *Frontiers in Psychology*, *5*, 1040.

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health*, 5, 258.

Skirka, N. (2000). The relationship of hardiness, sense of coherence, sports participation, and gender to perceived stress and psychological symptoms among college students. *Journal of Sports Medicine and Physical Fitness*, 40(1), 63.

Spiers, J. P., Silke, B., McDermott, U., Shanks, R. G., & Harron, D. W. (1993). Time and frequency domain assessment of heart rate variability: a theoretical and clinical appreciation. *Clinical Autonomic Research*, *3*(2), 145-158.

Summanen, R., & Westerlund, E., (2001). Let's Beat Them in Ice Hockey. Polar Electro Oy.

Task Force of the European Society of Cardiology. (1996). Heart rate variability, standards of measurement, physiological interpretation, and clinical use. *Circulation*, *93*, 1043-1065.

Teisala, T., Mutikainen, S., Tolvanen, A., Rottensteiner, M., Leskinen, T., Kaprio, J., ... & Kujala, U. M. (2014). Associations of physical activity, fitness, and body composition with heart rate variability–based indicators of stress and recovery on workdays: a cross-sectional study. *Journal of Occupational Medicine and Toxicology*, *9*(1), 16.

Triposkiadis, F., Karayannis, G., Giamouzis, G., Skoularigis, J., Louridas, G., & Butler, J. (2009). The sympathetic nervous system in heart failure: physiology, pathophysiology, and clinical implications. *Journal of the American College of Cardiology*, *54*(19), 1747-1762.

Twist, P., & Rhodes, T. (1993). Exercise physiology: The bioenergetic and physiological demands of ice hockey. *Strength & Conditioning Journal*, *15*(5), 68-70.

Watson, D., & Clark, L. A. (1984). Negative affectivity: the disposition to experience aversive emotional states. *Psychological Bulletin*, *96*(3), 465.

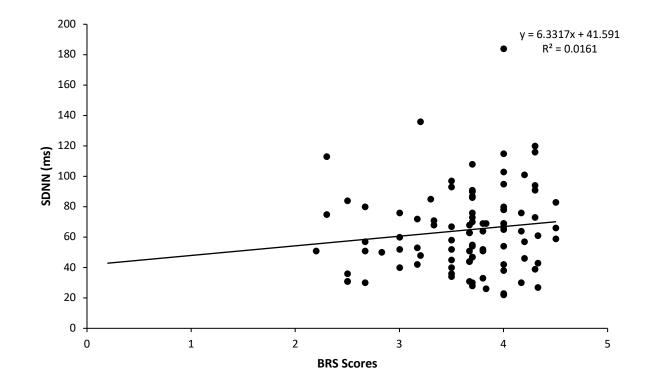
Williams, D. P., Cash, C., Rankin, C., Bernardi, A., Koenig, J., & Thayer, J. F. (2015). Resting heart rate variability predicts self-reported difficulties in emotion regulation: a focus on different facets of emotion regulation. *Frontiers in Psychology*, *6*, 261.

World Health Organization. (2017). Depression and other common mental disorders: global health estimates.

Yang, J., Peek-Asa, C., Corlette, J. D., Cheng, G., Foster, D. T., & Albright, J. (2007). Prevalence of and risk factors associated with symptoms of depression in competitive collegiate student athletes. *Clinical Journal of Sport Medicine*, *17*(6), 481-487.

Yazar, Ş. (2016). Impact of menstrual cycle on cardiac autonomic function assessed by heart rate variability and heart rate recovery. *Medical Principles and Practice*, 25(4), 374-377.

APPENDICES



APPENDIX A - Supplementary HRV Data

Figure A.1. Global Correlation of all SDNN-HRV and Brief Resilience Scale (BRS) Scores pooled from all three time points and all participant data.

