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ORIGINAL ARTICLE

Prevalence of Triad–RED–S symptoms in high-level Kenyan male and female distance runners and corresponding control groups

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Abstract

Purpose This study examined and compared select Triad–RED–S components/risk factors in high-level Kenyan male and female distance runners to corresponding control groups; focusing on examining energy intake (EI), bone indices, and hormonal markers.

Methods A cross-sectional, observational design was used in which Kenyan male and female ($n=30$ and $n=26$, respectively) middle- and long-distance runners and corresponding male and female control groups ($n=29$ and $n=29$, respectively) were examined.

The participant's bone mineral density (BMD) at the lumbar spine, right femur, and total body were measured using a dual-energy X-ray absorptiometry analysis. Complete blood counts (CBC) were done on the whole blood specimens and hormonal measurements were performed on plasma specimens. In addition, athletes completed metabolic testing to determine maximal oxygen uptakes and 7-day dietary diaries.

Results Overall daily EI across runners and controls within each sex were low, but not significantly different ($p>0.05$). Prevalence of low BMD values (Z score <-2.0) was comparable across groups in each sex ($p>0.05$). CBC measures suggested that both runners and controls were healthy. Finally, slight hormonal differences between runners and their respective controls existed ($p<0.05$), but were not clinically meaningful or observed in typical Triad–RED–S-related parameters.

Conclusion High-level Kenyan male and female runners had low daily EI, but no tendency toward a higher prevalence of low BMD, or Triad–RED–S–related hormonal abnormalities. The occurrence of low EI was not a major risk factor in our athletes; this calls into question whether the current criteria for Triad–RED–S are entirely applicable for athletes of African ethnicity.

Keywords East Africa · Elite athletes · Energy intake · Bone mineral density · Hormones

Abbreviations

BMD	Bone mineral density
BMI	Body mass index
CBC	Complete blood count
Cort	Cortisol
DXA	Dual-energy X-ray absorptiometry
E_2	Estradiol
EA	Energy availability
EI	Energy Intake
FSH	Follicle-stimulating hormone
GH	Growth hormone
HCT	Hematocrit
HGB	Hemoglobin
IAAF	International Association of Athletics Federations (world athletics since October 2019)
IGF-1	Insulin like growth factor-1
Ins	Insulin
IOC	International Olympic Committee
LBM	Lean body mass
LEA	Low energy availability
LH	Luteinizing hormone
LS-BMD	Lumbar spine bone mineral density
Prol	Prolactin

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RBC	Red blood cells
RED-S	Relative energy deficiency in sport
RF-BMD	Right femur bone mineral density
T_3	Triiodothyronine
T_4	Thyroxine
TB-BMD	Total body bone mineral density.
Testo	Testosterone
Triad	Female athlete triad
TSH	Thyroid-stimulating hormone
VO_2	Oxygen consumption
VO_{2max}	Maximal oxygen consumption
WBC	White blood cells

Introduction

Excessive physical activity without a concomitant increase in appropriate dietary intake can be associated with energy deficiency, which can disrupt the reproductive cycle and result in amenorrhea in females (Sangenis et al. 2006; Loucks et al. 1989, 1992). Furthermore, an inadequate caloric intake and the resulting consequence of decreases in endogenous estrogen may eventually result in an imbalance in bone remodeling leading to low bone mineral density (Sangenis et al. 2006; Drinkwater et al. 1984; Ilhe and Loucks 2004). In women, the consequences of these events are referred to as the Female Athlete Triad (Triad) condition. This model is defined as the existence of three specific components: (1) low energy availability (with or without disordered eating); (2) menstrual dysfunction; and (3) low bone mineral density, which can occur in isolation or all three concurrently. Over a period of time, an athlete may move along on a continuous spectrum ranging from optimal health with appropriate energy availability (EA), regular menses, and healthy bones to the opposite end of the spectrum characterized by amenorrhea, low EA, and osteoporosis (Nattiv et al. 2007; Mountjoy et al. 2014). According to De Souza et al. (2014) for female athletes who present with one or more of the Triad components, an early intervention is essential to prevent the progression of the condition to the potentially serious clinical endpoints.

