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## Risk of Low Energy Availability in Elite Male Athletes with Spinal Cord Injury

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RISK OF LOW ENERGY AVAILABILITY IN ELITE  
MALE ATHLETES WITH SPINAL CORD INJURY

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A Thesis

Presented to

The Graduate Faculty

Central Washington University

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In Partial Fulfillment

of the Requirements for the Degree

Master of Science

Nutrition

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by

Savannah Jane Glasgow

June 2020

CENTRAL WASHINGTON UNIVERSITY

Graduate Studies

We hereby approve the thesis of

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## ABSTRACT

### RISK OF LOW ENERGY AVAILABILITY IN ELITE MALE ATHLETES WITH SPINAL CORD INJURY

by

Savannah Jane Glasgow

June 2020

Recent research examining male able-bodied athletes has suggested that males are at risk for low energy availability (LEA); however, there is a paucity of research pertaining to prevalence amongst male athletes with an impairment. Therefore, the purpose of this study was to examine the risk of LEA and prevalence of symptoms related to Relative Energy Deficiency in Sport (RED-S), including risk of disordered eating, hormonal profiles, and bone mineral density (BMD) scans, in male wheelchair para athletes. Collegiate- and national-level male athletes ( $n = 9$ ) with spinal cord injury (SCI) completed 7-day concurrent diet and training logs, Eating Disorder Examination Questionnaire (EDE-Q), Dual Energy X-Ray Absorptiometry (DXA) scans, and hormonal blood spot testing. Zero (0) athletes were considered “at-risk” for LEA via EDE-Q score. Useable data to calculate energy availability (EA) was only available for 4 athletes, none of which presented with LEA (using a threshold of  $\leq 25 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$ ). Testosterone was low in 100% of athletes (mean:  $7.9 \pm 2.3 \text{ nmol/L}$ ). 25% of subjects had clinically low hip Z-scores (Z-score  $< -2$ ), and 63% of subjects were at an increased risk for fracture (Z-score  $< -1$ ). Based on the EDE-Q and calculated EA, the risk for LEA appears to be low; however, the risk for LEA appears to be high based on

DXA scans and hormonal profiles. These results are consistent with literature examining LEA in able-bodied athletes, which shows calculated EA to be a poor field assessment tool for LEA. These results also underscore the need for additional research to establish para athlete-specific thresholds for LEA, assessments for disordered eating, and normal clinical hormone ranges.

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## TABLE OF CONTENTS

Chapter		Page
I	INTRODUCTION.....	1
II	LITERATURE REVIEW.....	5
	Introduction.....	5
	Energy Intake.....	6
	Energy Expenditure .....	7
	Energy Availability.....	9
	Eating Attitudes and Behaviors .....	12
	Hormone Function .....	14
	Bone Mineral Density.....	17
	Conclusion .....	18
III	METHODS.....	20
	Participants.....	20
	Study Design.....	20
	Energy Availability.....	21
	Eating Attitudes and Behaviors .....	21
	Bone Mineral Density and Anthropometrics .....	23
	Hormone Function .....	23
	Statistical Analysis.....	24
IV	RESULTS.....	25
V	DISCUSSION.....	28
VI	CONCLUSION .....	33
	REFERENCES.....	34

## LIST OF TABLES

Table		Page
1	Method for assessing EA based on diet and training logs.....	22
2	Descriptive characteristics of participants ( $n = 9$ ).....	25
3	Risk factors related to RED-S .....	25
4	Calculated EA per day and average EA for the week .....	26



## I.

### INTRODUCTION

Until recently, research examining the prevalence of low energy availability (LEA) has focused extensively on females via the *Female Athlete Triad* (Triad). [1] As the body of research on the Triad grew, it became clear that the three conditions of the Triad, menstrual dysfunction, impaired bone health, and LEA, do not occur independently from one another. Rather, chronic LEA is the underlying etiology of the other two conditions, and also leads to several other physiological health impairments. [2] In addition, recent research suggests male athletes also experience negative health effects due to LEA. [3] Following these recent advances in the literature, the International Olympic Committee published a consensus statement which expanded the Triad into the more comprehensive syndrome known as *Relative Energy Deficiency in Sport* (RED-S). [2] RED-S is a collection of ten physiological impairments that stem from LEA, including disrupted endocrine function, immune function, metabolism, and cardiovascular health, among others. [4]

Energy availability (EA) represents the amount of energy available for normal health and physiological function (e.g. regular menses, endocrine production, bone remodeling, etc.) after energy expended during exercise is accounted for. [4] To determine whether or not an athlete has LEA, EA is calculated by subtracting the energy expended during exercise (EEE) from the athlete's total energy intake (EI). This is then divided by the athlete's fat free mass (FFM) in kilograms (kg). [4]

$$EA = \frac{(EI) \text{ (kcal)} - (EEE) \text{ (kcal)}}{(FFM) \text{ (kg)}}$$

Literature suggests a minimum EA of 45 kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> is required for optimal health in able-bodied female athletes, with LEA defined as 30 kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> or less [5-8]; these values should be applied to all populations with caution, as there can be significant variability and discrepancy when assessing an individual's energy intake and expenditure. [9] A threshold for which LEA occurs in male able-bodied athletes has yet to be established, although some research suggests the threshold may be closer to 20-25 kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>. [3,10] Similarly, there is not an established LEA threshold for para athletes of either sex, in part due to the significant discrepancies in energy expenditure between individual para athletes related to the nature of their impairment. [11,12] LEA may impair sport performance, but more importantly the body will not have sufficient energy to carry out normal physiological functions. [4,6,13] Health impairments such as decreased levels of testosterone (TES) and insulin-like growth factor-1 (IGF-1) associated with LEA have subsequently been linked to impaired bone health in male endurance athletes. [3,6,13,14]

Para athletes display variability in body composition, mobility, bone health, and metabolic and neurological function, all which can significantly impact the athlete's energy requirements. [11,12] The heterogenous nature of this population makes it difficult to determine a generalized LEA threshold, but these differences (*between* able-bodied and para-athletes, and *within* the para-athlete population due to injury-related factors) underscore the importance of this research in order to provide safe and effective

nutrition recommendations unique to the para athlete. [11,12,15] Athletes with spinal cord injury (SCI) generally expend 25-75% less energy than that of an able-bodied athlete, depending on the level of lesion and functional level of the SCI athlete. [11] The decreased energy expenditure can, in part, be explained by muscle atrophy below the lesion of injury related to decreased sympathetic nervous system function and/or impaired motor function. [16,17] A consequence of this overall decrease in energy expenditure is athletes with SCI are at a greater risk for developing overweight or obesity compared to able-bodied athletes, [16] so it is not uncommon for elite-level athletes with SCI to restrict their energy intake in order to control body weight. [12,18] Athletes with SCI may restrict eating around training to limit gastrointestinal (GI) discomfort during activity related to impaired gastric emptying or pressure related to position in their wheelchair; they may also have chewing or swallowing difficulties or food aversions related to their SCI. [11,19] These deliberate and unintentional restrictions of energy intake may put athletes with SCI at an increased risk for LEA. [19] Additionally, while para athletes exhibit better bone health than their sedentary counterparts, [20] individuals with SCI display a greater risk for fracture than able-bodied individuals, [21] in part due to decreased skeletal loading. [11,17] Therefore, LEA in athletes with SCI could further increase their risk of bone injury.

