Energy Availability and RED-S Risk Factors in Competitive, Non-elite Male Endurance Athletes

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Abstract

Relative Energy Deficiency in Sport (RED–S) is predicated on the assumption that low energy availability (EA) induces deficiencies—dysfunction in multiple physiologic systems. However, research on RED–S and EA in male athletes is limited in comparison to women. The aim of this study is to investigate EA and the risk factors for RED–S, and their potential associations in non-elite male endurance athletes. Laboratory assessments for resting metabolic rate (RMR), bone mineral density (BMD), blood hormonal biomarkers and maximal aerobic capacity were conducted on 60 competitive, recreationally trained male endurance athletes (age=43.4±11.6 years [mean±SD], training=10.9±2.7 h/wk, 7.1±8.8 years). Participants provided 7–days of training logs and 4–days of diet records. Diet and training records were used to calculate EA. Correlations were used to examine associations between EA and RMR, BMD, stress fractures and reproductive, metabolic and bone biomarkers. Mean EA was 28.7±13.4 kcal/kg fat free mass (FFM), which categorized our sample as low EA (based upon published criterion, < 30 kcal/kg FFM) and at a high risk for RED–S. Hormonal and bone biomarkers were in normal clinical ranges, even though EA was low. The only interesting significant association was EA being negatively associated with total body BMD (r = –0.360, P=0.005), opposite of expectations. On average our subjects displayed a state of low EA based upon the criterion which has been primarily developed from female-based research. Nonetheless, our participants displayed no major hormonal or bone health disturbances found in athletes diagnosed with RED-S. A value of < 30 kcal/kg FFM to diagnose low EA may not be appropriate for non-elite endurance trained men.

Keywords: sex steroids; endocrine; hormones; hypogonadism; testosterone

Key Facts

Athletes are considered at a high risk for RED–S if their energy availability is low (< 30 kcal/kg FFM), which is based upon primarily female research. Our recreationally trained male endurance athletes, as a group, were below this criterion but displayed no RED-S symptomology. As such, the < 30 kcal/kg FFM criterion “cut-point” for defining low EA status and categorizing individuals at a high risk for RED-S may not be appropriate for non-elite male endurance athletes.

Introduction

The female athlete triad (Triad) was first formally identified in the 1990s, as a woman’s issue consisting of three major components: eating disorders, amenorrhea, and osteoporosis. Since the initial 1997 position statement by the American College of Sports Medicine¹, research originally considered the components interrelated but perhaps of different etiologies. However, subsequent evidence supports that the causal link between the components is primarily due to a condition of low energy availability (EA)². Low EA occurs when sufficient energy is
not consumed in order to maintain regular and necessary body functions and match the energy demands of daily exercise. This deficiency in energy disrupts the reproductive system hormones, leading to limited estrogen production and amenorrhea. The estrogen deficit in turn affects bone health resulting in decreased bone mineral density (BMD), increasing the risk of injury such as stress fractures\(^1\). Extensive research in female athletes on the Triad established the required energy needs to optimize performance and minimize health risk; i.e., a female athlete is considered at risk when she is consuming < 30 kcal per kg of fat free mass (FFM) a day, and is considered safe with a consumption of ≥ 45 kcal per kg of FFM\(^3\).

In 2014, a consensus statement, released by an International Olympic Committee (IOC) medical commission, introduced a larger overarching diagnosis premise encompassing not only the female Triad, but also including male athletes with related symptoms. The term Relative Energy Deficiency in Sport (RED-S) was proposed as an umbrella term covering both sexes\(^4\). Unlike in females, in males there is no overt external sign of a problem developing (e.g., loss of menses in women) that can rapidly provide an indication of health problems. However, exercise trained males are known to have higher incidence of stress fractures, disordered eating and reduced resting testosterone levels which are comparable changes to those in women diagnosed with Triad when sex-related differences are accounted for\(^5\). However, there are extremely limited number of studies in men—leaving the potential effects of EA on aspects of the male physiology unclear (i.e., specifically relative to metabolic function and bone health).

Therefore, this study was conducted to investigate EA and the risk factors for RED-S, and their potential associations in non-elite male endurance athletes. Participants were competitive, recreationally trained male endurance athletes (i.e., sports such as cycling, marathons, triathlons in which aerobic endurance-based training was the key component). This population was chosen as to date, most male research has been limited such as cycling, marathons, triathletes (n = 7) and individuals training in a combination of modes (n = 5), where a significant portion of training included aerobic exercise. Participants self-identified their competitive sport activity that was confirmed with their exercise training records. Eleven subjects were excluded from our final analysis due to study protocol violations.

