NUTRITION AND ATHLETIC PERFORMANCE: IMPLICATIONS OF HEART RATE VARIABILITY

by

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Abstract

The purpose of this review is to examine the role of heart rate variability (HRV) as a predictor of athletic readiness and performance and assess whether nutrition strategies can be implemented to create favorable HRV indices with the goal of improving athletic readiness and performance. The balance between training and recovery is crucial for reaching an optimal adaptation and avoiding overtraining, allowing for improved readiness to train and compete. The measurement of HRV is non-invasive and is used primarily to quantify physical and mental stress in athletes by monitoring the effects of the autonomic nervous system on the heart. Current data suggests a relationship between resting parasympathetic tone, via time and frequency domains, and athletic performance. Parasympathetic modulated HRV indices have been associated with performance metrics such as peripheral work capacity, aerobic power, running and sprint performance, swimming performance, weight lifting performance, anaerobic capacity, strength, and enhanced mental focus/skill execution. The use of nutrition to help enhance sports performance is becoming more common. Evidence-based sports nutrition provides fuel for training/competition, assists in maximizing training adaptations, enhances recovery, improves mental focus, and aids in injury prevention and recovery. The use of nutrition strategies to influence HRV is novel and current evidence is scarce in regards to nutritional effects on HRV, specifically in athletes. Current research suggests that achieving energy balance and decreasing body fat in overweight/obese individuals has positive effects on the vagal component of HRV indices. Proper hydration, fruit and vegetable intake, a moderate carbohydrate diet, omega-3 fatty acid supplementation/intake also seem to have positive effects on HRV indices. Certain individual supplements have been studied in regards to HRV including casein hydrolysate, amaranth oil, and bovine colostrum. Caffeine seems to have the opposite effect on HRV indices,

increasing sympathetic modulation while decreasing parasympathetic modulation. Much more research needs to be done in regard to potential nutritional influences on HRV so that sport dietitians feel confident in the methods currently used to assess athlete readiness and determining what types of nutrition strategies may be used to further improve the performance of an athlete.

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Chapter 1 - Introduction

Improving athletic performance is one of the principle goals of all coaches, strength coaches, athletic trainers, sports dietitians and sports scientists alike. The manner in which the athlete, and their cardiovascular system, responds to the stress of training has intrigued sports professionals for many years. The balance between training and recovery is crucial for reaching an optimal adaptation and avoiding overtraining. Many technological methods are emerging to allow proper monitoring of athlete recovery and readiness to perform. One prevalent method is to assess heart rate variability (HRV). The measurement of HRV is noninvasive, and is used to quantify physical and mental stress of an athlete by monitoring the effects of the autonomic nervous system on the heart[1].

The importance of nutrition as a modality for recovery and therefore also for increased readiness and performance in the stressed athlete is widely accepted.[2,3]. Nutrition provides fuel for training/competition[4], can assist in maximizing training adaptations[5], enhance recovery[6], improve mental focus[7], and aid in injury prevention and recovery[8]. To that end, there are increasing numbers of full-time sports dietitians in the collegiate and professional ranks to help programs assist their athletes meet their performance potential. With the emergence of interdisciplinary high performance models and the inclusion of sport scientists and sport science technologies, sport dietitians often must embrace the methods currently used to assess athlete readiness and determine what types of nutrition strategies may be used to further improve the performance of an athlete. Therefore, the purpose of this paper is to review the role of HRV as a predictor of athletic readiness and performance and assess whether nutrition strategies can be implemented to create favorable HRV indices with the goal of improving athletic readiness and performance.

The Importance of the Autonomic Nervous system in Heart Rate Variability

The Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology[9] defines heart rate variability as "the heart's ability to produce fluctuations in the beat-to-beat interval in response to different situations[9]." The cardiovascular system is predominantly governed by autonomic regulation through the activity of the parasympathetic and sympathetic branches of the autonomic nervous system (ANS). The parasympathetic nervous system is often used interchangeably with "vagal", because the parasympathetic influence on heart rate is mediated by the vagus nerve[9]. The control of heart rate is primarily determined by adjustments in ANS activity. Increased parasympathetic nervous activity decreases heart rate, decreases blood pressure, and decreases blood flow to skeletal muscle. In contrast, increased sympathetic nervous system activity, mediated by the release of epinephrine and norepinephrine from the adrenal medulla, increases heart rate, increases blood pressure, and increases blood flow to the working skeletal muscle[10].

Measurement of Heart Rate Variability

Heart rate variability indices can be obtained from the measurement of the ECG (electrocardiography) recordings. Oscillations in heart rate variability can be quantified using time and frequency domains, and non-linear measures[9]. Non-linear measures such as 1/f scaling of Fourier spectra[11], H scaling exponent[9], and Coarse Graining Spectral Analysis (CGSA) [12] are promising but are not commonly used in the literature or practice at this time due to the lack of validation in some measures[1]. Therefore, only time and frequency domains will be discussed in this review, as these are the most commonly used indices in literature and practice.

Time Domain

The simplest method to evaluate variability in heart rate is the time domain measures.[9] Each QRS complex in an ECG is detected and the so called normal-to-normal (NN) intervals are detected. NN intervals are the intervals between proximate QRS complexes that result from sinoatrial depolarization[9,13]. The most commonly used time domain variables, as defined by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology[9] include standard deviation (SD) of the NN interval (SDNN) [ms] over the recorded time interval (short-term and long-term), SD of the 5-minute mean NN interval (SDANN) [ms] over the entire recording, the square root of the mean squared successive differences between adjacent RR intervals rMSSD [ms] over the entire recording, and the percentage of successive interval differences larger than 50ms (pNN50) [%]computed over the entire recording[9,13]. Additional indices will be discussed in this review and can be seen in table 1.1 below. All of these time-domain measurements are suggested to estimate high frequency variations (from frequency domain) in heart rate and thus are highly correlated to high frequency power (HF)[9]. Essentially, a higher value of these components suggests a more dominant parasympathetic or "vagal" tone.

Table 1.1 Time-Domain Measures of HRV

Variable	Units	Description
SDNN	ms	Standard deviation of all NN intervals
SDANN	ms	Standard deviation of the averages of NN intervals in all 5 min segments of the entire recording
RMSSD	ms	The square root of the mean of the sum of the squares of differences between adjacent NN intervals
SDNN index	ms	Mean of the standard devnations of all NN intervals for all 5 min segments of the entire recording
SDSD	ms	Standard deviation of differences between adjacent NN intervals
NN50 count		Number of pairs of adjacent NN intervals differing by more than 50 ms in the entire recording.
		Three variants are possible counting all such NN interval pairs of only pairs in which the first or second interval is longer
pNN50	%	NN50 count divided by the total number of all NN intervals

Created from: Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J 1996, 17(3):358.

Frequency Domain

Power spectral density (PSD) analysis provides the primary information of how power distributes as a function of frequency[9]. Methods for the calculation of PSD may be classified as non-parametric and parametric. The method seen most frequently in the literature, due to simplicity and availability for application on computers, is the Fast Fourier Transform (FFT), a non-parametric method [9,13]. In regard to frequency domain measures, the power spectral density of HRV in short- term recordings (2-5 minutes) consists of very-low-frequency (VLF; <0.04 Hz), low-frequency (LF; 0.04 to 0.15 Hz), and high-frequency (HF; 0.15 to 0.4 Hz) oscillations[1]. Spectral analysis may also be measured over the long-term (24 hour period). VLF is generally thought to reflect vasomotor changes and possibly parasympathetic influences, but may not be meaningful in short-term recordings and therefore VLF is generally not interpreted or used in studies with short-term recordings[1,9,14]. Therefore, the two main frequency bands most commonly seen for short-term recordings and that will be discussed further are LF and HF, in both absolute and normalized terms. When normalized, these indices reflect the relative value of each power component in proportion to the total power minus the VLF component[9]. Therefore, normalization essentially controls for changes in total power, as they occur during physiological maneuvers such as postural changes that may occur during testing. It is well accepted that efferent vagal activity is a major contributor to the HF component[9,13]. The interpretation of the LF component, however, is more controversial. Some evidence suggests that LF is a marker of sympathetic modulation[15] whereas other evidence[9] suggests that it is more likely a parameter that includes both sympathetic and vagal influences. Consequently, the LF/HF ratio is considered by some investigators[1,9,13] to mirror sympatho/vagal balance or to reflect sympathetic modulations. Therefore, it is generally accepted that an increase in the LF/HF ratio suggests an increase in

sympathetic activity, via increases in LF power, decreases in HF power, or both[9]. Refer to table 1.2 for frequency domain measures and descriptions.