While early research has shown the potential of LEA to negatively impact hormone balance and bone mineral density in male athletes with SCI, there is a lack of research pertaining to the prevalence of LEA risk factors among this athlete population. [11] There has been a growing participation and public interest in elite-level para sports

over the last decade, [22] and this increased participation in sport warrants this research in order to develop appropriate assessment and treatment protocols specific to this population. [11] Therefore, the purpose of this study was to examine the prevalence of symptoms related to RED-S in collegiate and national-level male para-athletes, including risk of LEA, hormonal profiles, and bone mineral density (BMD) scans.

## II.

### LITERATURE REVIEW

#### *Introduction*

In 2014, the International Olympic Committee published a consensus statement introducing the syndrome *Relative Energy Deficiency in Sport* (RED-S). [2] RED-S, an expansion of the previously defined *Female Athlete Triad* (Triad), [1,2] is a collection of ten health consequences that stem from chronic low energy availability (LEA). These health consequences include impaired bone health, altered endocrine function, poor immune function, compromised gastrointestinal (GI) health, and irregular menstrual function in female athletes, among others. RED-S was developed as a more comprehensive and inclusive condition to describe the collection of negative health outcomes seen in all types of athletes who experience chronic LEA; its introduction highlighted a lack of research for several groups of athletes, such as male athletes and para athletes, regarding the consequences of under fueling. [4]

Over the last decade, there has been a growing participation and public interest in elite-level para sports. The Rio 2016 Paralympic Games hosted over 4,300 athletes in 22 different sports, the most in Paralympic Games history; the upcoming Tokyo 2020 Paralympic Games will also feature 22 sports, and has already had record-breaking interest in tickets for the Games. [22] With this increased participation in sport comes a need for sports nutrition recommendations specific to individuals with spinal cord injury (SCI). As it stands, research on proper nutrition practices for para athletes has not kept up with the growth of participation in sport. There is currently very little primary research

related to LEA in male athletes with SCI. A review of literature was conducted between March and May of 2020. Terms used to search *PubMed* included RED-S, LEA, energy availability (EA), para athlete, SCI, bone mineral density (BMD), Paralympic athlete, and energy intake (EI). Due to the limited number of articles on male para athletes specifically, some studies and reviews on female para athletes, able bodied athletes of both sexes, and non-athlete individuals with SCI were also included.

### *Energy Intake*

In order to assess EA, daily EI must first be determined. There are many difficulties associated with accurately assessing EI. A common method for estimating EI in free-living athletes is through self-reported, prospective diet logs. [9,23-26] Diet logs are inexpensive to use and require minimal equipment. They may be kept during formal training camps or during the athlete's normal home environment. While training camps can introduce dietary behaviors that deviate from an athlete's normal routine, research has suggested this difference is minimal. [23] In one study, researchers analyzed self-reported diet logs kept by 32 elite Canadian para athletes. [23] The athletes recorded their diets for 3 consecutive days while at a training camp, followed by 3 consecutive days in their normal home environment. The researchers found no significant difference between diets reported at the training camp compared to diets at home.

There are tradeoffs for the simple design of prospective diet logs. Self-reported logs place a significant burden on the athlete to complete, and the records often fail to provide an accurate portrayal of an athlete's true long-term energy intake. [9] Users may underreport their intake, inaccurately measure portions, or deviate from their normal

behavior as a result of the awareness that tracking brings. Errors may also be introduced during the analysis of the diet, which can depend on the analysis software used and the researcher's interpretation of the logs. [9]

### *Energy Expenditure*

Energy expenditure must also be assessed to evaluate EA. It has been reported that basal metabolic rate (BMR), the largest contributor to total daily energy expenditure (TDEE), is 14-27% lower in individuals with SCI compared to matched, able-bodied individuals. [15,16,27] This is primarily related to a decrease in activity from the sympathetic nervous system, which causes a loss of muscle mass below the level of lesion. [12,28] Athletes with SCI do have a greater energy expenditure compared to sedentary counterparts as a result of increased physical activity, but studies have suggested para athletes still expend 25-75% less energy than matched, able-bodied athletes. [12,28] Some research, however, has reported similar resting energy expenditure (REE) between para athletes and able-bodied athletes. Pelly et al. [29] compared REE between 7 male athletes with SCI and 7 athletic able-bodied controls, matched for age, body mass, and stature. The researchers reported no significant difference in REE between the para athletes and controls. This demonstrates the potential for sport participation to attenuate the expected decrease in energy expenditure seen in individuals following SCI.

Several studies have evaluated the accuracy of using prediction equations to estimate energy expenditure in individuals with SCI. [27,29-32] Chun et al. [30] used a regression equation to attempt to develop a prediction equation specifically for

individuals with SCI, however this equation has not been validated and has not been widely used in subsequent research. These researchers concluded the Cunningham prediction equation [33] provided the most accurate estimation of BMR when compared to BMR measured via indirect calorimetry. [30] Another study looking at 30 male non-athletes with chronic motor complete SCI found similar results. [31] Broad et al. [32] demonstrated the Cunningham equation is also the most accurate prediction equation for estimating REE in athletes. In a sample of 14 male wheelchair rugby athletes, no significant difference was found between measured REE and estimated REE. [32] Similar results were reported in a study comparing 7 male athletes with SCI against 7 matched, able-bodied controls. [29]

There are several methods available for assessing exercise energy expenditure (EEE) (i.e. energy expended during deliberate physical activity) in individuals with SCI, including questionnaires, heart rate monitors, accelerometers, indirect calorimetry, and doubly labelled water (DLW), but there are limitations to each of these methods including, but not limited to, high cost, limited access to equipment, and recall bias. [12,27,34] DLW and indirect calorimetry often serve as the reference standards in research, but these methods are usually limited to research settings and therefore are not practical field methods of assessment. In free-living athletes, self-reported training logs are a low-cost method for assessing EEE. [3,34] Athletes record training activity, duration, and intensity for a period of time. In able-bodied athletes, those activities can be converted into energy expended in calories (kcal) using metabolic equivalents (METs). METs are not appropriate to use with individuals with SCI, as the equivalents were



established using able-bodied subjects. [34] Instead, energy expenditure can be calculated using a compendium of energy values for physical activities of wheelchair users, [28,35] but the compendiums currently available provide only limited lists of activities and are based on relatively small sample sizes, so caution must be taken in generalizing the results. Significant variability in energy expenditure exists between individual para athletes, and is influenced by the level of lesion, the degree of completeness of the lesion, and the energy demands of the sport in which the athlete competes. [8,11,16] This significant variability within the population presents a challenge in accurately assessing an individual's EEE.