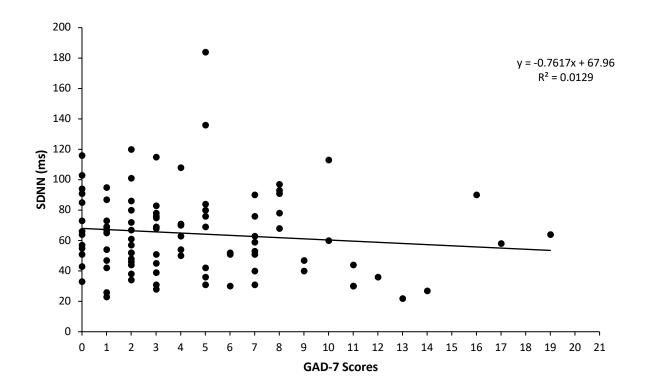


Figure A.2. Global Correlation of all SDNN-HRV and Generalized Anxiety Disorder (GAD-7) Scores pooled from all three time points and all participant data.

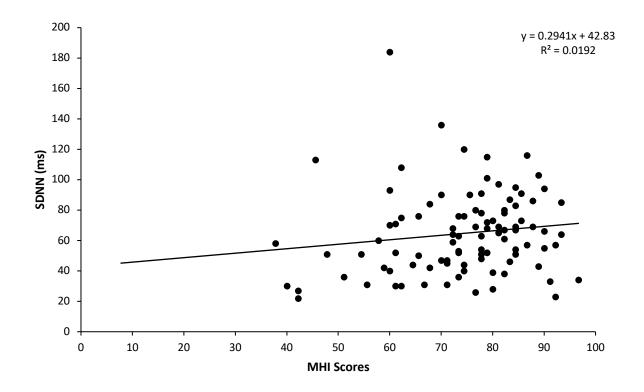


Figure A.3. Global Correlation of all SDNN-HRV and Mental Health Inventory (MHI) Scores pooled from all three time points and all participant data.

Questions	НОСК	CTRL
Qu 1	2.37 ± 0.85	2.38 ± 0.94
Qu 2	5.4259 ± 0.77	$5.11\ 5.11\pm 0.93$
Qu 3	1.70 ± 0.63	2.10 ± 1.24
Qu 4	4.37 ± 1.03	4.11 ± 0.98
Qu 5	1.98 ± 0.79	2.44 ± 1.01 *
Qu 6	4.35 ± 1.15	4.02 ± 1.18
Qu 7	2.94 ± 1.19	3.30 ± 1.04
Qu 8	2.11 ± 0.79	2.58 ± 0.97 *
Qu 9	5.11 ± 0.95	4.71 ± 0.84 *
Qu 10	2.65 ± 1.23	3.13 ± 1.32
Qu 11	4.39 ± 1.16	4.38 ± 1.03
Qu 12	4.63 ± 0.85	4.44 ± 1.06
Qu 13	2.50 ± 0.82	2.24 ± 0.68
Qu 14	5.30 ± 0.86	4.89 ± 1.07 *
Qu 15	2.10 ± 0.68	1.96 ± 0.67
Qu 16	5.48 ± 1.06	5.31 ± 1.02
Qu 17	5.57 ± 0.84	5.51 ± 0.73
Qu 18	4.41 ± 1.10	3.84 ± 1.09 *

Table A.1. Average response for the 18 items of the MHI questionnaire over the sevenmonth protocol separated by participant group.

	Т	1	T2		Τ3	
	НОСК	CTRL	НОСК	CTRL	НОСК	CTRL
Well-being day one (%)	89.2 ± 18.5	85.0 ± 7.7	89.4 ± 10.7	83.8 ± 8.5	92.6± 9.4	85.8± 7.6
Well-being day two (%)	90.3 ± 10.7	81.6± 7.9	88.2± 12.8	76.4 ± 12.7	86.4 ± 16.9	85.6 ± 10.3

Table A.2. Well-being scores (out of 100%) for each time point on Days one and two, separated by group.