Table 1.2 Frequency-Domain Measures of HRV

Variable	Units	Description: Analysis of short-term recordings (5 min)	Frequency Range
5 min total power	ms^2	The variance of NN intervals over the temporal segment	Approximately ≤ 0.4 Hz
VLF	ms^2	Power in very low frequency range	≤ 0.04 Hz
LF	ms^2	Power in low frequency range	0.04-0.15 Hz
LF norm	n.u.	LF power in normalized units : LF/(Total Power-VLF) x 100	
HF	ms^2	Power in high frequency range	0.15-0.4 Hz
HF norm	n.u.	HF power in normalized units: HF/(Total Power-VLF) x 100	
LF/HF		Ratio LF [ms^2]/HF[ms^2]	
		Description: Analysis of entire 24 hour recording	
Total Power	ms^2	Variance of all NN intervals	Approximately ≤ 0.4 Hz
ULF	ms^2	Power in the ultra low frequency range	≤0.003 Hz
VLF	ms2	Power in the very low frequency range	0.003-0.04 Hz
LF	ms2	Power in the low frequency range	0.04-0.15 Hz
HF	ms2	Power in the high frequency range	0.15-0.04 Hz
a		Slope of the linear interpolation of the spectrum in a log-log scale	approximately ≤ 0.04Hz

Created from: Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J 1996, 17(3):360.

Autonomic Nervous System Response to Training

Physical training is interpreted by the body as a stress or a biological stimulus[16]. Acute and chronic physical exertion is considered a physiological stressor with significant influence on cardiac autonomic balance[17]. A prolonged reduction in parasympathetic modulation following acute and/or chronic exercise bouts has been associated with inadequate physical recovery[18]. Because of the effects of exercise training on the physiological systems of the body, in particular the nervous system and adrenal glands, the body responds during and after a training bout to maintain homeostasis[19]. Although physical training is considered a stress, repeated exposure to training stress causes positive adaptations that are usually associated with improved performance [20]. Parasympathetic tone appears to predominate at rest and the initial increase in heart rate at the onset of training is primarily due to parasympathetic withdrawal[21]. As intensity of exercise increases to more vigorous

levels, the sympathetic system is activated such that at maximal intensity it is the combination of parasympathetic withdrawal and sympathetic activation that contribute to the observed tachycardia [20]. It has been suggested that chronic endurance exercise training and/or elevated aerobic fitness leads to enhanced resting parasympathetic (vagal) modulation of the heart that can be detected by HRV [22]. However, recent evidence suggests that well trained athletes with a high training volume may exhibit reduced vagal and/or elevated sympathetic control of the heart. It has been reported[23] that world class rowers exhibited a shift towards elevated resting sympathetic modulation during a period of heavy training (near 100% intensity) [23]. Similarly, another study[24] found that an increase in an athlete's individualized training volume was associated with reductions in vagal and increases in sympathetic cardiac modulation at rest [24].

Chapter 2 - Heart Rate Variability as a Predictor of Athletic Performance

Resting parasympathetic power, as represented by a higher HRV, is closely related to the health status and well-being in humans[25-27], but it is not clear whether increased parasympathetic power results in better prepared athletes and improved athletic performance. Hedelin et al[28] investigated whether changes in muscle performance and aerobic power were associated with changes in HRV after a seven-month training period in regional and national elite cross country skiers and canoeists. They found that a shift of resting HRV indices towards the LF component, potentially a marker of sympathetic activity, was associated with a reduced muscle strength/peripheral work capacity, as measured by a shoulder extension-flexion test in an isokinetic dynamometer. The authors also noted that subjects who improved VO_2 max were characterized by consistently higher resting absolute HF power and total HRV [28], therefore suggesting a relationship between resting parasympathetic dominance and aerobic power. Similarly, Marocolo et al[29] were interested in the relationship between HRV as a predictor of aerobic power (VO_2 max). Eighteen high performance long distance runners and a group of 18 untrained controls were put through a field test to estimate VO_2 max. Results showed that VO_2 max correlated significantly ($p < 0.05$) with resting rMSSD ($r = 0.47$), a time domain component of HRV, suggesting a potential relationship between improved resting parasympathetic tone and aerobic power [29]. Similarly, Da Silva et al[30] found that national level endurance runners who were able to increase or even maintain the resting parasympathetic modulation of HRV were more likely to improve their running performance in a longitudinal study. Correlations qualitatively classified as "large" and "very large" between changes in VO_2 max and parasympathetic HRV indices (rMSSD and normalized HF) with changes in 5-km performance were observed [30].

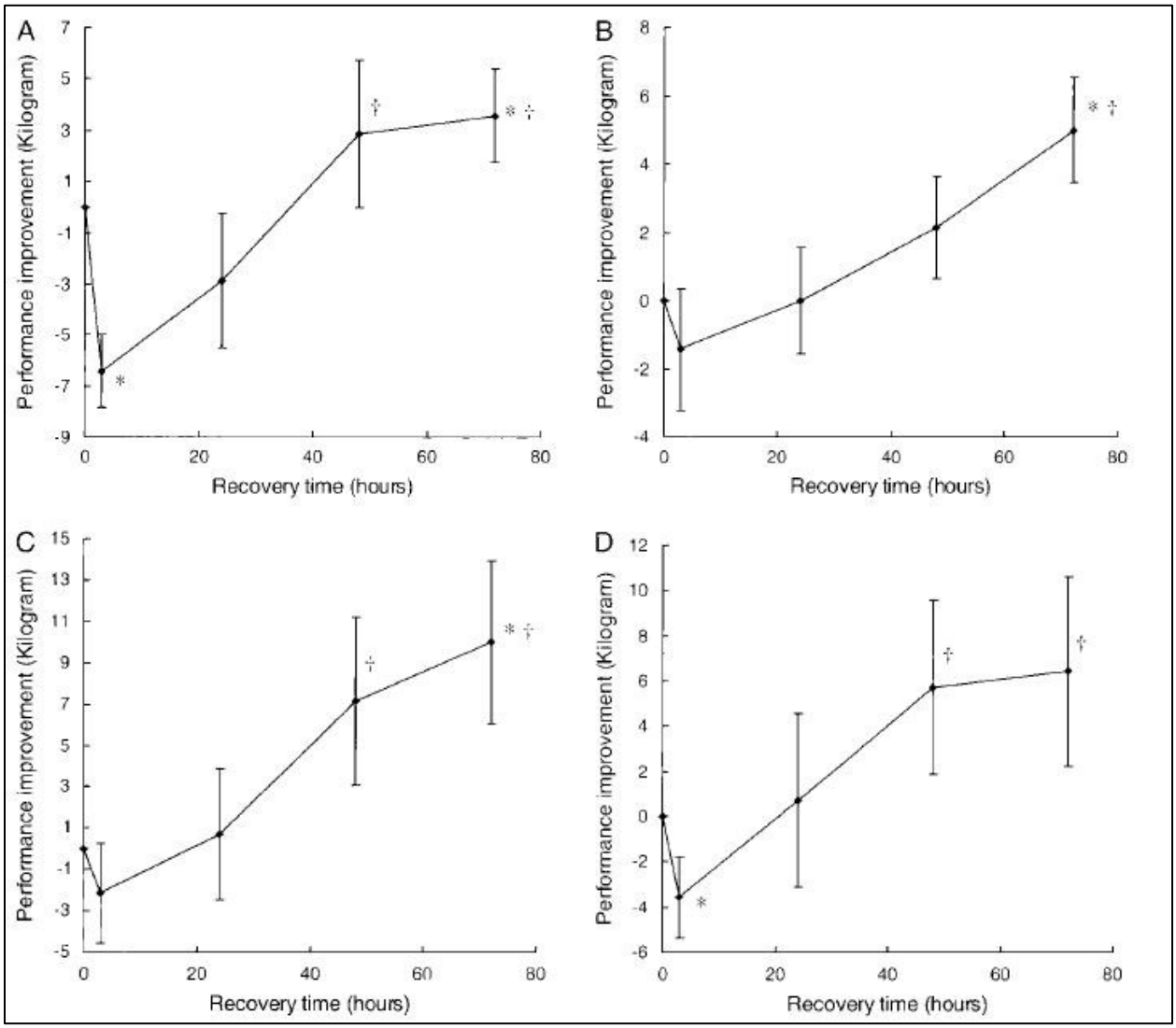
Garet et al[31] analyzed the association between individual changes in nocturnal ANS activity and changes in performance during a period of intensive training followed by a period of tapering in a group of seven regional level swimmers. They discovered a decrease in ANS activity during the intensive training period (decreased absolute HF power, SDNN, rMSSD, and pNN50) which was correlated with a loss in performance, and a rebound in ANS activity during tapering which was correlated with a gain in performance. The best maximal aerobic performance for a 400-m freestyle occurred when parasympathetic indices of HRV (absolute HF power, SDNN, rMSSD, and pNN50) were highest the night before a race [31]. Subsequently, Atlaoui et al[32] also sought out to determine the relationship between HRV changes in performance in 13 elite French swimmers competing nationally and internationally. Significant increases in normalized HF was correlated positively to swimming performance, defined as a competition of their best event ($r=0.81$; $p<0.01$) and negatively to fatigue ($r = -0.63$; $p < 0.03$) during a three week period of reduction in training load[32]. Conversely, the indices of sympathetic activity, normalized LF and LF/HF ratio, was inversely related to performance ($r = -0.81$; $p < 0.01$) [32]. In a similar study, Chalencon et al[33] discovered a clear relationship between swimming performance (400-meter freestyle time-trial) and nocturnal parasympathetic activity, measured as absolute HF power, in regional to national level simmers with mean intensive training history of 5.6 years [33]. The authors noted a high degree of model fit between measured performance ($R^2 = 0.84 \pm 0.14$, $p < 0.01$) and the measured and calculated HF power of the nocturnal ANS activity ($R^2 = 0.79 \pm 0.07$, $p < 0.01$)[33].