### *Energy Availability*

Energy availability is calculated by subtracting EEE from the athlete's total EI, and then dividing by the athlete's fat free mass (FFM) in kilograms (kg). [4] In order to calculate EA, all 3 components (EI, EEE, and FFM) must be assessed. As discussed above, there are many barriers to accurately assessing EI and EEE. FFM is usually assessed with dual energy x-ray absorptiometry (DXA) scans in research settings, and limitations in using this method as a field assessment tool are acknowledged.

The threshold for LEA in able-bodied females is defined as  $< 30 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  and optimal EA as  $> 45 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$ . [4,5] The LEA threshold has not yet been established for able-bodied males, but preliminary research suggests the threshold may be closer to  $20\text{-}25 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$ . [3,10] The LEA threshold for athletes with SCI athletes is also unknown, and may depend on level of impairment and completeness of the spinal cord lesion. [8,11] Applying the threshold(s) established using able-bodied

individuals to athletes with SCI may not accurately represent the risk of LEA in this population.

The body of literature suggesting able-bodied male athletes are at risk for LEA is growing. [3,4,10,13,36,37] Generally, athletes that compete in sports emphasizing leanness, such as horse racing, endurance sports like running and cycling, and make-weight sports like wrestling and body building, are at a greater risk for LEA. Refer to Burke et al. [38] for a more comprehensive review on RED-S in male able-bodied athletes.

It is unclear how long an athlete must experience LEA before negative health outcomes are observed. Some research suggests male athletes appear to be more resilient to short term disruptions in EA than female athletes. [13,39,40] A study by Papageorgiou et al. [39] did not observe a change in hormone levels following energy restriction. In a randomized, crossover design, 11 male and 11 female subjects underwent 2 trials of differing energy availability, each lasting 5 days. The subjects' energy intakes were  $45 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  in the energy-balanced condition and  $15 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  in the energy-restricted condition. Following both conditions, the researchers did not find a significant difference in insulin-like growth factor-1 (IGF-1) levels in the male subjects. Interestingly, the researchers observed a significant decrease in IGF-1 levels in the female subjects. [39] In another study with a randomized, cross-over design, 6 men experienced 2 different EA conditions during 4 separate 4-day trials. [40] Energy availability was  $15 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  during the 2 trials of LEA, and was  $40 \text{ kcal} \cdot \text{kg}$

FFM<sup>-1</sup>·day<sup>-1</sup> during the 2 trials of normal EA. The subjects did not experience significant changes in hormones associated with RED-S during either energy availability condition.

Conversely, other research has suggested even short-term disruptions in EA can impact health. A recent pilot study by Heikura et al. [41] demonstrated that alternating days of optimal EA and LEA had negative effects on hormones. Six (6) cyclists participating in an elite road cycling competition, consisting of 4 days of cycling alternated with 4 days of rest, consumed food *ad libitum* and recorded their intake. On race days, the athletes consumed an average EA of  $14.4 \pm 8.5$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>, and on rest days they consumed an average EA of  $56.9 \pm 9.8$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>. Despite sufficient energy intakes on rest days, the athletes with the lowest EA on race days ( $n = 2$ ) exhibited reduced levels of testosterone (TES) and IGF-1, suggesting even short-term extreme LEA may have negative health outcomes. [41] In another study, Zanker et al. [14] subjected 8 experienced male distance runners to 2 different energy intake conditions:  $\sim 40$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> and  $\sim 20$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> for 3 days each in a randomized, cross-over design. The researchers observed a significant drop in IGF-1 following the energy-restricted condition, and no significant change following the energy-balanced condition. [14]

An example of long-term, or chronic, LEA is the well-known Minnesota Starvation Study [42] lead by Ancel Keys, which demonstrated many potential and significant negative health effects resulting from LEA. During 6 months of energy restriction, the researchers observed decreased resting heart rate, increased irritability, social withdrawal, a significant drop in libido, and a hyper-fixation on hunger and food,

among many other symptoms. [42] A recent estimate of the EA for the subjects in Keys' study was  $\sim 22 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  for 6 months. [10] Further research is warranted to identify the necessary degree and duration of LEA that is required before negative health outcomes are observed, and to establish how long the recovery process could be and what it might need to entail.

While there is a paucity of research examining the prevalence of LEA in male para athletes, several studies suggest male athletes are at an increased risk for LEA. [11,19] This population appears to present with risk factors associated with RED-S, independent from measured LEA, such as increased risk for disordered eating behaviors, low bone mineral density, and altered hormone levels. These risk factors will be discussed individually in subsequent sections.

#### *Eating Attitudes and Behaviors*

There are several SCI-associated health issues that indirectly increase risk for LEA by affecting an athlete's dietary behaviors. [12,17,43] Depending on the level of lesion, SCI can alter GI function and cause delayed gastric emptying. As a result, athletes may restrict eating prior to training to avoid the discomfort of exercising on a full stomach, especially if the position in their wheelchair increases intra-abdominal pressure. Athletes may also increase their fiber intake in order to improve GI transit, but the satiating effect of fiber can result in a decreased energy intake. Pain medications and antibiotics often have side effects such as nausea, diarrhea, or constipation, all which can alter food intake. Finally, decreased mobility can make grocery shopping or food

preparation more difficult, which may limit the quality or quantity of meals an athlete consumes.