**Participants**

A total of 71 males meeting the inclusion criteria of being competitive, recreational endurance athletes (i.e., runners, cyclists, triathletes) were recruited to participate, of which 60 completed the entire study and were eligible for data analysis. Additional inclusion criteria included being over 18 years of age, a self-reported weekly athletic training volume of 7–10 h and preparing for competitive endurance events. Exclusion criteria included: 1) extended injury time impacting training, 2) being within the first two weeks of a new training cycle or the final two weeks before a competition and 3) a history of medical or surgical events that may significantly affect the study outcome, including cardiovascular disease, endocrine, metabolic, renal, hepatic or musculoskeletal disorders.

Participants included runners (n = 27), cyclists (n = 21), triathletes (n = 7) and individuals training in a combination of modes (n = 5), where a significant portion of training included aerobic exercise. Participants self-identified their competitive sport activity that was confirmed with their exercise training records. Eleven subjects were excluded from our final analysis due to study protocol violations.

**Procedures**

Participants made two visits to our laboratory, with a 7-day observational period between visits. Participants signed an informed written consent, completed several questionnaires (see below) and received directions for diet and training records and a resting electrocardiogram during the initial visit. Following the observational period (at least eight days later), participants returned to the laboratory following an 8 h fast and having performed no exercise in the previous 24 h for resting metabolic rate, BMD, and blood draw assessment. Each participant also completed in this final visit a maximal cardiopulmonary exercise test (VO\(_{2}\text{max}\)), which was used to more objectively calculate the caloric cost of training (see below for details).

**Energy intake**

Each participant maintained a diet record for four days, including two weekdays and an entire weekend (Saturday and Sunday). Participants were encouraged to maintain their standard eating habits and were provided instructions for determining food serving size. They were encouraged to include as much detail as possible, using a scale and (or) packaging measures when possible. Dietary records were inspected upon return for immediate clarification on entries. Only two participants needed to be contacted within two weeks for follow-up clarification. All logs were analyzed for average total calories by the Food Processor Nutrition Analysis Software (ESHA Research, Salem, OR, USA). To be included in the analysis, a minimum of three days (2 weekdays and 1 weekend day) must have been completed. Only one participant

\(^{1}\)Hagstrom et al. Amer J Sports Med. 2009, 37:82–90

\(^{2}\)Herman et al. J Bone Miner Res. 2009, 24:1867–73

\(^{3}\)Witt et al. Appl Physiol Nutr Metab. 2016, 41:1188–95

\(^{4}\)Horakova et al. Obes Rev. 2016, 17:116–31

\(^{5}\)Lane AR, et al. *Transl Med Exerc Prescr* 2021, 1(1):25–32. DOI: 10.53941/tmep.v1i1.29
Exercise energy expenditure

To determine exercise energy expenditure (EEE), a daily training record for seven consecutive days was maintained during the same timeframe as the diet records. No directions for training were provided other than to maintain their normal exercise training to capture habitual behavior. Training records included all training conducted, including mode, duration, focus (i.e., long, intervals, hills), average heart rate (HR), rating of perceived exertion and distance covered. HR was collected with strap-based telemetry and was used to determine caloric cost for each training session. Individuals using wrist measuring devices were provided with a Polar HR monitor (FT4, Polar Electro, Inc., Bethpage, NY, USA) with chest strap for the duration of the study. The Compendium of Physical Activity was utilized in calculations when HR data was unavailable due to type of exercise (i.e., swim). Metabolic equivalents (METs) determination was utilized to calculate caloric cost considering duration of activity and participant fat free mass.

Participants completed VO_{2\text{max}} testing on either a treadmill or cycle ergometer, determined by their primary training mode. This approach of allowing either modality for testing was to permit the athlete to maximize their test response due to sports specific training adaptations. For the VO_{2\text{max}} testing, metabolic respiratory gases were collected and analyzed continuously throughout (Parvo Medics TrueOne® 2400, Sandy, UT, USA) to measure caloric cost at each exercise test workload. HR was also measured throughout each exercise workload.