APPENDIX B – Approval of Ethics

Western Research		Researc
	Western University Health Science Research Ethics Board HSREB Delegated Initial Approval Notice	
Principal Investigator: Dr. Kevin Shoemaker Department & Institution: Health Sciences\K	ünesiology,Western University	
Review Type: Delegated HSREB File Number: 108318 Study Title: Smart, Healthy Campus		
HSREB Initial Approval Date: November 11 HSREB Expiry Date: November 11, 2017 Documents Approved and/or Received for In		
Document Name	Comments	Version Date
Revised Western University Protocol		Version Dute
Letter of Information & Consent	Phase 2 - Version 2	2016/11/01
Letter of Information & Consent	Phase 3 - Version 2	2016/11/01
Instruments	Sense of Belonging Scale	2010/10/1
Instruments	SF-36	
Instruments	Mental Health Survey	
Instruments	NVRL health questionnaire	2016/05/24
Instruments	Brief Resilience Scale	
Instruments	Flourishing Scale	
Instruments	International Physical Activity Questionnaire	
Instruments	anxiety, visual analog scale	
Instruments	Thriving CIT	
Instruments	Alcohol Use Questionnaire	
Instruments	GAD-7	
Instruments	24 hour diet recall	
Other	Academic counselor discharge codes	
Other	Notification Letter re: access to student health services database	2016/09/29
Other	Notification Letter re: permission to access Student Experience Databases	2016/10/17
Advertisement	Received November 4, 2016	
	December Interviews/Focus Groups	
Instruments		

The Western University Health Science Research Ethics Board (HSREB) has reviewed and approved the above named study, as of the HSREB Initial Approval Date noted above.

HSREB approval for this study remains valid until the HSREB Expiry Date noted above, conditional to timely submission and acceptance of HSREB Continuing Ethics Review.

The Western University HSREB operates in compliance with the Tri-Council Policy Statement Ethical Conduct for Research Involving Humans (TCPS2), the International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use Guideline for Good Clinical Practice Practices (ICH E6 R1), the Ontario Personal Health Information Protection Act (PHIPA, 2004), Part 4 of the Natural Health Product Regulations, Health Canada Medical Device Regulations and Part C, Division 5, of the Food and Drug Regulations of Health Canada.

Members of the HSREB who are named as Investigators in research studies do not participate in discussions related to, nor vote on such studies when they are presented to the REB.

The HSREB is registration number IRB 00000940.

Ethics Officer: Erika Basile _____ Nicole Kaniki ___ Grace Kelly ___ Katelyn Harris ____ Vikki Tran ___ Karen Gepaul

Western University, Research, Support Services Bldg., Rm. 5150 London, ON, Canada N6G 1G9 t. 519.661.3036 f. 519.850.2466 www.uwo.ca/research/ethics C.1 Mental Health Inventory (MHI)

MENTAL HEALTH INVENTORY (MHI)

The next set of questions are about how you feel, and how things have been for you during the <u>past 4 weeks</u>. If you are marking your own answers, please <u>circle</u> the appropriate response (0, 1, 2,...). If you need help in marking your responses, <u>tell</u> <u>the interviewer the number</u> of the best response. <u>Please answer every question</u>. If you are not sure which answer to select, please choose the one answer that comes closest to describing you. The interviewer can explain any words or phrases that you do not understand.

During the past 4 weeks,

how much of the time...

	All of the <u>time</u>	Most of the <u>time</u>	A good bit of <u>the time</u>	Some of the <u>time</u>	A little bit of <u>the time</u>	None of the <u>time</u>
1. has your daily life been full of things that were		•	2		-	6
interesting to you?	1	2	3	4	5	6
2. did you feel depressed?	1	2	3	4	5	6
3. have you felt loved and wanted?	1	2	3	4	5	6
4. have you been a very nervous person?	1	2	3	4	5	6
5. have you been in firm control of your behavior, thoughts, emotions, feelings?	1	2	3	4	5	6

Western S HealthSciences

During the <u>past 4 weeks</u>, how much of the time...

	All of the <u>time</u>	Most of the <u>time</u>	A good bit of <u>the time</u>	Some of the <u>time</u>	A little bit of <u>the time</u>	None of the <u>time</u>
6. have you felt tense or high-strung?	1	2	3	4	5	6
ingi ou ungi	-					•
7. have you felt calm and peaceful?	1	2	3	4	5	6
8. have you felt emotionally stable?	1	2	3	4	5	6
9. have you felt downhearted and blue?	1	2	3	4	5	6
10. were you able to relax without difficulty?	1	2	3	4	5	6
11. have you felt restless, fidgety, or impatient?	1	2	3	4	5	6
12. have you been moody, or brooded about things?	1	2	3	4	5	6
13. have you felt cheerful, light-hearted?	1	2	3	4	5	6
14. have you been in low or very low spirits?	1	2	3	4	5	6
15. were you a happy person?	1	2	3	4	5	6
16. did you feel you had nothing to look forward to?	1	2	3	4	5	6
17. have you felt so down in the dumps that nothing could cheer you up?	1	2	3	4	5	6
18. have you been anxious or worried?	1	2	3	4	5	6

ш
Ξ.
2
C
Ō
Щ
S.
Z
Ξ
_
$\overline{\mathbf{\omega}}$
v ,
Щ
Ľ
ш
ш
~
2
ш.