Maximal aerobic speed and 10 km running time have also been assessed with HRV indices. Buchheit et al[34] found a correlation ($r = 0.60$; $p < 0.01$) between changes in resting vagal-related indices and changes in maximal aerobic speed and 10 km running. In 11 of 14 moderately trained runners, those who decreased their 10 km running time > 0.5% over the course of an eight week

training period showed a progressively increasing trend towards resting vagal-related time domain indices (rMSSD) of HRV [34]. Oliveira et al[35] determined that both time domain indices (RRmean, rMSSD) and HF power improvements throughout a Brazilian National Division League futsal season correlated with improvements in high intensity intermittent running endurance and average repeated sprint ability times [35].

Very few studies have analyzed the relationship between HRV indices and weight lifting performance. Chen et al[36] is one of the first to determine whether HRV measures could reflect the recovery status and performance of weight lifters. Seven male weight lifters with > 6 years training history of national or international level competitions were put through a series of two hour weight training sessions after 10 days of detraining (including squat, seated bench press, deadlift, and front squat), which elicited approximately four-fold increases in circulating creatine kinase levels and protracted pain feeling. Weightlifting performance was represented by the maximal weight lifted on the previous four lifts from 3 attempts each. The participants were forced to recover in sedentary conditions for 72 hours. Assessments of HRV and weight lifting performance were performed before training, and 3, 24, 48 and 72 hours during recovery. Weightlifting performance was recorded 5 minutes after HRV assessments at each time point. After training, weightlifting performance of the subjects decreased below baseline in parallel with suppressed parasympathetic power (absolute HF), whereas sympathetic power, represented by normalized LF, was slightly elevated at three hours post training ($p < 0.05$). Weightlifting performance was gradually regained during the rest of the 72 hours in all subjects, and this trend paralleled a regain in absolute HF power and a decrease in normalized LF power (Figure 2.1 and 2.2). The data from this study suggests that parasympathetic power, indicated by resting absolute HF power, is able to reflect the recovery status and performance of weightlifters following an initial training bout [36].

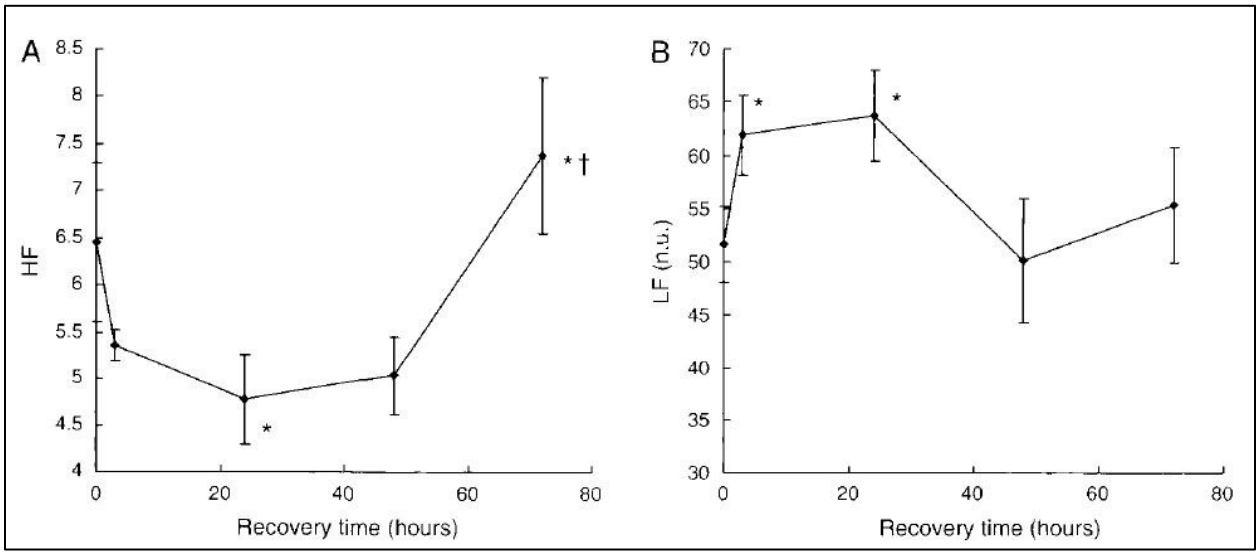
Figure 2.1 Weight Lifting Performance During Recovery



Maximal weight lifted from 3 attempts were recorded for 4 types of exercise: A) back squat, B) seated shoulder press, C) deadlift, and D) front squat. *Significant difference from pre; ǂSignificant difference from the bottom line (3 hour value) (N = 7, $p < 0.05$).

From: Chen JL, Yeh DP, Lee JP, et al. Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. J Strength Cond Res. 2011;25(6):1547. doi: 10.1519/JSC.0b013e3181da7858 [doi].

Figure 2.2 Heart Rate Variability During Recovery



Heart rate variability. A) High frequency reflects parasympathetic modulation; B) low frequency in normalized units (n.u.) reflects sympathetic modulation. *Significant difference from pre (N = 7, p < 0.05); ǂSignificant difference from the bottom line (3 hour value) (N = 7, p < 0.05).

From: Chen JL, Yeh DP, Lee JP, et al. Parasympathetic nervous activity mirrors recovery status in weightlifting performance after training. J Strength Cond Res. 2011;25(6):1549. doi: 10.1519/JSC.0b013e3181da7858 [doi].

A few studies have been conducted in Judo athletes specifically. First, Blasco-Lafarga et al[37] examined possible associations between measures of HRV and judo performance (represented by a judo test score) in 22 male judokas with 3-7 years of competitive experience. The judo test was aimed at reproducing both energy and neuromuscular demands of judo training and competition, requiring anaerobic and aerobic pathways, specifically with a focus on anaerobic capacity of the upper body [37]. Athletes with higher resting vagal modulation (higher absolute HF power) and lower sympathetic modulation (lower HR) before the test, were capable of increasing their heart rate to a greater extent during the judo test protocol. Change in heart rate during the test was correlated with a better performance score. Similarly, higher resting rMSSD was correlated with a better judo performance score, which may reinforce the idea that a higher baseline vagal balance would allow further usage of autonomic resources during training or competition [37]. The authors suggested that an ANS balance leaning to an increased vagal modulation during resting conditions may contribute to a higher capacity

to manage stress responses during training, and thus result in a better performance. Recently, Morales et al[38] randomly split a group of 14 male national level judo athletes in to two groups, high training load (HTL) and moderate training load (MTL). HRV measures, a recovery stress questionnaire, and strength measurements were administered. The HTL group showed lower resting rMSSD and HF power and a higher resting LF/HF ratio in the post-test vs. the pre-test ($p < 0.05$), while the MTL group did not show any significant changes. Judo athletes enrolled in the HTL program showed an imbalance of the autonomic nervous system with decreased vagal modulation, together with a decrease in strength parameters, higher markers of stress, and a lower perception of recovery [38].

Botek et al[39] assessed the change in athletic performance in response to HRV-adjusted training load in 10 national level athletes, including a decathlete and a heptathalete. The authors found that a high level of ANS activity, particularly resting vagal activity (represented by absolute HF power and rMSSD), accompanies an enhancement in performance changes over the course of the season. In contrast, deterioration in performance tended to be seen in those athletes who had the lowest resting vagal activity and highest indices of sympathetic modulation (represented by absolute LF power and LF/HF ratio). The authors concluded that high basal activity may indicate athletes have a higher training potential in terms of training adaptability and readiness to train [39].

Finally, a study conducted by D'Ascenzi et al[40] examined the changes in HRV indices in elite female volleyball players before a stressful match during play-offs to evaluate the impact on sport-specific performance. Resting HRV analysis was conducted in the morning one and two days prior to the match and the day of the competition. Performance measurements were evaluated by an experienced statistician and included volleyball service and reception, which are thought to be influenced by anxiety [40]. The authors found that the number of positive volleyball receptions was inversely correlated with the LF/HF ratio. That is, the higher the resting sympathetic activity

measured, the lower the number of positive receptions, suggesting a significant influence of ANS activity on sport-specific performance [40].