The HR at each workload during the exercise test was used to provide a more objective calculation of caloric cost for training sessions from the average HR. Training session HR was matched to appropriate exercise test workload HR and that workload cost was multiplied by the duration of the training session. For HRs between specific workloads, the average caloric cost of both workloads was utilized for the calculation.

Participants primarily training and competing in cycling were tested on an electronically braked cycle ergometer (Lode, Groningen, Netherlands). Following a warm-up at 50 Watts (W) and stretching for five minutes each, the metabolic mouthpiece (Two-way “T” non-rebreathing valves, Hans Rudolph Co., Shawnee, KS, USA) was fitted and the assessment began with 3 min of resting collection. Testing began with resistance at a speed comfortable to maintain for 30–40 min. They warmed up at that predetermined speed then stretched for five minutes each. The metabolic mouthpiece was fitted and three minutes of resting data was collected in a seated position. Following rest, the test began at the predetermined speed, which increased by 0.8 km/h every two minutes until anaerobic threshold was met, indicated by respiratory exchange ratio (RER) at 1.00. Subsequently, 2% increases in grade occurred every minute until volitional fatigue.

BMD and body composition

BMD was measured for total body, as well as the lumbar and femoral neck regions using dual energy X-ray absorptiometry (DXA) with a GE Lunar iDXA (GE Healthcare, Waukesha, WI, USA). Participants removed shoes, additional layers of clothing (other than running shorts) and any metal. All scans were performed by the same technician and participants were positioned on the machine per the GE Lunar iDXA standards for each scan. Low BMD was defined as a Z-score ≤ –1 per literature recommendations.

Fat free mass (FFM) and percent body fat were also determined from the DXA scan assessments. The FFM values were utilized in the calculation of EA.

Resting metabolic rate

After resting quietly in a seated position for 30 min, participant resting metabolic rate (RMR) was measured in the supine position using the canopy technique of respiratory gas collection for 30 min with the Parvo Medics TrueOne® 2400 (Parvo Medics, Sandy, UT, USA) metabolic cart, discarding the first five minutes for stabilization of flow rate. This resulted in the total caloric cost per day being determined from 25 min of collected respiratory gases. As a surrogate marker for energy deficiency, RMR\_ratio (measured RMR [mRMR] / predicted RMR [pRMR]) was also calculated and reported. Measurement and use of low RMR\_ratio (< 0.90) is suggested to be representative of a low EA status.

EA

EA status was calculated according to the following equation:

\[
EA = \frac{\text{energy intake (EI)} - \text{exercise energy expenditure (EEE)} - \text{resting metabolic rate (RMR)}}{} \cdot \text{[minutes of exercise]}^{1/3} \cdot \text{[kilograms of fat free mass (FFM)]}^{1/3}
\]

Individual measures were calculated using the average caloric cost for each training session provided and the average caloric intake from the 4-day diet record. RMR was subtracted as the resting caloric demands would have been necessary regardless of exercise activity.

Questionnaires

All participants were asked to complete the following questionnaires along with screening questions developed for female athletes (excluding menstrual cycle questions).

1) The standard Medical History Questionnaire to confirm medical eligibility.
2) Pittsburgh Sleep Quality Index (PSQI), which assesses sleep quality, duration, efficiencies and disturbances as well...
as medication use and daytime dysfunction. The PSQI is a valid and reliable questionnaire with a published cut-off score of ≥ 5 indicating sleep disturbance.  

3) Recovery Stress Questionnaire for Athletes (REST-Q Sport 52). The REST-Q analyzes individual recovery-stress state for the previous 72 h through 19 sub-scales related to training recovery stress. Questions are rated on a 7-point Likert scale from 0 (never) to 6 (always).

In addition, participants received diet and training record documentation forms with instructions for completing them during the 7-day observation period.