As a new dimension to the Triad–related area of study, Mountjoy et al. (2014) in the IOC consensus statement introduced a more comprehensive, broader terminology for the overall syndrome—Relative Energy Deficiency in Sport (RED-S), which encompasses a range of negative health consequences from exercise training beyond that traditionally associated with the Triad, such as: cardiovascular, gastrointestinal, immunological, endocrine, metabolic, hematological, growth and development, and psychological health. Additionally, these investigators argued that a relative energy deficiency is also affecting male athletes (Mountjoy et al. 2014). Interestingly, there is growing evidence that males may experience low energy availability (LEA) in situations when there is a mismatch between energy intake and the exercise energy expenditure of training or competition. Populations of male athletes at increased risk for LEA and the resulting health consequences of RED-S include cyclists, rowers, runners, jockeys, and athletes in weight class combat sports (Burke et al. 2018). The need to further investigate potential effects of poor energetic status to male athletes was also acknowledged in the first publication of the Female Athlete Triad in 1992. In 2019, Male Athlete Triad was formally recognized as a syndrome which reflects that similar energetic, reproductive, and bone health problems occur in men (Nattiv et al. 2021). The underlying mechanism of RED-S/Triad is an inadequacy of energy to support the full range of body functions involved for optimal health and performance in female as well as male athletes. Low energy availability, which occurs with a reduction in energy intake and/or increased exercise load, causes adjustments to body systems to reduce/conserve energy expenditure, leading to disruption of an array of hormonal, metabolic, and functional characteristics (Mountjoy et al. 2014; Loucks 2004; Hackney 2008).

The success of east African middle- and long-distance runners, especially from Kenya and Ethiopia, is well known as they have dominated international running events for decades (Burke et al. 2018). To this end, Kenyan male and female runners have collectively won astonishing 63 Olympic medals in middle- and long-distance running since the 1996 Atlanta Olympic Games. However, a recent study (Muia et al. 2015) showed that elite-level adolescent Kenyan female runners had LEA, with 17.9% reaching clinical criteria levels and 76% at subclinical levels. Furthermore, it has been shown previously that Kenyan elite runners have low bone mineral density (BMD). For example, Tam and colleagues reported that 40% of their male study participants had a Z score less than -2 at lumbar spine; however, they did not collect dietary intake data from their study cohort (Tam et al. 2017).

To date, published research on the Triad and/or RED-S status is greatly lacking in athletes of diverse racial backgrounds (i.e., African, African-American, Hispanic, and Asian athletes), especially relative to bone health indices. Thus, there is a need to include more diverse athlete populations into such research and to integrate race/ethnicity to determine appropriate prevention and treatment strategies (Mountjoy et al. 2018). Therefore, the purpose of this study is to examine and compare the prevalence of select Triad–RED–S components/risk factors in Kenyan male and female distance runners to corresponding control groups with special interest on examining and comparing energy intake, bone indices, and hormonal markers commonly associated with Triad–RED–S. Our primary hypothesis was that Kenyan male and female distance runners would present

lower bone mineral indices compared to ethnically similar control groups.

Methods

Participants and study design

For this study, a cross-sectional, observational design approach was used in which high-level Kenyan male and female ($n = 30$ and $n = 26$, respectively) middle- and long-distance runners and corresponding male and female control groups ($n = 29$ and $n = 29$, respectively) were examined. However, due to technical reasons, the number of participants varies across the different measurements. Study participants were recruited from their training camps in the Eldoret area and from the Moi University campus area in Kenya. The control group consisted of students from Moi University of similar age to the runners. All participants received written and oral information about the study before signing the informed consent statement.

The seasonal best running performance of each runner was assessed using the International Association of Athletics Federations (IAAF) Scoring Tables (Spiriev 2017) which enables comparison between events and participants (Legaz and Eston 2005; Mooses et al. 2015). All runners were required to be actively engaged in training and competing at the time of the study for middle- to long-distance athletics events, had done so for a number of years, and were viewed by their coaches as high-level performers. For the study, each participant (runner and control) was required to complete three visits in our laboratory facilities in Kenya.

First visit

Anthropometry and body composition

During the first visit, body composition measurements were obtained for both runners and control group and a familiarization session for the runners with the equipment that was used during the metabolic testing to determine maximal oxygen uptake (i.e., VO_{2max} [see later description]).

Height (Power Tape; Lidu Hardware, Zhejiang, China) and body mass (Seca robusta 813; Seca GmbH and Co., Hamburg, Germany) of the participants was measured to the nearest 0.01 m and 0.1 kg, respectively. Body mass index (BMI, kg/m^2) was derived as body mass (kg) divided by body height squared (m^2). The participant's bone mineral density (BMD) at the lumbar spine (LS-BMD), right femur (RF-BMD), and total body (TB-BMD) were measured using a dual-energy X-ray absorptiometry (DXA) analysis (Hologic Discovery QDR Series, Waltham, MA, USA). All the DXA procedures were performed in the morning between 8 and 11 am and all the procedures were carried out by the hospital, and the DXA system was calibrated by the same technician in accordance with the manufacturer's guidelines. DXA procedure was carried out by the same qualified person from the hospital. During the measurements, participants wore shorts and T-shirt. Low BMD was defined as Z score between − 1.0 and − 2.0, while an osteoporosis condition was considered if < − 2.0 (Nattiv et al. 2007). Body composition was evaluated from whole-body DXA scan as described by Shepherd and colleagues (2017).