Not all studies suggest that a resting parasympathetic dominance is best when it comes to performance. Iellamo et al[23] looked at seven Italian junior national rowers over a whole season, and investigated the athletes three months apart on three subsequent occasions. The first at baseline, the second when the training load was 75% of maximum, and the third measurement at 100% training load. From baseline to 75% maximum, there was a progressive increase in absolute HF power and a decrease in absolute LF power. At 100% training load, about 20 days before the rowing world championship event, opposite changes were observed, with a significant decrease in absolute HF power and an increase in the absolute LF component, resulting in an enhanced sympathetic tone. Although the mechanisms were not examined, three out of the seven athletes won a medal (1 silver and 2 gold). The authors concluded that because of their observations, they believe that reducing vagal tone and increasing sympathetic tone leading in to a competition might serve to prepare the cardiovascular system to the rapid and wide, even anticipatory, variations in heart rate, cardiac output, blood flow redistribution, and muscle perfusion of highly demanding competitions [23]. Similarly, Manzi et al[24] observed eight recreational long distance runners leading up to a marathon and concluded that absolute LF oscillations in HRV at peak training load predicted athletic achievement. The increase in LF component of HRV was accompanied by a significant decrease in markers of vagal cardiac modulation, which were directly related to a longer competition time (decreased performance) [24].

Chapter 3 - Effects of Nutrition on Heart Rate Variability

Energy Balance

Energy balance is a critical principle when it comes to sports nutrition and athletic performance and it is well accepted that a positive energy balance is more advantageous than a negative energy balance in regards to athletic performance[41,42], although a chronically positive energy balance is most likely not ideal for most athletes. A few studies have looked at the relationship between energy balance and ANS activity. In a study looking at the different in HRV indices between normal weight (BMI 18.5-25) vs. underweight (BMI <18.5) males, total power, absolute LF power, and absolute HF power were reduced in underweight male subjects compared with normal weight subjects [43]. The impact of diet-induced weight loss on the cardiac autonomic nervous system on subjects with severe obesity was examined [44]. Subjects were put on a three-month weight loss program followed by a three-month reduced weight maintenance regimen. During maintenance, either a high (defined by authors) carbohydrate diet (60%) or a high fat diet (55%) of appropriate energy content for weight maintenance was prescribed. A 10% weight loss was achieved in both groups and these changes were associated with significant improvements in ANS cardiac modulation, represented by increased 24-hour absolute HF, rMSSD, and pNN50 indices. In regard to the macronutrient breakdown, high carbohydrate intake was associated with the better cardiovascular autonomic modulation, evidenced by increased parasympathetic modulation compared to what was observed in the high fat group [44].

De Jonge et al[45]examined the effects of different approaches of caloric restriction on ANS function. 28 healthy men and women were split in to four groups, a control group (weight maintenance), a caloric restriction by diet group, a caloric deficit by diet and exercise group, and a very low calorie diet until 15% weight reduction. Short-term resting HRV was measured at baseline,

three months, and six months. There was a decrease in sympathetic nervous system activity (absolute LF) and an increase in parasympathetic activity (absolute HF) in all weight loss groups, and this response was more pronounced with the caloric restriction plus exercise [45]. The results of this study suggest that a weight loss effort incorporating exercise and dietary modifications is more beneficial than one or the other when assessing HRV indices.

Subsequently, Krishna et al[46] evaluated the cardiac autonomic activity by measuring HRV in underweight and overweight young healthy adults (Table 3.1). Subjects were grouped according to BMI. A BMI of 18.5-24.9 signified normal weight, <18.5 underweight, and 25-30 overweight. Overweight subjects had higher LF in normalized units compared to normal weight group ($p < 0.05$). The overweight group had higher LF and lower HF power when expressed in absolute units than the normal weight with no statistical significance. Though not statistically significant, the LF/HF ratio was also higher in the overweight group, representing sympathetic dominance in overweight individuals. The underweight group had lower TP, LF, and HF than normal weight group with no statistical significance [46]. These results suggest that being overweight results in a more sympathetic dominant state whereas being underweight seems to have an opposite effect, with a seemingly decrease in the total power of the ANS.

Table 3.1 Comparison of HRV Spectral Power between the BMI Based Study Groups

Parameter	*UW (n=12)*	*NW (n=72)*	*OW (n=40)*
Total Power (ms^2)	1808.10±1225.82	2438.27±1720.02	2120.64±1784.50
LF (ms^2)	448.93±286.79	633.34±622.79	645.62±774.84
HF (ms^2)	618.75±617.93	932.33±830.02	678.71±835.07
LFnu	43.69±12.59	40.35±17.52	47.75±15.76*
HFnu	46.90±12.66	47.99±16.33	41.82±16.01
LF/HF	1.06±0.52	1.09±0.90	1.48±1.07
Heart Rate	86.24±8.70	80.35±9.90	83.17±8.88

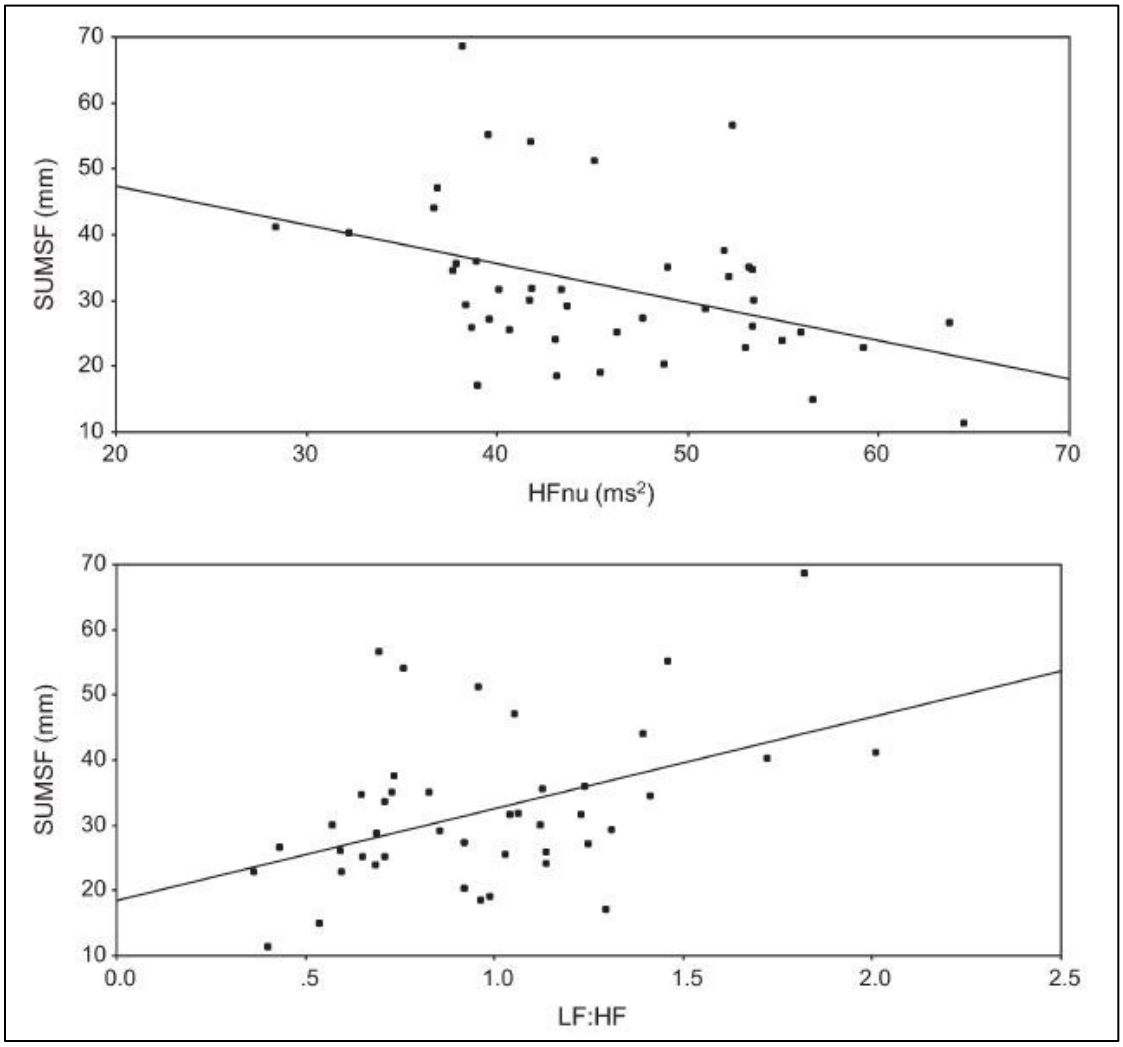
Values are mean ± SD.
*p<0.01 compared with NW group
UW= underweight; NW= normal weight; OW= overweight

From: Krishna P, Rao D, Navekar VV. Cardiac autonomic activity in overweight and underweight young adults. Indian J Physiol Pharmacol. 2013;57(2):148.