**Blood draw—hormonal analysis**

Blood was collected into vacutainer tubes (SST) via a 21-gauge needle from a vein in the antecubital fossa and immediately placed in a refrigerator to clot. Once clotted, tubes were then centrifuged at 4°C at 3000 rpm for 10 min and separated serum was aliquoted into four 2 mL cryovials and frozen in a −80°C ultra-freezer until later analysis. Commercially available specific enzyme-linked immunosorbent assay (ELISA) kits were utilized to measure the bone marker (NeoScientific, Woburn, MA, USA) and all hormonal biomarkers (ALPCO, Salem, NH; Abcam, Cambridge, MA, USA). Serum biomarkers’ minimal detectable concentrations identified by the manufacturer were as follows: bone alkaline phosphatase (0.1 ng/mL), growth hormone (0.5 ng/mL), sex hormone binding globulin (SHBG; 0.1 nmol/L), free triiodothyronine (0.05 pg/mL), free thyroxine (0.05 ng/dL), total testosterone (0.022 ng/mL) and luteinizing hormone (1.0 mIU/mL). Additionally, free testosterone and bioavailable testosterone were calculated using procedures from Vermeulen et al. Relative to their age, the subjects were of a high cardiovascular fitness level based on normative data for VO2max. Evaluation of the PSQI questionnaire score (8.8 ± 4.7 points, [7.6, 9.9]) suggested the participants experienced only slight, minor sleep disruption and the REST-Q Sport 52 indicated they were in a normal psychological state (i.e., low stress) and not experiencing overtraining symptoms or undue fatigue during the study period (data not reported). The parameters used in determining EA and various related components are presented in Table 2; while reproductive and metabolic hormonal outcomes are reported in Table 3.

### Results

Participant physical and training characteristics as well as VO2max testing responses are shown in Table 1.

| Characteristics | Mean ± SD | CI |
|-----------------|-----------|----|
| Age (years)     | 43.4 ± 11.6 | 40.4, 46.3 |
| Height (m)      | 1.78 ± 0.06 | 1.76, 1.80 |
| Mass (kg)       | 76.6 ± 9.6 | 74.1, 79.0 |
| FFM (kg)        | 62.7 ± 6.2 | 61.1, 64.3 |
| % BF            | 17.9 ± 5.0 | 16.6, 19.1 |
| VO2max (mL · kg⁻¹ · min⁻¹) | 55.7 ± 8.0 | 53.4, 57.7 |
| Training (h · wk⁻¹) | 10.9 ± 2.7 | 10.2, 11.6 |
| Training (years) | 7.1 ± 8.8 | 4.9, 9.3 |

SD: standard deviation; CI: confidence interval; FFM: fat free mass; % BF: percent body fat.

The parameters used in determining EA and various related components are presented in Table 2; while reproductive and metabolic hormonal outcomes are reported in Table 3.

| Measurements | Mean ± SD | CI |
|--------------|-----------|----|
| Energy availability (kcal/kg FFM) | 28.7 ± 13.4 | 25.3, 32.1 |
| Energy intake (EI) (kcal/d) | 3073.8 ± 777.1 | 2877.2, 3270.4 |
| Exercise energy expenditure (EEE) (kcal/d) | 1296.0 ± 466.7 | 1177.9, 1414.1 |
| Resting metabolic rate (RMR) (kcal/d) | 1795.5 ± 209.4 | 1742.5, 1848.5 |
| Resting metabolic rate (mRMR/pRMR) | 0.99 ± 0.08 | 0.97, 1.01 |

SD: standard deviation; CI: confidence interval.

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Association analysis

EA was significantly negatively associated with total body BMD ($r = -0.360, P = 0.01$). No other physiologically meaningful significant associations between EA and other outcome measures (e.g., biomarkers, history of stress fracture, etc.) were detected.

Discussion

This study was conducted to identify if low EA and risk factors for RED-S existed in non-elite (i.e., recreational) endurance trained men. Our intent was also to identify if any associations existed among our measures in order to understand the effect of EA on the health and well-being of male endurance athletes. The mean EA in our sample placed them in the high-risk category for EA; and yet, they were healthy and showed no major hormonal–BMD disturbances or potentially detrimental associations between EA and the risk factors measured. Furthermore, their hormonal and BMD biomarkers were within normal clinical reference ranges.

The majority of studies to date that have included a direct measurement of EA have used female samples. There are only a few studies that have focused on EA in males and the sample populations vary considerably by age, sport and performance level. The current sample of competitive, recreationally trained male endurance athletes demonstrated an EA status ($28.7 \pm 13.4 \text{ kcal/kg FFM}$) which was below the criteria of “low status,” based upon the female cut-point standards. This is, however, not an unprecedented finding as a low EA was reported by other researchers in males. For example, Kochler et al. found a low EA status ($< 30 \text{ kcal/kg FFM}$) in 55.7% ($n=167/352; 21.7 \text{ kcal/kg FFM [mean]}$) of young adolescent male athletes who participated in a variety of sport types. This coincides with the prevalence of low EA subjects in the present study ($61.7\%, n=37/60, 20.7 \text{ kcal/kg FFM [mean]}$), although our subjects were much older. This is also in line with low EA prevalence in other studies which ranged from 50.8% to 63.2% in male and female athletic populations, respectively.