Please respond to each item by marking one box per row:

	Strongly Disagree	Disagree Neutral	Neutral	Agree	Strongly Agree
I tend to bounce back quickly after hard times					
I have a hard time making it through stressful events					
It does not take me long to recover from a stressful event					
It is hard for me to snap back when something bad happens					
I usually come through difficult times with little trouble					
I tend to take a long time to get over set-backs in my life					

GENERALIZED ANXIETY QUESTIONNAIRE

Over the last 2 weeks, how often have you been bothered by the following problems?	Not at all sure	Several days	Over half the days	Nearly every day
1. Feeling nervous, anxious, or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it's hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3
Add the score for each column	+	+	+	
Total Score (add your column scores) =				

Generalized Anxiety Disorder 7-item (GAD-7) scale

If you checked off any problems, how difficult have these made it for you to do your work, take care of things at home, or get along with other people?

Not difficult at all ______ Somewhat difficult _____ Very difficult _____ Extremely difficult _____

Western HealthSciences

VISUAL ANALOG SCALE - ANXIETY

"Please rate your level of anxiety level <u>at this moment in time</u>. The bottom of the scale signifies no anxiety whereas the top of the scale indicates extreme anxiety"

Extremely anxious	
s-1-2	
12 <u></u> 51	
10 <u></u> 10	
25 <u></u>	
12 <u></u> 13	
<u></u>	
77 <u></u> 78	
20 00	
10 <u>14</u> (3)	

82

Western HealthSciences

Medical Outcomes Study Questionnaire Short Form 36 Health Survey

This survey asks for your views about your health. This information will help keep track of how you feel and how well you are able to do your usual activities. Thank you for completing this survey! For each of the following questions, please circle the number that best describes your answer.

1. In general, would you say your health	
is:	
Excellent	1
Very good	2
Good	3
Fair	4
Poor	5
2. Compared to one year ago,	
Much better now than one year ago	1
Somewhat better now than one year ago	2
About the same	3
Somewhat worse now than one year ago	4
Much worse now than one year ago	5

3. The following items are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much? (Circle One Number on Each Line)

	Yes, Limited a Lot (1)	Yes, Limited a Little (2)	No, Not limited at All (3)
a. Vigorous activities, such as running, lifting	1	2	3
heavy objects, participating in strenuous sports			
b. Moderate activities , such as moving a table, pushing a vacuum cleaner, bowling, or playing golf	1	2	3
c. Lifting or carrying groceries	1	2	3
d. Climbing several flights of stairs	1	2	3
e. Climbing one flight of stairs	1	2	3
f. Bending, kneeling, or stooping	1	2	3

g. Walking more than a mile	1	2	3
h. Walking several blocks	1	2	3
i. Walking one block	1	2	3
j. Bathing or dressing yourself	1	2	3

4. During the **past 4 weeks**, have you had any of the following problems with your work or other regular daily activities **as a result of your physical health**? **(Circle One Number on Each Line)**

	Yes	No
	(1)	(2)
a. Cut down the amount of time you spent on work or other activities	1	2
b. Accomplished less than you would like	1	2
c. Were limited in the kind of work or other activities	1	2
d. Had difficulty performing the work or other activities (for example, it took extra effort)	1	2

5. During the **past 4 weeks**, have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

(Circle One Number on Each Line)

	Yes	No
a. Cut down the amount of time you spent on work or other	1	2
activities		
b. Accomplished less than you would like	1	2
c. Didn't do work or other activities as carefully as usual	1	2

6. During the past 4 weeks, to what extent has your physical	
health or emotional problems interfered with your normal	
social activities with family, friends, neighbors, or groups?	
Not at all	1
Slightly	2
Moderately	3
Quite a bit	4
Extremely	5