Body Composition

The body composition of an athlete can play a role in athletic performance. It has been suggested that decreased lean body mass and increased fat mass can put an athlete at increased risk of injury[47,48], and body composition has also been used as a predictor of athlete performance[49,50]. A couple of studies have also examined the association between body composition and HRV indices. Esco et al[51] sought out to determine if cardiovascular ANS modulation was related to aerobic fitness, BMI, waist circumference, and the sum of skinfold thickness across the chest, abdomen, and thigh (SUMSF) in 50 healthy young men. Results indicated a significant inverse relationship between resting normalized HF power and SUMSF ($r = 0.40$, $p < 0.01$). There was also a significant positive relationship between resting LF/HF ratio and SUMSF ($r = 0.44$, $p < 0.01$) (Figure 3.1). No other variable significantly correlated with either normalized HF or LF/HF [51]

Figure 3.1 Scatter Plots Displaying the Significant Independent Relationships between SUMSF (y-axis) and HRV Indices (x-axis)



SUMSF = the sum of skinfolds across the chest, abdomen, and thigh; HF_{nu} = normalized high frequency component of the heart rate variability spectrum (0.15-0.40 Hz), a marker of parasympathetic modulation; LF:HF = low frequency to high frequency ration, a heart rate variability marker that represents parasympathetic to sympathetic balance.

From: Esco MR, Williford HN, Olson MS. Skinfold thickness is related to cardiovascular autonomic control as assessed by heart rate variability and heart rate recovery. J Strength Cond Res. 2011;25(8):2307. doi: 10.1519/JSC.0b013e3181f90174 [doi].

Subsequently, Esco et al[52] did a follow up study to determine whether grouping subjects based on the sum of skinfold thickness (SF) would reflect differences in HRV following maximal exercise. Fifty-four healthy men were split in to two groups based on SF. Group 1 had a SF of 62.3 ± 9.5 while group 2 had a SF of 107.1 ± 26.9, meaning that group 2 had a much

higher total body fat as seen via skinfold measurements. HRV was evaluated pre, at 0-5 minutes post, and 25-30 post exercise. HF and LF/HF were significantly higher and lower, respectively, at pre and post in group 1 vs. group 2 ($p < 0.05$). Furthermore, there was a significant trend toward baseline in post-exercise HRV indices in group 1 ($p < 0.05$) but not in group 2 ($p > 0.05$). Similar to the 2011 study, this study suggests a greater SF is related to a delayed return of HRV toward baseline from maximal exercise, indicating a potential impairment or delay in recovery in overweight athletes. The association between SF and HRV was also found to be independent of VO2 max and BMI [52].

Hydration

Dehydration amounting to the loss of 3-4% body weight can adversely affect athletic performance and poses a risk to health[7,53]. Carter et al[54] wanted to determine the effect of hydration status on HRV before and after exercise induced heat stress [54]. Five healthy, moderately fit males completed two experimental trials in counterbalanced order. One group (hypohydration group) performed an intermittent treadmill and running test in a 40°C environment with 20% humidity, and drinking was restricted to produce a 4% loss of body mass. The other group (euhydration group) did the same but was administered water *ad libitum* to maintain body mass. The hypo hydration group showed significant reductions in total power of the ANS at rest and recovery from exercise heat stress, due to greater shifts towards parasympathetic influence (as evidenced by increased normalized HF), which may serve to limit the cardiovascular stress induced by dehydration[54]. However, fluctuations in normalized LF and HF power during recovery from exercise were blunted in the hypo hydration state, suggesting that hydration status contributes importantly to restoration of autonomic balance following exercise heat stress (Table 3.2). This suggests that dehydration alone positively influences

parasympathetic control of HRV but the reduction in overall HRV and the blunted oscillations in normalized LF and HF changes following exercise results in an overall negative effect of dehydration on autonomic cardiac balance. This was evidenced by the euhydrated group being able to do 11% (significant) more work than the dehydrated group during the treadmill test.

Table 3.2 Frequency Domain Indexes (normalized units) Before and After Exercise Heat Stress

	Pre-exercise	25 min post-exercise	35 min post-exercise	45 min post-exercise
LF, n.u.				
Euhydration	53.4 ± 24.5	79.9 ± 26.5	77.7 ± 25.4	68.3 ± 21.5
Hypohydration	39.2 ± 19.8	47.3 ± 19.8	50.1 ± 19.8	27.4 ± 14.7
P value	< 0.05	< 0.05	< 0.05	< 0.05
HF, n.u.				
Euhydration	42.1 ± 25.4	9.5 ± 4.9	16.1 ± 10.6	28.0 ± 18.9
Hypohydration	55.6 ± 16.5	48.5 ± 24.7	46.4 ± 15.4	64.7 ± 24.5
P value	< 0.05	< 0.01	< 0.05	< 0.05
LF:HF				
Euhydration	1.4 ± 0.6	8.40 ± 2.5	4.8 ± 1.5	2.5 ± 0.8
Hypohydration	0.7 ± 0.2	0.9 ± 0.3	1.1 ± 0.9	0.4 ± 0.5
P value	0.05	0.01	0.01	0.05
VLF, n.u.				
Euhydration	27.9 ± 16.5	36.9 ± 14.9	44.5 ± 21.4	25.7 ± 19.2
Hypohydration	19.6 ± 5.6	48.9 ± 25.6	29.0 ± 20.9	63.1 ± 36.5
P value	< 0.05	NS	< 0.05	< 0.05
TP ($m s^2$)				
Euhydration	3178 ± 265	2714 ± 394	2814 ± 212	2913 ± 231
Hypohydration	2278 ± 389	1889 ± 436	1963 ± 298	2016 ± 269
P value	< 0.01	< 0.01	< 0.05	< 0.05

LF, low frequency; HF, high frequency; VLF, very low frequency; TP; total power.

From: Carter III R, Cheuvront SN, Wray DW, Kolka MA, Stephenson LA, Sawka MN. The influence of hydration status on heart rate variability after exercise heat stress. J Therm Biol. 2005;30(7):498. doi: http://dx.doi.org/10.1016/j.jtherbio.2005.05.006.

Moreno et al[55] evaluated the effects of hydration protocols on ANS modulation in young, active, healthy males to assess linear indices of HRV in the young men with and without isotonic solution intake (Gatorade®). Subjects were split in to two groups, experimental and control. Both groups rested in a supine position for 10 minutes, followed by 90 minutes of exercise (60% of VO2 max) and 60 minutes of recovery. In the control group, subjects were not given any fluids to drink during the protocol. In the experimental group, the subjects were given the aforementioned isotonic solution based on body weight, which was administered in 10 equal

portions of regular intervals during the exercise until the end of recovery. Body weight loss in the control group was 2.0 ± -0.6%, vs. -0.2 ± 0.7% in the experimental group. Interactions between time and hydration state were observed in the LF and HF indices in normalized units, suggesting a better recovery in the hydrated protocol. Despite producing lower modifications in the HRV indices, the hydration protocol was insufficient in significantly influencing HRV indices during physical exercise in this study. However, during the recovery period it induced significant changes in the cardiac ANS modulation, promoting faster recovery of HRV indices, observed by increased rMSSD and normalized HF. The results of this study promotes the importance of hydration, specifically for athletes doing multiple bouts of exercise/competition per day [55].

Most recently, Castro-Sepulveda et al[56] compared the effects of dehydration and rehydration after exercise on HRV in college athletes. The athletes were split into a dehydration group and a rehydration group. Both groups performed a circuit of exercises in a room that was 32°C for 45 minutes. The dehydrated group rested with no fluids, while the rehydration group received a fluid intake equivalent to 150% of body mass loss. HRV was measured at baseline and 4 hours post-exercise in both groups. The dehydrated group showed significant changes in pNN50 (-44%), pNN20 (-27%) and rMSSD (-35%) and LF/HF ratio (+41%). This suggests that dehydration reduces HRV by reducing parasympathetic activity and increasing sympathetic activity [56]. No performance outcomes were discussed in this article.

Fruit and Vegetable Intake

High fruit and vegetable intake for athletes is widely accepted due to their antioxidant effects[57-59]. Park et al[60] examined whether high consumption of fruit and vegetables was associated with beneficial changes in HRV measures in a community-based, longitudinal study of a population of 586 participants, free of known chronic medical conditions. Dietary intake was evaluated with a self-administered semi-quantitative food-frequency questionnaire. Positive associations with normalized HF and inverse associations with normalized LF and LF/HF ratio suggest that high consumption of fruit and vegetables may enhance parasympathetic response and may reduce the sympathetic tone at rest. After controlling for potential confounders, intake of green leafy vegetables such as spinach and collard greens in particular was positively associated with normalized HF and inversely related with normalized LF power ($p < 0.05$). These significant associations were retained after further adjustment for healthy lifestyle factors, such as physical activity and use of multi-vitamins. Non-citrus fruit intake and total fruit and vegetable intake were borderline significantly associated with increasing trends in normalized HF and decreasing trends in normalized LF power, but there associations were no longer observed after controlling for physical activity and vitamin supplement use[60].

Subsequently, Dai et al[61] examined whether a Mediterranean diet was associated with HRV indices in adult twins, controlling for potential confounding effects of family and genetic influences. A Mediterranean diet in general contains high amounts of cereals, fruits, vegetables, nuts, legumes, and fish. A self-administered quantitative food questionnaire was used to collect dietary data over a period of 12 months. Participants were ranked on the similarity of their diet to the characteristics of the Mediterranean diet. Associations of diet and HRV were assessed by fitting linear regression models and were examined at two levels: between-subject and within-

pair. Higher scores (related to a diet closer to the meditarranean diet) were positively associated with a higher HRV (SDNN, rMSSD, pNN50; all $p < 0.05$)[61].