As noted, RMR$\text{ratio}$ has been used as a surrogate marker for energy deficiency and as such, a low RMR$\text{ratio}$ ($< 0.90$) is suggested to be representative of a low EA status. Even though our overall sample mean for EA reached the criteria for a “low EA status,” our RMR$\text{ratio}$ response did not meet the proposed surrogate level. Interestingly the linkage between RMR$\text{ratio}$ and low EA has been identified in some but not all female studies; ours appears to be the first study to formally address this issue in males. As such, our findings call into question whether the RMR$\text{ratio}$ is an appropriate low EA surrogate for male athletes.

Table 3 Resting reproductive and metabolic hormonal measures ($n=60$)

| Reproductive hormones                       | Mean ± SD | CI          |
|---------------------------------------------|-----------|-------------|
| Testosterone (ng/mL)                        | 7.8 ± 4.3 | 6.7, 8.8    |
| Free testosterone (ng/dL)                   | 18.3 ± 14 | 14.7, 21.8  |
| Bioavailable testosterone (ng/dL)           | 466.3 ± 357.4 | 375.8, 556.7 |
| SHBG (nmol/L)                               | 36.2 ± 20.9 | 30.9, 41.4  |
| Luteinizing hormone (mIU/mL)                | 5.0 ± 1.6 | 4.6, 5.5    |

Table 4 Measures for bone health status ($n=60$)

| Measurement                        | Mean ± SD | CI          |
|------------------------------------|-----------|-------------|
| Total body BMD (Z-score)           | 0.73 ± 0.95 | 0.49, 0.97  |
| (g/cm$^2$)                         | 1.34 ± 0.13 | 1.31, 1.37  |
| Lumbar BMD (Z-score)               | 1.46 ± 1.36 | 1.11, 1.80  |
| (g/cm$^2$)                         | 1.23 ± 0.18 | 1.19, 1.28  |
| Femoral neck BMD (Z-score)         | 1.59 ± 1.33 | 1.25, 1.93  |
| (g/cm$^2$)                         | 1.07 ± 0.17 | 1.03, 1.11  |
| Bone alkaline phosphatase (ng/mL)  | 10.68 ± 0.30 | 10.60, 10.75|

| Stress fracture history(n, %)       | Yes | No |
|-------------------------------------|-----|----|
|                                    | 11(18.3%) | 49(81.7%) |

SD: standard deviation; CI: confidence interval; SHBG: sex hormone binding globulin.

Table 4 presents the measurements of bone health. BMD was transformed and expressed and reported as Z-scores for standardization purposes. Additionally, BMD values are also reported in physiological units (g/cm$^2$).

BMD: bone mineral density.
Low EA is recognized as a secondary cause of low BMD, via hormonal regulation disruption, and there is research indicating low EA increases the risk of diminished bone health (i.e., low BMD) in both males and females. We found no such relationship and bone-related injuries (stress fractures) were not unusually prevalent. Studies in female and male athletes have also indicated low body mass index (BMI) (≤ 17.5 kg/m²) to be positively associated with increased risk of low BMD. No participants in the current study had a low BMI approaching this level. A weak negative association was identified between EA and total body BMD, indicating as EA decreased, BMD actually increased. To further investigate this finding, an exploratory post hoc analysis was conducted by separating our sample into low EA (< 30 kcal/kg FFM) and adequate EA (≥ 30 kcal/kg FFM) groups. A t test was used to compare total body BMD between groups. The low EA group (n=37) had a significantly higher BMD Z-score (0.98±0.82, P=0.015) when compared to the adequate EA group (n=23, 0.33±1.03). Although it should be noted, the variance in both groups was high, especially in the adequate EA group. Correlation analysis within the low and adequate groups and total body BMD Z-score revealed no significant associations. It is important to note that not all research studies in men have identified any association between EA and BMD, and that ours was negatively associated was certainly unexpected. It is not clear why this weak but significant negative association between EA and BMD existed, although it could be a matter of a casual statistical significance, without the existence of a meaningful physiological relationship. Further research is necessary to pursue this issue and determine if physiological reasons (e.g., other potential endocrine factors) may exist for this finding.