Para athletes also may exhibit eating attitudes and beliefs that may increase risk for LEA. Krempien and Barr [18] reported that male para athletes are more likely to engage in disordered eating behaviors than able-bodied counterparts. In one study looking at elite para athletes, researchers found that the male athletes scored equally as high as the female athletes for cognitive dietary restraint on the Three-Factor Eating Questionnaire (TFEQ), i.e. all the athletes reported deliberate monitoring of energy intake in order to manipulate or control body composition. In able-bodied populations, males historically score significantly lower on this category of the TFEQ. In another study, Brook et al. [8] surveyed 260 male and female elite para athletes regarding risk factors associated with RED-S, including disordered eating behaviors. The researchers found that while only 0.7% of male athletes reported a history of a diagnosed eating disorder, 14% had elevated Eating Disorder Examination Questionnaire (EDE-Q) scores for dietary restraint and 36.7% had scores for pathological behavior. Additionally, 63.3% reported they were actively attempting to alter their body composition specifically for sport performance. There were no significant differences between sexes in any of these measures.

The EDE-Q was developed using females, and thus may not accurately reflect the desires present in males with disordered eating behaviors; it likely under-represents attitudes and beliefs that are specific to male athletes, such as a desire for increased muscularity. [44] A questionnaire similar to the Low Energy Availability in Females

Questionnaire (LEAF-Q) is reportedly in development; the Low Energy Availability in Males Questionnaire (LEAM-Q) may be able to better assess male athletes for disordered eating behaviors in the future. [37] Currently, there is no eating disorder questionnaire that is designed for use in the para athlete population specifically, despite expressing unique concerns such as fitting into a racing chair or maintaining the comfortable fit of a prosthesis. This underscores the need for a para athlete-specific addendum or survey to appropriately assess risk of LEA related to eating attitudes and behaviors.

### *Hormone Function*

Low energy availability has been associated with disrupted hormone function in male able-bodied athletes. [3,4,6,10,13,36,41] Associations between energy deficiency and low levels of IGF-1, specifically, have been inconsistent in male able-bodied athletes. [3,6,14,41] In the previously mentioned studies, IGF-1 decreased by 17% from baseline ( $p = 0.007$ ) in 8 male distance runners following an EA of  $\sim 15 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  for 3 days, [14] and an extreme EA of  $< 10 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  on race days was associated with an overall 25% decrease in IGF-1 from baseline in 2 elite cyclists, even when EA was  $> 46 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$  on rest days. [41] However, other studies did not report a significant change in IGF-1 levels following short term EA  $< 15 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$ . [39,40] Additionally, Heikura et al. [3] did not find an association between LEA and depressed IGF-1 in elite male distance runners.

The body of research regarding IGF-1 levels in SCI individuals is also mixed. It is common to observe depressed levels of IGF-1 in individuals with SCI. [45,46] However, some studies have reported normal levels. [47,48] At baseline, Bauman et al. [47]

compared serum IGF-1 levels in 9 pairs of adult monozygotic twins with SCI against a cohort of matched, able-bodied twins and found that there was no statistical difference in serum IGF-1 levels. Gorgey and Gater [48] demonstrated that IGF-1 is positively associated with higher muscle spasticity in individuals with SCI. The authors hypothesize that this increased muscle activity may promote an increase in extrahepatic IGF-1 synthesis. [48] This idea is supported by a separate research study that documented low IGF-1 levels in individuals with poliomyelitis, where it is more common to see flaccid paralysis (i.e. decreased muscle activity). [49] Currently there is no research examining the relationship between IGF-1 and energy restriction in male para athletes. At this point, it is unclear whether IGF-1 is a useful tool in assessing risk for LEA.

Research has suggested a stronger association between energy restriction and depressed levels of TES in male able-bodied athletes. [3,4,6,10,13,36,41] Athletes competing in sports emphasizing leanness, such as endurance running, cycling, rowing, and combat sports appear to have an increased risk for low TES. [38] Gomez-Merino et al. [50] reported a significant decrease in TES in 26 male soldiers after 3 weeks of physical training followed immediately by a 5-day combat course. During the combat course, soldiers experienced significant energy restriction (a deficit of ~1,800 kcal) and were only allowed to sleep 3-4 hours per 24 hours. From the beginning of the physical training to the end of the combat course, TES dropped from  $15.1 \pm 0.7$  nmol/L to  $9.8 \pm 0.6$  nmol/L. [50] An observational study found TES was significantly lower in the group with an EA of  $\sim 27$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> (male distance runners) compared to the group with an EA of  $\sim 45$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> (healthy, sedentary controls). [51]

It has been suggested that using the clinical threshold ( $< 9$  nmol/L) for low TES may not be sensitive enough to identify athletes experiencing LEA. [13] Therefore, some studies have defined “low TES” as within or below the lowest quartile (9-16.5 nmol/L) of the normal clinical range (9-38 nmol/L). [3,52] Using this definition, Heikura et al. [3] reported low TES in 40% of elite male distance athletes ( $n = 24$ ), which was associated with lower EA and significantly lower free triiodothyronine (fT<sub>3</sub>) levels. Another study with 53 male endurance athletes reported an average EA of  $37.7 \pm 10.9$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> and found TES levels to be above the lowest quartile range, with fT<sub>3</sub> and IGF-1 also within normal ranges. [52]

The relationship between low TES and energy status in male para athletes is unclear. Research suggests that low levels of TES may be a natural consequence of SCI and declining levels are associated with duration of injury. [45,46] Studies looking at non-athletic males with chronic SCI have reported 45-60% of subjects present with hypogonadism, defined as  $< 10.4$  nmol/L. [46,53,54] While the underlying etiology of hypogonadism in males with SCI has not yet been identified, it is suspected that disruption of the hypothalamic-pituitary-gonadal axis (HPG axis) by the SCI may alter TES production. [46,54] To date, there are no published studies assessing the relationship between energy availability and TES in male para athletes.

It is worth noting, the aforementioned studies reporting hormone levels used thresholds and clinical ranges that were established using able-bodied males; therefore, it may be inappropriate to apply these thresholds to individuals with SCI. Altered hormone levels appear to be both a consequence of LEA and a natural outcome of SCI. As such, it



is important to establish normal clinical hormone ranges for individuals with SCI so that it will be possible to distinguish between impaired hormone function as a result of SCI and impaired function as a consequence of LEA.