Carbohydrate Intake

High to moderate carbohydrate intake is generally accepted as beneficial for athletic performance[62-64]. There has been some research done on the effects of different carbohydrates on nervous system activity in rats[65], but not measuring HRV in humans specifically until more recently. Tentolouris et al[66] compared the effects of two different iso-energetic meals on ANS activity assessed by HRV in obese and lean young, healthy women. Subjects were examined on two occasions, after a carbohydrate rich meal (130g CHO) and a fat rich meal (52g fat). Spectral analysis of HRV was performed at baseline and every one hour for three hours after meals. After carbohydrate rich meals, there was a greater increase in LF/HF observed in lean, compared to obese women (1.21 ± 0.6 vs. 0.32 ± 0.06, $p = 0.04$), compared to the fat rich meal, which was comparable between lean and obese women. The LF/HF ratio increased progressively after the CHO rich meal in lean ($p = .04$) but not in obese subjects ($p = .33$). No significant changes from baseline values were observed after the fat-rich meal in either study group ($p = 0.80$ and $p = 0.46$, respectively. Increases in LF/HF after the carbohydrate rich meal was due to the combination of a significant increase in the power of LF and a decrease in the power of HF components of HRV ($p = 0.03$ and $p = 0.02$, respectively). These data suggest that meal content can affect HRV indices, especially in lean subjects [66]. In regard to interpretation for athletes, it seems as though there is a shift to sympathetic dominance after a carbohydrate rich meal, but this study was done in in-active subjects and there was no exercise involved. Therefore, it is unclear what the implications of this study would be in regards to post-training or post-competition nutrition, specifically when talking about carbohydrate intake.

Havemann et al[67] investigated the effect of a high fat diet followed by one day of carbohydrate loading versus a continuous high carbohydrate diet on HRV and cycling performance in eight well-trained cyclists in a randomized single-blind crossover study. There were no significant differences between trials for normalized supine HF or LF/HF but there was a tendency for the normalized LF power component of HRV to be greater in the high fat/carbohydrate load group, ($p = 0.056$), suggestive of increased sympathetic activation. Overall, time trial performance was not different between diets, however, 1km sprint power output after the high fat diet/carbohydrate load group was lower ($p < 0.05$) compared to the high carbohydrate diet [67], suggesting a potential benefit of carbohydrate intake in regards to power output.

Lima-Silva et al[68] sought to investigate the effects of short term (two days) low or high carbohydrate diet consumed after high volume and intensity exercise on ANS activity at rest, when compared with a control condition. Twelve healthy, physically active males were assigned to either a low carbohydrate (10%), high carbohydrate (80%) diet, or control (~55%) carbohydrate. Each group performed all trials with a washout period between. It was found that normalized HF and LF were lower and higher, respectively, in the low carbohydrate diet than in the control diet ($p < 0.05$). The mean values of normalized HF power decreased significantly after the low carbohydrate diet, compared with control. Similarly, the normalized LF power increased significantly after the low carbohydrate diet ($p < 0.05$) compared with control. Consequently, the LF/HF ratio was higher with the low carbohydrate diet than with the control diet ($p < 0.05$). The low carbohydrate diet resulted in increased sympathetic activity and a decrease in vagal activity, while the high carbohydrate diet had no significant effect on HRV. Interestingly, the control diet group had the best effect on HRV as seen below. This could

suggest that a moderate carbohydrate diet is best in regards to HRV indices and athletic performance versus extremes one way of the other, but there were no significant differences between the high carbohydrate group and the control group, suggesting either approach may be better than the low carbohydrate approach.[68]. Refer to table 3.3 for details in indices.

Table 3.3 Effects of Diet Regimens on the Time and Frequency Domains of HRV Measurements

HRV Measurements	Control Diet	Low-CHO diet	High-CHO diet
Time domain			
R-R intervals (ms)	899.0 ± 146.1	876.8 ± 115.8	878.7 ± 127.7
HR (beats·min^{-1})	69 ± 11	70 ± 8	70 ± 10
SDNN (ms)	73.9 ± 36.9	76.3 ± 32.1	66.1 ± 20.5
RMSSD (ms)	49.9 ± 30.8	42.3 ± 25.1	42.7 ± 21.2
pNN50 (%)	26.0 ± 23.3	19.0 ± 18.4	20.3 ± 17.9
Frequency domain			
Total poewr (In ms^2)	7.83 ± 0.96	7.90 ± 0.78	7.74 ± 1.00
HF (In ms^2)	6.35 ± 1.38	5.97 ± 1.08	5.98 ± 1.26
LF (In ms^2)	7.48 ± 0.90	7.72 ± 0.78	7.51 ± 0.99
HF (NU)	27 ± 11	17 ± 9*	20 ± 11
LF (NU)	73 ± 17	83 ± 9*	80 ± 11
LF/HF ratio	4.2 ± 3.2	7.2 ± 6.2*	6.2 ± 6.1

Note: Values are means ± SD. CHO, carbohydrate; HF, high frequency; HR, heart rate; LF, low frequency; pNN50, percentage of successive intervals differing more than 50 ms; RMSSD, root mean square of successive differences of R-R intervals; SDNN, standard deviation of R-R intervals; NU, normalized units.
*Significant differences between low-CHO diet and control ($p < 0.05$).

From: Lima-Silva AE, Bertuzzi R, Dalquano E, et al. Influence of high- and low-carbohydrate diet following glycogen-depleting exercise on heart rate variability and plasma catecholamines. Appl Physiol Nutr Metab. 2010;35(4):545. doi: 10.1139/H10-043 [doi].

Omega-3 Fatty Acids

Omega-3 fatty acid consumption and supplementation has become popular among the athletic population due to its anti-inflammatory effects[69,70], although there are currently no studies available determining the impact of athletic performance with omega-3 supplementation. Christensen et al[71] were among the first to examine correlations between blood cell fatty acid content of Eicosapentaenoic Acid (EPA) and Docosahexaenoic Acid (DHA) and HRV and to assess the effects of a dietary intervention of omega-3 fatty acids at different doses. Sixty healthy subjects were randomly assigned to three treatment groups in a double-blind study design. Subjects received a daily omega-3 fatty acid supplement (EPA + DHA) of either 6.6g, 2.0g, or placebo for 12 weeks. Before supplementation, there was a significant positive correlation found between DHA in granulocytes and all HRV indices in male subjects ($r = 0.50$, $p < 0.01$), while such a correlation was not found in women[71]. Dietary interventions revealed a dose-dependent effect of omega-3 fatty acids on time domain variables of HRV in men and women, although changes were not statistically significant[71] (Table 3.4).

Table 3.4 Heart Rate Variability Indices, The Content of Eicosapentaenoic Acid (EPA) and Docosahexaenoic Acid (DHA) in Cell Membranes in the 3 Treatment Groups (including both women and men) Before and After Dietary Supplementation for 12 wk with Placebo, 2.0g n-3 PUFA daily, or 6.6g n-3 PUFA Daily

	Placebo (n = 20)			2.0 g n−3 PUFA (n = 20)			6.6 g n−3 PUFA (n = 20)		
	Before	After	Difference[2]	Before	After	Difference[2]	Before	After	Difference[2]
Heart rate variability indexes									
RR (ms)	821 ± 105	812 ± 92	10 ± 94	802 ± 91	813 ± 90	−11 ± 54	788 ± 77	801 ± 88	−13 ± 66
SDNN (μs)	170 ± 43	157 ± 36	13 ± 33	164 ± 44	155 ± 38	10 ± 34	136 ± 27	136 ± 33	0 ± 27
SDNNindex (ms)	65 ± 15	65 ± 12	0 ± 11	65 ± 19	63 ± 18	2 ± 8	58 ± 20	59 ± 22	−1 ± 10
SDANNindex (ms)	163 ± 48	145 ± 43	18 ± 42	155 ± 44	144 ± 42	11 ± 39	125 ± 29	124 ± 34	1 ± 32
pNN50 (%)	13 ± 9	12 ± 6	1 ± 8	14 ± 11	12 ± 9	2 ± 5	9 ± 10	11 ± 10	−2 ± 7
RMSSD (ms)	35 ± 12	35 ± 8	0 ± 9	37 ± 15	34 ± 12	3 ± 6	30 ± 16	33 ± 15	−3 ± 9
n−3 PUFA in granulocytes (% of total fatty acids)									
EPA	0.51 ± 0.2	0.55 ± 0.3	−0.04 ± 0.3	0.56 ± 0.5	1.82 ± 0.8[3]	−1.24 ± 0.6[5]	0.58 ± 0.2	4.07 ± 1.0[3]	−3.49 ± 1.0[6]
DHA	1.54 ± 0.4	1.54 ± 0.5	−0.01 ± 0.4	1.48 ± 0.4	1.85 ± 0.6[3]	−0.36 ± 0.4[5]	1.58 ± 0.5	2.14 ± 0.4[3]	−0.56 ± 0.4[5]
n−3 PUFA in platelets (% of total fatty acids)									
EPA	0.73 ± 0.3	0.74 ± 0.4	−0.01 ± 0.2	0.72 ± 0.3	2.06 ± 0.6[3]	−1.33 ± 0.6[5]	0.85 ± 0.3	4.66 ± 1.3[3]	−3.81 ± 1.3[6]
DHA	2.45 ± 0.5	2.40 ± 0.6	0.05 ± 0.4	2.42 ± 0.4	2.81 ± 0.4[3]	−0.39 ± 0.3[5]	2.66 ± 0.5	3.57 ± 0.5[3]	−0.91 ± 0.4[6]

[2] Defined as before-after
[3,4] Significantly different from before supplementation (paired t test). [3] $p < 0.01$, [4] $p < 0.05$
[5] Significantly different from placebo, $p < 0.05$
[6] Significantly different from placebo and the 2.0-g n-3 PUFA group, $p < 0.05$ (one-way ANOVA)

From: Christensen JH, Christensen MS, Dyerberg J, Schmidt EB. Heart rate variability and fatty acid content of blood cell membranes: A dose-response study with n-3 fatty acids. Am J Clin Nutr. 1999;70(3):334.