7. How much bodily pain have you had during the past 4	
weeks?	
None	1
Very mild	2
Mild	3
Moderate	4
Severe	5
Very severe	6
8. During the past 4 weeks, how much did pain interfere with	
your normal work (including both work outside the home and	
housework)?	
Not at all	1
A little bit	2
Moderately	3
Quite a bit	4
Extremely	5

These questions are about how you feel and how things have been with you **during the past 4 weeks**. For each question, please give the one answer that comes closest to the way you have been feeling. (**Circle One Number on Each Line**)

	All of the Time	Most of the Time	A Good Bit of the Time	Some of the Time	A Little of the Time	None of the Time
a. Did you feel full of pep?	1	2	3	4	5	6
b. Have you been a very nervous person?	1	2	3	4	5	6
c. Have you felt so down in the dumps that nothing could cheer you up?	1	2	3	4	5	6
d. Have you felt calm and peaceful?	1	2	3	4	5	6
e. Did you have a lot of energy?	1	2	3	4	5	6

9. How much of the time during the **past 4 weeks** . . .

	All of the Time	Most of the Time	A Good Bit of the Time	Some of the Time	A Little of the Time	None of the Time
f. Have you felt	1	2	3	4	5	6
downhearted and blue?						
g. Did you feel worn out?	1	2	3	4	5	6
h. Have you been a happy	1	2	3	4	5	6
person?						
i. Did you feel tired?	1	2	3	4	5	6

10. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives, etc.)? (Circle One Number)	
All of the time	1
Most of the time	2
Some of the time	3
A little of the time	4
None of the time	5

11. How TRUE or FALSE is each of the following statements for you. (Circle One Number on Each Line)

	Definitely True	Mostly True	Don't Know	Mostly False	Definitely False
a. I seem to get sick a little easier than other people	1	2	3	4	5
b. I am as healthy as anybody I know	1	2	3	4	5
c. I expect my health to get worse	1	2	3	4	5
d. My health is excellent	1	2	3	4	5

CURRICULUM VITAE

Kaitlyn Jacobs

EDUCATION

September 2016 – August 2018

M.Sc., School of Kinesiology, Integrative Physiology

Western University

Dissertation: pilot study: heart rate variability analysis and mental health outcomes in university female hockey players

Advisors: Dr. J. Kevin Shoemaker, Dave Humphreys, Dr. Craig Hall

September 2012 – June 2016

B.A. (Honors), School of Kinesiology

Western University

AWARDS & EDUCATIONAL ACCOMPLISHMENTS

2016 - 2017 2017 - 2018	Western Graduate Research Scholarship, Western University
2016 - 2017	Tornado Insulation Women's Hockey Award (\$2,000 for the academic year), Western University

PEER REVIEWED CONFERENCE ABSTRACTS

- Jacobs, K., Woehrle, E., Klassens, S., Smith, S., Knestch, R., Barker, A., Shoemaker, J.K. (2018). Sex differences in heart rate response to isometric handgrip exercise with concurrent contralateral forearm somatosensory stimulation.
- Woehrle, E., Jacobs, K.G., Shoemaker, J.K. (2018). Posture Modifies Neuro-Cardiac Heart Rate Responses at the Onset of Moderate Intensity Isometric Handgrip Exercise.

- Smith, S.O., Woehrle, E., Klassen, S.A., Jacobs, K.G., Knetsch, R., Shoemaker, J.K. (2018). Effects of Contralateral Forearm Somatosensory Stimulation on Heart Rate Responses to Isometric Hand Grip Exercise.
- Jacobs, K., Woehrle E., Humphreys, D., Hall, C., Shoemaker, J.K. (2017) Pilot Study: Heart rate variability and mental health outcomes in university female hockey players.

RELEVANT ACTIVITIES

Teaching:

2018	Teaching Assistant (Winter), 3 rd year anatomy of the human body: a description of systemic structure & function, Western University
2017	Teaching Assistant (Fall), 3 rd year laboratory in exercise physiology, Western University
2017	Guest Lecture: Undergraduate course in physical growth & motor development, "Social & Cultural Constraints in Motor Development". Instructor: Vincent Liardi
2017	Teaching Assistant (Winter), 3 rd year physical growth & motor development, Western University
2016	Teaching Assistant (Fall), 1 st year introduction to biomechanics, Western University