### *Bone Mineral Density*

Impaired bone health has been associated with LEA in male able-bodied athletes. [3,4,6,13,36] In the previously mentioned study by Zanker et al., [14] a significant reduction in procollagen type 1 N-terminal propeptide (P1NP), a marker for bone formation, was reported in male distance runners following LEA for 3 days. Loss of BMD is also an expected consequence of SCI and is reported in nearly every case. [11,21,55,56] Bone responds to the prevailing forces, i.e. bone is strengthened with increased skeletal loading. Individuals who become wheelchair-bound due to SCI consequently decrease their weight-bearing activities, which increases their risk for osteoporosis and related fractures. Loss of BMD following SCI is most rapid within the first few months of an acquired SCI, and reaches steady-state after roughly 2 years. [21] Some studies report no significant differences in lumbar spine BMD between SCI individuals and able-bodied controls. [56] It was previously hypothesized that this may be due to continued loading of the spine in a seated position, however recent research has challenged this idea, suggesting DXA scans may overestimate lumbar spine BMD due to increased neuropathic calcification at this site. [55]

Research on wheelchair athletes has suggested that participation in sport can attenuate whole-body decreases in BMD. [20,29,57] Goktepe et al. [57] compared the bone densities of 17 male wheelchair basketball players against 17 male sedentary

paraplegic individuals and found that BMD in the arms of the athletes was significantly higher than in the controls. The athletes also had a greater mean T-score for the distal radius than the control group. Interestingly, there was no difference in BMD below the level of lesion. [57] This finding was supported by Miyahara et al., [20] who compared 28 male wheelchair athletes against 25 active male able-bodied controls and also found that the wheelchair athletes had greater arm BMD than the controls. In another study comparing whole body BMD between 7 wheelchair athletes with SCI and 7 matched, active able-bodied controls, no significant differences were observed between athletes and controls, [29] further supporting the notion that physical activity is beneficial for overall bone health in wheelchair-bound athletes.

Currently, there is no research assessing the relationship between BMD and LEA in male SCI athletes. Low BMD is a natural consequence of SCI and it is also a key risk factor for LEA. Therefore, further research must be done to develop reference standards for BMD in athletes with SCI so that it will be possible to discern whether low BMD is more closely related to SCI or LEA on a case-by-case basis.

### *Conclusion*

In conclusion, there are several areas of research that warrant further investigation. The parallels between symptoms related to SCI and symptoms related to RED-S make it difficult to discern the underlying etiology of certain health concerns in male para athletes, such as altered hormonal profiles and low measures of BMD. There is a need to establish reference standards and clinical thresholds using para athletes in order to more accurately screen for LEA and RED-S in this population. Additional research to

develop and validate assessments of eating attitudes and behaviors that address the unique concerns of this population is also needed. Currently, there is insufficient evidence available to assess the risk of LEA in elite male para athletes.

### III.

## METHODS

### *Participants*

Nine (9) male para athletes were recruited from the University of Illinois wheelchair track and field team, and from the United States and Canadian Paralympic teams via word-of-mouth and email. Participants were > 19 years old (mean age  $28.6 \pm 7.7$  years), had a spinal cord impairment (e.g. an acquired SCI or congenital conditions such as cerebral palsy, or spina bifida), and were restricted to a wheelchair for locomotion. Athletes were excluded from the study if they were not participating in their normal training due to an injury. All participants were informed of and consented to the study design. This study was approved by the Central Washington University Human Subjects Review Committee.

### *Study Design*

A descriptive study, adapted from DiFolco et al., [26] was conducted in January 2019 during a normal training schedule at the University of Illinois Urbana-Champaign (Urbana, IL), and again in May 2019 during a training camp in Daytona Beach, FL. Data collected included anthropometric data such as height, weight, and impairment details; 7-day diet logs with concurrent 7-day training logs; blood spot tests; Dual Energy X-Ray Absorptiometry (DXA) scans; and eating behavior questionnaires. Results and responses were used to evaluate risk of LEA and assess prevalence of symptoms related to RED-S.

### *Energy Availability*

EI and EEE were estimated via the prospective diet and training logs kept by each participant over 7 consecutive days (see Table 1). Before recording, the participants viewed a training video for how to properly complete these logs and were instructed to follow their normal diet and training patterns. Completed diet logs were reviewed by a registered dietitian nutritionist (RDN), who asked the participants follow-up questions as needed to clarify recorded intake. A nutrient analysis program (Elizabeth Stewart Hands and Associates Food Processor, Salem, OR) (ESHA) was used to analyze each participant's EI. Completed training logs were used to estimate EEE (see Table 1). Together, EI and EEE were used to assess EA in participants. LEA was defined as  $\leq 25$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>.

### *Eating Attitudes and Behaviors*

Risk of disordered eating behavior was assessed with the Eating Disorder Examination Questionnaire (EDE-Q, version 6.0). [58] The EDE-Q is a self-report questionnaire used to evaluate behaviors and attitudes associated with disordered eating and eating disorders. The questionnaire includes 22 attitudinal questions that are scored on a 7-point Likert scale ( $0 = no\ days$ ;  $6 = every\ day$ ), and these questions are distributed within 4 subscales: (i) Restraint, (ii) Eating Concern, (iii) Shape Concern, and (iv) Weight Concern. Six (6) additional questions ask for frequency of occurrence of an eating disorder-related behavior. All questions refer to behaviors that occurred over the last 4 weeks (28 days) only. Each participant's mean global score was calculated by

**Table 1:** Method for assessing EA based on diet and training logs

EA	<ul style="list-style-type: none"> <li>EA = (EI – EEE)/FFM</li> </ul>
EI	<ul style="list-style-type: none"> <li>EI was assessed via self-reported 7-day diet logs.</li> <li>A training video was used to educate athletes on how to properly complete diet logs (e.g. serving sizes, timing, food description).</li> <li>Instructions within diet log further described how to record accurate portion sizes using weights and household measures.</li> <li>RDN analyzed completed diet logs with ESHA nutrient analysis software to estimate EI.</li> </ul>
EEE	<ul style="list-style-type: none"> <li>EEE was assessed via self-reported, concurrent 7-day training logs. Athletes recorded exercise description, training duration, and intensity.</li> <li>Each exercise activity was assigned an energy cost (kcal·kg<sup>-1</sup>·hr<sup>-1</sup>) according to intensity and type of activity, using a compendium of activity energy costs for wheelchair users. [35]</li> <li>tEEE was calculated by multiplying energy cost for each activity by duration of activity and athlete’s weight (kg).</li> <li>REE was estimated using the Cunningham prediction equation, [33] then divided by 24 to get hourly REE. Hourly REE was multiplied by duration of activity to get REE during activity.</li> <li>REE during activity was subtracted from tEEE to get EEE, so that only the additional energy cost of exercise was included in EEE.</li> </ul>
FFM	<ul style="list-style-type: none"> <li>Fat-free mass assessed via DXA scans</li> </ul>
LEA Threshold	<ul style="list-style-type: none"> <li>Low EA [3]: <math>\leq 25 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}</math></li> </ul>