Similarly, Mozaffarian et al[72] evaluated the associations between usual dietary consumption of fish and omega-3 fatty acids during the prior year in a population based cohort of US adults. After multi-variable adjustment, consumption of tuna or other fish was correlated with specific HRV indices, including indices suggesting greater vagal modulation (higher rMSSD ($p = 0.001$), higher normalized HF power ($p = 0.008$), and lower LF/HF ratio ($p = 0.03$) [72]. Conversely, Geelan et al[73] found that 3.5g of omega-3 fatty acid supplementation had no effects on short-term time and frequency domain HRV indices [73].

A randomized, double blind, placebo controlled, parallel dose-response supplementation trial of omega-3 fatty acids for 12 weeks in 67 adults showed that the LF/HF ratio of HRV decreases with increasing doses of omega 3 fatty acids ($r = -0.34$, $p = 0.023$), with 6g/d of total omega-3 fatty acids having the most beneficial effect [74]. The mean value of normalized HF

power significantly increased from baseline over 12 weeks while normalized LF power decreased significantly over 12 weeks in the 6g/d dosing group. The only other significant change to note over 12 weeks was the mean LF/HF ratio value in the 4g/d group. There were no other significant changes from baseline to 12 weeks in the 0g/d, 2g/d and 4g/d of fish oil supplementation groups, suggesting a dose-response relationship.

Finally, Xin et al[75] performed a meta-analysis of randomized controlled trials performed to investigate the influence of omega-3 fatty acids on HRV. Fifteen studies were included in the meta-analysis that included >18 year olds assigned to oral fish oil supplementation for > 1 month. HF indices were significantly increased by fish oil supplementation (SMD = 0.30, p = 0.005). SDNN, rMSSD were not significantly influenced by fish oil supplementation. The LF/HF ratio showed a trend of reduction (SMD = -0.22, p = 0.08), but was not significant. The treatment effect on LF/HF became significant (p = 0.01) when two studies with an omega 3 fatty acid dose <1000mg/d were omitted. The author concluded that short term fish oil supplementation may favorably influence the frequency domain of HRV, as indicated by enhanced vagal tone [75]. In regards to omega-3 fatty acids and HRV, intake or supplementation of 4g/d seems to be the minimum threshold in regards to significant positive changes in HRV indices, with the best responses seen as high as 6g/d.

Caffeine

Caffeine, present in many beverages and food including coffee, energy drinks, tea, and chocolate, is a popular performance enhancer[76]. Caffeine is a potent stimulator of sympathetic nerve activity, which exerts various effects on cardiovascular function including increased catecholamine release, elevated blood pressure, inhibited baroreflex function, prolonged QT interval, and depressed HRV[77,78]. Sondermeijer et al[79] determined the effect of acute caffeine

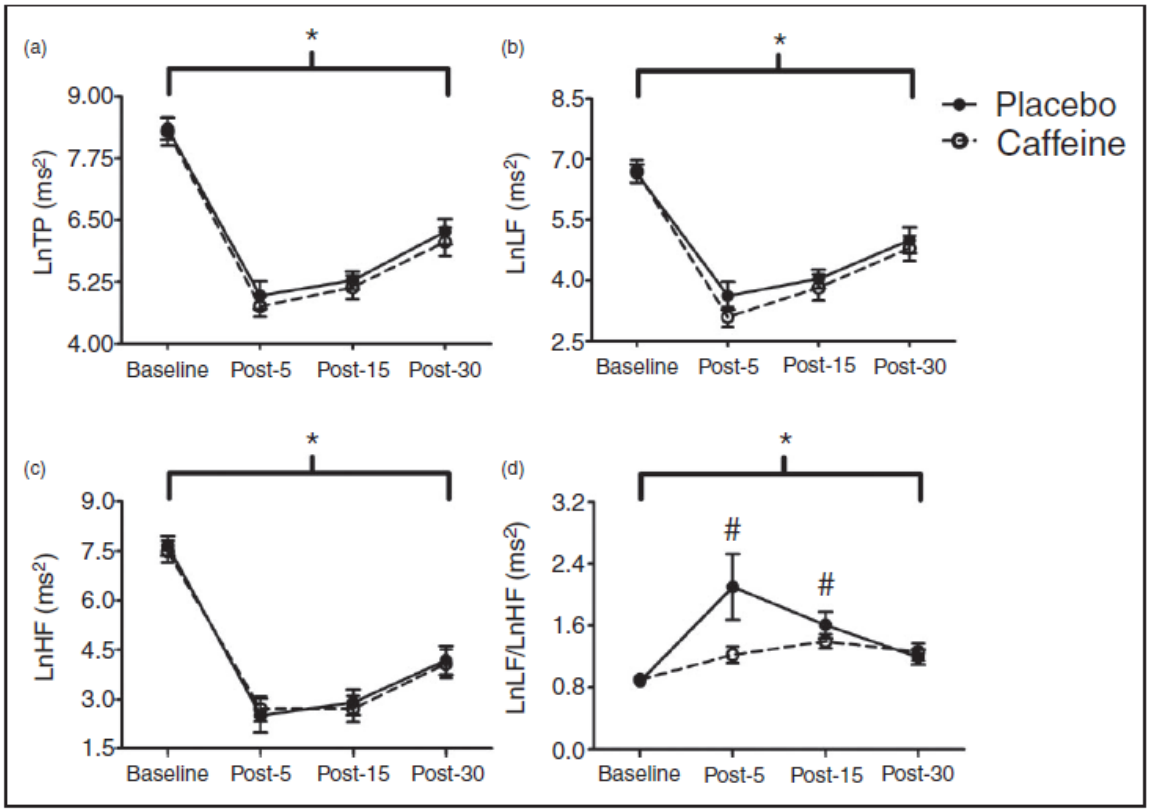
ingestion on parasympathetic nervous system activity in non-habitual caffeine users in a randomized, 3-way, single blind crossover study. Subjects either received single oral doses of placebo, 100mg, or 200mg caffeine dissolved in honey per day with a minimum 4-day washout period between trials. On each study day, short-term HRV was assessed before and after administration of the caffeine/placebo as well as during the period 60-90 minutes after ingestion. HRV measures of parasympathetic nervous system activity measured 60-90 minutes after ingestion were significantly decreased with caffeine compared to placebo, despite no significant differences in heart rate. The decrease in parasympathetic modudlation was reflected by the decrease in SDNN, rMSSD, pNN50, and absolute HF indices. Furthermore, the LF/HF power ratio was increased with high-dose caffeine compared with placebo, supporting a shift in sympathovagal balance towards sympathetic predominance [79].

Nishijima et al[80] investigated the effects of caffeine ingestion on the activities of the ANS during endurance exercise at low intensity using power spectral indices of HRV. Eight healthy, active men were given a placebo or caffeine (300mg) capsules randomly. Each subject ingested the samples 2 hours prior to cycling. HRV was monitored during rest and exercise. There were no significant differences between trials at rest, but the spectrum integrated values of absolute LF power and total power components in the caffeine trial were significantly greater than in the placebo trial during exercise. Absolute HF, LF, and TP in the caffeine trial were around double those of the placebo trial. The differences were significant at 25-30 min into exercise. Caffeine ingestion 2 hours prior to exercise enhanced the activity of the ANS during exercise at low intensity exercise [80].