The impact of low EA on the endocrine system has been studied extensively in females and is viewed as the stimulus for disruption of hormones such as estrogen, luteinizing hormone (LH) and triiodothyronine. Studies in men have identified reduced levels of testosterone in endurance trained versus sedentary men, decreased LH pulsatility and abnormal responses in resting LH, as well as prolactin (the latter interferes with the LH feedback mechanism). As noted, all of our hormonal biomarker levels were within normal clinical reference ranges and we found no association between EA status and the hormone levels. Perhaps most importantly relative to RED-S diagnosis, testosterone and LH levels were normal and the hypothalamic–pituitary–testicular axis appeared functional as the expected inverse relationship between these hormones existed (r = -0.664, P < 0.001). While low EA, more specifically RED-S is usually associated with reduced testosterone in males, animal-based work does not support that this is always the case.

The low EA cut-point or threshold for risk of health decrements is based on research conducted almost exclusively in females. Given the different energy demands between male and female reproductive systems, it is reasonable to question the appropriateness of the use of the female-based cut-points for assessing risk status in men (i.e., high risk, < 30 kcal/kg FFM). Fagerberg et al. have suggested a threshold of 20–25 kcal/kg FFM may be more appropriate for men. Along these lines, Koehler et al. suggested an optimal level for men at 40 kcal/kg FFM and potentially a 15 kcal/kg FFM level as a low point criterion. Interestingly though, in their work, even with an EA of 15 kcal/kg FFM the participants still displayed no change in testosterone, triiodothyronine or insulin-like growth factor–1 (IGF–1) hormones as reported in some cases of RED-S. Perhaps a more chronic state of low EA or even a substantially low threshold level of EA is necessary to induce altered endocrine status and hormonal changes in males. Most certainly additional research is warranted to address this point.

As with all studies, there are limitations to our data which need to be acknowledged. In most studies measuring EA, there is reliance on self-reporting from participants which is a perpetual limitation (i.e., are the participants being accurate and truthful). Diet records are known to have error, but to capture habitual behavior within this study it was necessary to rely on such self-reports. Similarly, with exercise training records, although HR monitoring was utilized, the reports were generated by participants. It is important to note that our EEE calculation was more individualized to our participants than in many previous studies through our use of the VO\textsubscript{2}max test specific to their primary training modality (running vs. cycling) to determine caloric cost at exercise workloads. This should have provided a more precise measure than exclusively incorporating the Compendium of Physical Activity alone, hence strengthening our data. Furthermore, diet/training records were reviewed one-on-one with the participants by the lead investigator to ensure the accuracy of the data. Our sample was quite variable on several parameters: for example, participants ranging in age from 21–70 years, exercised across several endurance-based modes of activity and their levels of competitive performance differed greatly. However, as the investigation was observational in design and examined associations, this variability in our participant pool was considered an asset and not a limitation; although we recognize, the issue of the large age range could have influenced the potential associations among some of our measurements. Finally, it should be noted that the participants in this study were competitive, recreational athletes, not elite, national level competitors. While they are competitive in their sport, it is not their livelihood, but nonetheless an important aspect of their lives. As such, the generalizability of the findings from our sample is quite broad when compared to the general public, as the majority of individuals who exercise are at a recreational level and not professionals.
Conclusions

The majority of our sample of recreational endurance athletes displayed a state of low EA based upon the criterion of < 30 kcal/kg FFM, which is typically utilized in the research community and based primarily upon female-based research. Nonetheless, our participants displayed no major hormonal, bone health disturbances or overt signs of being at a high risk for RED-S. As such, this criterion “cut-point” for defining low EA and determining the level of risk for RED-S may not be appropriate for non-elite recreationally endurance trained men. We do acknowledge our findings are a “snap-shot” of the status of our athletes, and where they might be in any potential timeline with regard to RED-S development and (or) risk level could not be determined. Nonetheless, we recommend that coaches, athletes and sports medicine practitioners recognize that the risk factors for RED-S development in male athletes may differ from those found in female athletes.

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Author Contributions

Conception and research design (ARL, ACH), acquisition of data (ARL, ACH, AESR) or analysis and interpretation of data (AESR, KK, JKRM), manuscript preparation (all authors).

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