*Abbreviations:* EA, energy availability; EI, energy intake; EEE, exercise energy expenditure; FFM, fat-free mass; RDN, registered dietitian nutritionist; ESHA, Elizabeth Stewart Hands and Associates; tEEE, total exercise energy expenditure; REE, resting energy expenditure; DXA, dual energy x-ray absorptiometry; LEA, low energy availability

*Note:* Table reproduced and adapted with permission from DiFolco et al. [26]

summing the scores of the 22 scaled questions, and then dividing that sum by the number of subscales (4); individual subscale scores were calculated by summing scores within the subscale, and then dividing that sum by the number of questions within the subscale. [59]

Recent evidence suggests the global score threshold for identifying risk of disordered eating behaviors is lower for males than it is for females [60,61]; therefore, a mean global score of  $\geq 1.68$  indicated the participant was “at risk” for disordered eating behaviors, and a score of  $< 1.68$  indicated the participant was “not at risk.” [44] Though the EDE-Q was developed using female subjects, the EDE-Q has demonstrated acceptable test-retest

reliability in males (subscale reliability ranges from  $r = 0.80$  to  $r = 0.94$ ; global scale  $r = 0.92$ ). [62]

### *Bone Mineral Density and Anthropometrics*

DXA scans (General Electric, Lunar iDXA) were used to assess body composition, including BMD, fat mass (FM), and FFM. Sites scanned include whole body, hip, femur, and lumbar spine. Participants arrived in the morning, and scans were performed while the participants were fasted and rested. The generated  $Z$ -scores were calculated based on an able-bodied population, as there is currently no reference data for the SCI population. [63] The accuracy of DXA has not yet been defined for wheelchair-bound populations; however, DXA has been shown to provide precise, reproducible measures of BMD, FM, and FFM in elite male wheelchair athletes. [64] Height, weight, nature of impairment, and level of lesion were self-reported by participants, and BMI was calculated.

### *Hormone Function*

Hormonal profiles for subjects were obtained using dried blood spot tests supplied by ZRT Laboratories (Beaverton, OR). Blood samples were collected onto blood spot cards via finger stick in the morning. Cards were allowed to dry for a minimum of 30 minutes, and then, samples were sent to be assayed by ZRT Laboratories for TES, IGF-1, free triiodothyronine ( $fT_3$ ), cortisol, and estradiol. Accuracy of the dried blood spot assays as compared to corresponding serum samples are as follows: TES ( $R = 0.99$ ), IGF-1 ( $R = 0.88$ ),  $fT_3$  ( $R = 0.82$ ), cortisol ( $R = 0.93$ ), estradiol ( $R = 0.86$ ). Reference values for the hormones were defined based on the ZRT Laboratories reference ranges: TES

(20-29 years: 231-1039 ng/dL; 30-29 years: 332-924 ng/dL; 40-49 years: 216-726 ng/dL; optimal range: 400-1200 ng/dL); IGF-1 (100-300 ng/mL); fT<sub>3</sub> (2.4-4.2 pg/mL); cortisol (morning: 8.5-19.8 µg/dL); estradiol (12-56 pg/mL). Low TES was defined as within or below 9-16.5 nmol/L, i.e. the lowest quartile of the normal clinical range (9-38 nmol/L). [3]

### *Statistical Analysis*

Values for subject characteristics, EI, EEE, EA, EDE-Q scores, body composition, and hormone levels were reported descriptively as mean ± standard deviation. Additionally, BMD was presented using Z-scores, where a Z-score < -1 indicated “increased risk for fracture” [21] and a Z-score < -2 indicated “clinically low.” [63] Participant’s risk for disordered eating from the EDE-Q were reported also in frequencies. Statistical significance was set at  $p < 0.05$ .



IV.

RESULTS

Nine (9) total athletes competing in para athletics ( $n = 5$ ) and wheelchair racing ( $n = 4$ ) participated in this study. Table 2 displays descriptive characteristics of the subjects, including anthropometric data and level of impairment. Table 3 displays risk factors related to RED-S, including EDE-Q scores, BMD Z-scores, and hormone levels.

**Table 2.** Descriptive characteristics of participants ( $n = 9$ )

	Participants									Mean $\pm$ SD
	1	2	3	4	5	6	7	8	9	
Age (yrs)	23	29	35	23	20	25	32	25	45	28.6 $\pm$ 7.8
Height (in)	66	66	72	71	69	64	56	69	63	66.2 $\pm$ 4.9
Weight (kg)	60.3	51.9	74.2	63.2	64.8	71.3	75	52	68.2	64.5 $\pm$ 8.7
BMI (kg/m <sup>2</sup> )	21.5	18.5	22.2	19.4	21.1	27.0	37.1	16.9	26.6	23.4 $\pm$ 6.2
Body fat (%)	34.5	27.3	26.3	16.5	25.1	21.5	26.6	18.9	31.6	25.4 $\pm$ 5.7
Level of Injury	n/a	T10	T11	T4/T6	L3/L4	L4/L5	N/A	C6/C7	n/a	n/a
Duration of Injury (yrs)	23	29	13	4	20	25	32	5	41	21 $\pm$ 12

**Table 3.** Risk factors related to RED-S

	Participants									Mean $\pm$ SD
	1	2	3	4	5	6	7	8	9	
<b>Eating Attitude/Behavior</b>										
EDE-Q Global Score	0.43	0.69	n/a	0	0	0.05	0.55	1.17	0.14	0.38 $\pm$ 0.41
<b>Bone Health</b>										
Whole Body BMD (g/cm <sup>2</sup> )	1.15	1.45	1.09	1.30	0.97	1.44	1.18	1.20	1.16	1.22 $\pm$ 0.16
Hip Z-Score	-0.9	<b>-2.5</b>	<b>-1.5</b>	<b>-1.3</b>	n/a	<b>-1.6</b>	<b>-1.3</b>	<b>-1.3</b>	<b>-2.9</b>	-1.7 $\pm$ 0.7
<b>Hormones</b>										
TES (nmol/L)	<b>8.5</b>	<b>10.0</b>	<b>11.0</b>	<b>4.3</b>	<b>7.6</b>	<b>7.8</b>	<b>5.7</b>	<b>10.2</b>	<b>5.9</b>	7.9 $\pm$ 2.3
IGF-1 (ng/mL)	321	n/a	574	227	389	135	328	225	106	288 $\pm$ 151
fT <sub>3</sub> (pg/mL)	3.1	3.2	3.0	3.6	3.2	3.0	2.5	3.0	3.4	3.1 $\pm$ 0.3
Cortisol ( $\mu$ g/dL)	14.0	9.9	2.7	5.6	13.0	8.2	4.1	12.1	12.4	9.1 $\pm$ 4.2
Estradiol (pg/mL)	1532	43	22	<10	20	36	45	41	38	n/a

*Abbreviations:* EI, energy intake; EEE, exercise energy expenditure; EA, energy availability; FFM, fat-free mass; EDE-Q, Eating Disorder Examination-Questionnaire [58]; BMD, bone mineral density; TES, testosterone; IGF-1, insulin-like growth factor-1; fT<sub>3</sub>, free triiodothyronine, and n/a, not available or applicable.