Similarly, Bunsawat et al[81] investigated the effects of pre-exercise caffeine ingestion on autonomic recovery using analyses of HRV following an acute bout of dynamic exercise in a

randomized crossover study. Baseline measurements of HRV were taken in 18 healthy individuals followed by 400mg caffeine or placebo pill before 45 minute treadmill exercise protocol. Participants were then moved to a supine position and HRV was assessed again. Data were collected at 5, 15, and 30 minutes post exercise (Figure 3.2). Normalized LF and HF components declined similarly from baseline in both trials ($p < 0.05$). LF/HF ratio increased from baseline in both trials, but was lower in the caffeine trial than in the placebo trial during 5 and 15 minutes post-exercise. The lower LF/HF ratio in the caffeine trial was caused by a reduced normalized LF without a change in normalized HF. Therefore, the increase in sympathetic nerve activity would actually present as a reduction in LF, such as seen in the results. LF/HF was increased compared with baseline at all-time points in both trials ($p < 0.05$), with less of an increase during 5 and 15 minutes post-exercise in the caffeine trial ($p < 0.05$). This data may suggest that caffeine ingestion disrupts post-exercise autonomic recovery due to increased sympathetic nerve activity [81] and a delayed parasympathetic modulation. The prolonged sympathetic recovery time could be important in athletes completing multiple bouts of competition/training. The main limitation of this study is that there was not another exercise bout performed after the initial bout, so the effects of this prolonged sympathetic recovery time is unclear in regards to actual performance.

Figure 3.2 Natural Log Transformation (L_n) of Frequency Domain HRV Variables during Placebo and Caffeine Trials



Natural log transformation (L_n) of frequency domain heart rate variability (HRV) variables at baseline, 5, 15, and 30 minutes post-exercise during placebo (solid line) and caffeine (dash line) trials. Values are mean ± SE. *$p < 0.05$, different from baseline for both the placebo and caffeine trials; #$p < 0.05$, different from the caffeine trial within the same time point.

From: Bunsawat K, White DW, Kappus RM, Baynard T. Caffeine delays autonomic recovery following acute exercise. Eur J Prev Cardiol. 2014. doi: 2047487314554867 [pii].

Additional Supplements

Nakamura et al[82] examined the influences of the oral ingestion of casein hydrolysate from bovine milk at rest. Subjects were 11 healthy males. Subjects were given a casein hydrolysate drink (H) or a maltitol drink (C), a carbohydrate, in a crossover study. Just before and one hour after ingestion, HRV was measured. After H ingestion, LF/HF was significantly lower than before ingestion ($p < 0.05$) and absolute HF was significantly higher ($p < 0.05$). For the C group,

intake had no significant effect on either LF/HF or HF. This study suggests that ingestion of casein hydrolysate might decrease sympathetic activity and increase parasympathetic activity [82]. However, this is only a single study with a small sample size.

Amaranth oil is derived from the seeds of Amaranthus Cruentus L. It has a high content of linoleic acid (omega-6 fatty acid), and several other minor components, such as squalene, tocotrienols, tocopherols, and phytosterins[83]. The presence of conjugated double bonds allows this substance to act simultaneously as an active modulator of free radical reactions and as an inducer of antioxidant defense[83]. This could potentially be important for athletes because mild and reversible disturbances of oxidative homeostasis are necessary for the maintenance of the optimal pro/antioxidant balance and the overall oxygen metabolism[83]. Yelisyeyeva et al[83] studied the effects of amaranth oil (AO) supplementation on aerobic metabolism and HRV in athletes and in patients with type II diabetes. Both groups, including twenty-four national level athletes were studied over 22 days. All investigations were performed on the first day and at the end of the study in the morning before a meal. On a daily basis, 1 mL of concentrated AO was administered orally in the evening at least two hours after a meal for 22 days. HRV measures were at rest in the morning not less than 24 hours after the last training bout. Significant increases in SDNN, pNN50, absolute total power, and absolute LF were found. Increases in absolute HF were also found but they were not statistically significant. There was no change in LF/HF [83]. Again, this is a single study and the sample size is small, and there was not a healthy control to compare results to.

Bovine colostrum is a concentrated source of immune and growth factors. Shing et al[84] examined the influence of bovine colostrum protein concentrate (CPC) supplementation on HRV and salivary hormones over consecutive days of competitive cycling in 10 highly trained

male road cyclists in a randomized, double-blind, placebo controlled trial. Subjects were randomly assigned to a control (10g whey protein concentrate/day) or bovine CPC group (10g bovine CPC/day). Cyclists provided a baseline saliva sample before beginning 8 weeks of supplementation (analyzed for cortisol, testosterone, and IgA concentrations) and competing in a five day competitive cycling race. Cyclists provided saliva samples and measured HRV each day of the race in the morning. There was no significant difference between groups for macro or micro nutrient intake during the race period. In addition, training kilometers were not significantly different between groups for each week from baseline to start of the cycle race and no significant differences were seen between groups across the cycle race for time to complete each stage, kilometers raced, or sleep quality ($p > 0.05$). There were no significant differences between groups in absolute LF or HF across race period ($p > 0.05$), however, rMSSD increased over the cycle race in the bovine CPC group while these indices were decreased in the control group. The bovine CPC supplementation was associated with increased morning cortisol concentration on the first day of racing when compared to the control group ($p = 0.004$) and CPC supplementation prevented a drop in testosterone concentration over the race period ($p < 0.05$) [84]. There were no performance results noted by the authors for either group.

Chapter 4 - Conclusion

The manner in which the athlete, and their cardiovascular system, responds to the stress of training has intrigued sports professionals for many years. The measurement of HRV is a noninvasive method used to quantify physical and mental stress of an athlete by monitoring the effects of the autonomic nervous system on the heart[1]. Current data suggests a positive relationship between resting parasympathetic tone, via time and frequency domains, and athletic performance. Parasympathetic modulated HRV indices have been associated with performance metrics such as peripheral work capacity[28], aerobic power[28-30], running and sprint performance[35,85,86] , swimming performance[31-33], weight lifting performance[36], anaerobic capacity[37], strength[38], and enhanced mental focus/skill execution[40]. A proposed mechanism would be that a sympathovagal balance leaning to vagal dominance during resting conditions may contribute to a higher capacity to manage stress responses, and thus a better athletic performance. A higher baseline vagal modulation may allow further usage of autonomic resources during exercise by adapting to different situations more efficiently.

There are increasing numbers of full-time sports dietitians in the collegiate and professional ranks to help programs assist athletes to meet their performance potential. With the emergence of interdisciplinary high performance models and the inclusion of sport scientists and sport science technologies, sport dietitians often embrace the methods currently used to assess athlete readiness and determine what types of nutrition strategies may be used to further improve the performance of an athlete. The use of nutrition strategies to influence HRV is novel and current data are scarce in regard to the effects of nutrition on HRV, specifically in athletes. Current research suggests that achieving energy balance[43-46] and decreasing body fat[51,52] in overweight/obese individuals has positive effects on the vagal component of HRV indices.

Proper hydration[54-56], fruit and vegetable intake[60,61], a moderate carbohydrate diet[68], omega-3 fatty acid supplementation/intake[71,72,74,75,87] also seem to have positive effects on HRV indices, for both the time and frequency domains. Certain supplements have been studied in regard to HRV including casein hydrolysate[82], amaranth oil[83], and bovine colostrum[84] and show promising results, although there are only single studies and sample sizes are small. Caffeine seems to have the opposite effect on HRV indices, increasing sympathetic modulation while decreasing parasympathetic modulation[79-81].

One limitation to this review is the current scarce use of HRV technologies in athletics. Anecdotally, the primary barriers seem to be cost and the current knowledge base of professionals. Therefore, it is no surprise that there is inadequate data in regards to nutrition and HRV, specifically in athletes.

There are many limitations in the methodology of measuring HRV. For example, recordings can be conducted both long-term (24 hours) and short-term (5-10 minutes). Most of the HRV recordings done in the research previously discussed were done in short-term recordings, whereas long-term recordings seem to be more stable and free of placebo effects[9], and is usually deemed as ideal when assessing intervention therapies[9]. Both frequency-domain and time-domain indices were measured in the short-term in some studies but it is generally accepted that frequency-domain methods are preferred when investigating short-term recordings[9]. Long-term recordings analyzed by the time-domain methods should contain at least 18 hours of analyzable ECG data that includes the whole night, due to long-term HRV values being contributed to day-night differences[9]. The actual equipment and technology used to assess HRV also varied among studies which makes comparisons from study to study very difficult. Also, some measurements were done in the morning at rest, while others were done at different times throughout the day, some post-exercise or post-meal, which could affect results

of the measurement. The body position in which HRV is measured is also of concern. Some studies put subjects in a supine position while others are in a standing position. HRV is also sensitive to changes in respiration[88], which may have varied across studies and could adversely affect results. In regard to nutrition, much more research needs to be done, specifically in athletic populations. Most of the research in the nutrition section was done in older adults and non-athletes, although all subjects were deemed healthy. The samples sizes for most studies were also rather small and there was almost nothing related to performance outcomes, even in studies where exercise was examined. Therefore, it is very difficult to generalize the nutrition effects of HRV to athletes at this time.

There is a solid base of evidence that HRV may be used as a valuable tool to assess athlete recovery and readiness. The use of HRV could potentially help sport professionals plan their training programs more efficiently due to their increased ability to monitor their athletes' physical and mental stress. The use of HRV to monitor stress and the potential use of nutrition to induce positive effects of HRV brings sports professionals one step closer to being able to create individualized training and nutrition protocols for athletes to enhance athletic performance.

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