**Bold** indicates risk factors associated with RED-S

Due to incomplete data, EA could only be calculated for 4 athletes. This data is presented in Table 4.

**Table 4.** Calculated EA per day and average EA for the week

	Days							Mean ± SD
	1	2	3	4	5	6	7	
<b>Participant 1</b>								
EI (kcal)	1993	2018	3905	1044	2076	3945	2230	2459 ± 1073
EEE (kcal)	432	0	182	103	154	222	0	156 ± 149
EA (kcal·kg FFM <sup>-1</sup> ·day <sup>-1</sup> )	40	51	94	<b>24</b>	49	94	56	58 ± 27
<b>Participant 4</b>								
EI (kcal)	1916	1846	1004	1552	1782	1963	1861	1703 ± 336
EEE (kcal)	n/a	n/a	0	0	377.2	142	430.1	190 ± 204
EA (kcal·kg FFM <sup>-1</sup> ·day <sup>-1</sup> )	n/a	n/a	<b>19</b>	29	27	34	27	27 ± 6
<b>Participant 6</b>								
EI (kcal)	2141	2392	1672	1759	2480	2282	1503	2033 ± 385
EEE (kcal)	19	0	118	n/a	0	n/a	123	52 ± 63
EA (kcal·kg FFM <sup>-1</sup> ·day <sup>-1</sup> )	38	43	28	n/a	44	n/a	<b>25</b>	35 ± 9
<b>Participant 9</b>								
EI (kcal)	1979	1711	1724	2938	2130	2874	2286	2234 ± 503
EEE (kcal)	23	0	679	337	0	569	134	249 ± 284
EA (kcal·kg FFM <sup>-1</sup> ·day <sup>-1</sup> )	42	37	<b>22</b>	56	46	49	46	43 ± 11

*Abbreviations:* EI, energy intake; EEE, exercise energy expenditure; EA, energy availability; FFM, fat free mass; SD, standard deviation

**Bold** indicates EA was below suggested male threshold<sup>3</sup> of  $\leq 25$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>

The mean EI for the sample was  $2408 \pm 580$  kcal·day<sup>-1</sup> (range: 1703 to 3695 kcal·day<sup>-1</sup>). The mean EEE of the 4 athletes with sufficient data was  $183 \pm 58$  kcal·day<sup>-1</sup> (range: 143 to 249 kcal·day<sup>-1</sup>). None of those participants had an average weekly EA below the suggested male LEA threshold<sup>3</sup> of  $\leq 25$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>, however each athlete experienced 1 day of LEA at some point during the week. The number of training sessions reported on the days the athletes experienced LEA ranged from a rest day to 2 training sessions per day ( $n = 1$ , no training;  $n = 1$ , 1 training session;  $n = 2$ , 2 training sessions). The mean EA for those participants was  $41 \pm 12$  kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup> (range: 27 to 58 kcal·kg FFM<sup>-1</sup>·day<sup>-1</sup>).

The mean EDE-Q global score was  $0.38 \pm 0.41$  (range: 0 to 1.17), indicating none of the participants were “at risk” for an eating disorder. The subscale with the highest average score ( $0.61 \pm 0.98$ ) was *Shape Concern*, followed by *Dietary Restraint* ( $0.50 \pm$

0.81), *Weight Concern* ( $0.35 \pm 0.44$ ), and finally *Eating Concern* ( $0.05 \pm 0.09$ ). One (1) participant reported using exercise in a “driven” or “compulsive” way to expend calories and manipulate body composition; they reported engaging in this behavior during 3 days out of the prior 28 days. Four (4) athletes (50%) reported multiple occasions during the prior 28 days where they ate an “unusually large amount of food,” and 1 participant reported 4 discrete days where binge eating occurred.

Whole body and lumbar Z-scores were unreliable due to the presence of surgical hardware and contractures in these regions, therefore hip Z-scores were reported. These scores indicated 5 participants (63%) were at an “increased risk for fracture” ( $Z < -1$ ), and 2 participants (25%) had clinically low scores ( $Z < -2$ ). None of the athletes reported a history of sports-related bone injury, such as a stress fracture, within the last year.

All participants exhibited low TES, defined as 9-16.5 nmol/L, i.e. within or below the lowest quartile of the normal clinical range (9-38 nmol/L). [3] Six (6) participants (67%) presented with clinically low TES ( $< 9$  nmol/dL). IGF-1 was elevated in 4 participants (50%) and within normal range for the rest; there was no observed trend between low TES and IGF-1 levels. All participants were in the reference range for  $fT_3$ . No athlete presented with elevated cortisol, but it was low in 4 participants (33%). One (1) athlete had an extremely high estradiol level of 1532 pg/mL, while the remainder of the athletes’ levels were within the reference range of 12-56 pg/mL (ZRT Labs).

## V.

### DISCUSSION

The purpose of this study was to assess the prevalence of symptoms related to RED-S in elite male athletes with SCI. Usable data was available to calculate LEA for 4 athletes. All 4 of those athletes experienced a single-day occurrence of LEA during the week, but they each had 7-day EA averages above the LEA threshold. Two (2) athletes exhibited clinically low hip *Z*-scores. All athletes presented with low TES. None of the athletes appeared to be at risk for disordered eating according to the EDE-Q scores. The primary findings indicate all of the athletes in this sample presented with at least 1 risk factor related to RED-S, independent from calculated LEA, and therefore suggests that these athletes may be at risk for LEA and negative health outcomes associated with RED-S.

Seven-day averages represent chronic EA better than any single day alone, but this data may still misrepresent actual EA of the athletes. [9] There are many difficulties associated with collecting quality data using self-reported diet and training records, including a significant burden to the subject, inaccurate reporting, and the potential for the action of tracking to influence the subject's intake. [3,9,12] As such, EA alone may not be a sufficient diagnostic tool for RED-S. Despite few reported intakes in the present study that resulted in LEA, most of the athletes presented with health concerns related to RED-S, in particular low hip *Z*-scores and low TES.

Out of the 4 athletes that provided sufficient data to calculate EA, each athlete exhibited LEA ( $\leq 25 \text{ kcal} \cdot \text{kg FFM}^{-1} \cdot \text{day}^{-1}$ ) on 1 day out of the 7 recorded days. These

athletes displayed wide variability in EI and EEE across the week, so despite these single occurrences of LEA, the average EA across the week was well above the LEA threshold for each subject. Research has suggested that short-term perturbations in EA do not appear to have as severe of an impact on male athletes overall compared to female athletes. [13,39] However, recent research has suggested that extremely low EA can still result in measurable disturbances in hormone levels in male athletes after only 8 days of intermittent LEA. [41] More research needs to be done to understand the point at which LEA becomes detrimental to the para athlete, and it is possible that this duration is different for para athletes compared to able-bodied, as well as males compared to females.

Sixty-three percent (63%) of the athletes presented with a hip  $Z$ -score  $< -1$ , indicating an increased risk for fracture, [21] and 25% presented with a hip  $Z$ -score  $< -2$ , which is clinically low [63] and indicates a high risk for fracture. [21] While hip  $Z$ -scores alone provide an incomplete assessment of overall bone health, these low  $Z$ -scores are consistent with the body of literature examining individuals with SCI in general. Decreased BMD is frequently a consequence of being wheelchair-bound, especially in the lower limbs. Bones adapt to the prevailing conditions, i.e. a decrease in skeletal loading leads to a decrease in bone strength and density. [8,11] While it has been suggested that participation in sport appears to help attenuate the expected loss of whole body BMD by increasing the stress imposed on the skeleton at specific sites such as the forearm, [20,29,57] osteoporosis is still present in nearly every individual with SCI. [11] Thus, in the absence of depressed IGF-1 and calculated LEA in the present study, it is

plausible the trend for low BMD in this sample was more closely related to the SCI rather than LEA.

It has been suggested that 45-60% of males with chronic SCI present with low TES, and although the exact cause is unclear, it is suspected that disruption of the hypothalamic-pituitary-gonadal axis (HPG axis) related to the spinal cord injury is partly responsible. [46,54] Sullivan et al. [54] compared TES levels in 58 males with complete SCI against TES levels in a cohort of age-matched, able-bodied males; the researchers found that individuals with SCI are four times more likely to experience low TES compared to men without. [54] In the present study, 100% of athletes exhibited low TES. Similar to the reported impaired bone health, it is plausible that the low TES was more closely related to the SCI rather than to LEA. To our knowledge, this is the first study to describe TES levels in male athletes with SCI.

None of the athletes in the present study exhibited low IGF-1. In fact, 50% of the participants had elevated IGF-1. Several studies examining IGF-1 and EA in able-bodied male wrestlers and endurance athletes (primarily running and cycling) have demonstrated a suppression of IGF-1 when the athletes were in an energy deficit [6,14]; however, the picture is less clear when looking at individuals with SCI. It is common to observe depressed levels of IGF-1, independent from EA, in individuals with SCI, [45] but some studies have reported normal IGF-1 levels in this population. [47,48] In one study, researchers found no statistical difference in serum IGF-1 levels of adult twins with SCI when compared to a cohort of matched, able-bodied twins. [47] Gorgey and Gater [48] demonstrated that IGF-1 is positively associated with higher muscle spasticity in

individuals with SCI and hypothesized that this increased muscle activity may promote an increase in extrahepatic IGF-1 synthesis. The results of the present study support this hypothesis, as all subjects are highly active and exhibited normal or above normal levels of IGF-1. It is possible that the athletes' high activity levels help to maintain higher levels of IGF-1, however additional research is needed to further support this hypothesis.

Previous research examining  $fT_3$  levels in male able-bodied athletes has reported low levels in athletes that also had LEA-associated low TES, [3] however studies examining  $fT_3$  in SCI populations specifically have reported normal levels. [26,47,52] The results of the present study are consistent with the latter findings, despite participants displaying low TES. This finding suggests a possible variability in the presentation of risk factors of RED-S between able-bodied and para athletes. It is important to note that the various clinical ranges discussed above were developed using able-bodied individuals, and therefore it may be inappropriate to apply the ranges to the SCI population.

According to the EDE-Q, no participants were at risk for an eating disorder. The reported low scores are consistent with previous research using the EDE-Q with male athlete populations. [52] Torstveit et al. [52] found only 1 out of 34 elite male endurance athletes to be at risk for disordered eating using the EDE-Q. One possible reason for the low scores could be that the EDE-Q does not take into account body shape desires that are more common in male athletes, such as degree of musculature. [44] Similarly, the EDE-Q does not address concerns unique to the para athlete population, such as fitting comfortably into a racing chair or assessing discomfort of eating prior to training.

Previous research has suggested that male para athletes are more likely to engage in disordered eating behaviors than able-bodied counterparts, [18] so male-specific and para athlete-specific surveys are needed to better assess the risk for disordered eating behaviors in this population.

This study has several limitations. The sample size is small and therefore larger samples are needed to confirm or challenge these results. Some of the data was incomplete, which further reduced the sample size for certain measures. Additionally, some of the data was collected during a training camp, which may have caused the athletes to deviate from their normal routines at times, impacting both their EI and EEE. Finally, all of the measurement tools and references values were developed in able-bodied individuals and have not been validated for use in SCI individuals.



## VI.

### CONCLUSION

In this sample of male para athletes, the screening tools and questionnaire used provided mixed results regarding risk for low energy availability. While the blood spot tests and DXA scans indicated all athletes had low TES and some degree of impaired bone health, respectively, the EDE-Q suggested no athletes were at risk for disordered eating and there was an absence of calculated LEA. As such, the risk for LEA appears to be high based on hormonal profiles and DXA scans, but the risk appears to be low based on EDE-Q scores and calculated EA. These results demonstrate that male para athletes can exhibit chronic indicators of RED-S, independent from the presence of LEA. This discrepancy between calculated EA and the results of other screening tools is consistent with the literature, and suggests that calculated EA alone is a poor field assessment to determine risk of LEA; a collection of screening tools is likely needed to provide a comprehensive evaluation.

The risk factors for LEA present in this sample are also common health outcomes in non-athletic individuals with SCI, therefore it is difficult to discern whether these risk factors are related to the spinal cord injury or if they are related to RED-S (or some combination of both). Additionally, the thresholds for the measures used in this study were established using able-bodied individuals, and therefore it may be inappropriate to apply them to para athletes. Further research is warranted to establish a para athlete-specific LEA threshold, normal BMD and clinical hormone ranges, and screening tools for disordered eating behaviors that account for concerns unique to this population.

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