Second visit

Blood analyses

During this visit, blood samples were obtained from all participants from a vein in the antecubital region of their preferred arm after completing an overnight fast between 7:30 and 9:30 am, and before the exercise testing was performed by the runners. Complete blood counts (CBC) (Analyser Coulter AC T 5 diff AL, Beckman Coulter, Brea, California, United States) were done immediately on the whole blood specimens at the University Moi Hospital Laboratory (Eldoret, Kenya). These blood specimens were then centrifuged, and the plasma separated and stored frozen at − 20 °C until later analysis. Hormonal measurements were performed on plasma specimens using a Cobas 6000 e601 module (Roche Holding AG, Basel, Switzerland) and consisted of luteinizing hormone, follicle-stimulating hormone, prolactin, estradiol, free thyroxine, thyroid-stimulating hormone, testosterone, triiodothyronine, insulin, and cortisol. An IDS-iSYS-automated immunoassay analyzer (Immunodiagnostic Systems Holdings PLC, Boldon, United Kingdom) was used for determining growth hormone and insulin-like growth factor. All hormonal analytical procedures were performed according to the manufacturer's instructions at the University of Tartu, United Laboratories (Tartu, Estonia). All appropriate clinical analytical procedures and steps were utilized to assure the samples were viable and accurate results (Hackney and Viru 2008).

Exercise testing protocol

Also, during this second visit, the runners completed exercise testing. They were instructed to abstain from high-intensity training and/or competition for at least 24 h before exercise testing, and to maintain their usual dietary habits as well as to refrain from caffeine and alcohol consumption prior to returning for their second visit.

Following the warm-up period (10 min, freely chosen speeds) with the treadmill, participants performed an incremental running test on a motorized treadmill (Cardionics Type 3113, Sweden) until voluntary exhaustion. Before

commencement of the test, each participant remained stationary on the treadmill for 3 min and resting cardio-respiratory data were collected. The initial running speed was set at 8 km·h^{-1} for female and 10 km·h^{-1} for male participants with a gradient of 1% (Mooses et al. 2015; Jones and Doust 1996). The running speed was increased 2 km·h^{-1} every 3 min until 16 km·h^{-1} for females and 18 km·h^{-1} for male participants. Following the 3 min stage at 16 km·h^{-1} for female and 18 km·h^{-1} for male participants, respectively, the speed remained constant until the end of the test; however, the elevation was increased 1% after each minute until voluntary exhaustion (MacDougall et al. 1982). Heart rate and expired gases were measured continuously using MetaMax 3B (Cortex Biophysic GmbH, Leipzig, Germany), which was calibrated before each test according to instructions by the manufacturer. The highest average oxygen uptake (VO_2) during a 30 s period as well as a failure to further increase VO_2 consumption despite an increase in the work rate was defined as the VO_{2max} (Wasserman et al. 2004).

Finally, participants were given 7-day dietary intake diaries to record food consumption to all participants and additionally 7-day training diaries to the runners. All participants were instructed on how to record and approximate food amounts—quantities by members of the research team before being excused from our laboratory. This included going through the food diary step-by-step with a description of one example day, which was given to the participant with the diary. To ensure accurate completion of these diaries, verbal explanations were given by a member of the research team and written instructions were sent home with each participant. After returning the filled out food diary, researcher went through the diary and, if necessary, asked additional information/clarification from the participant.

Third visit

Energy intake

During the third and final visit, food diaries were collected from the participants, checked in their presence, and any discrepancies in entries were clarified by a member of the research team. The subsequent dietary analysis was conducted with NutriSurvey (Germany, 2007) software. Based on prior reports with similar populations, it was expected that there would be the potential for substantial underreporting of dietary intake (Mertz et al. 1991; Fudge et al. 2006; Scagliusi et al. 2006; Pfrimer et al. 2015; Orcholski et al 2015). To that end, to be conservative in our assessment of dietary status, we included three highest energy intakes (EI) days from the 7-day dietary intake diary and a correction factor was devised from this literature and applied (~46% underreport). Herein, we report the nutritional findings in both an uncorrected and corrected fashion.

Statistical analysis

Statistically, the data are presented descriptively as means and standard deviations (± SD); additionally, range values are reported. Between-groups comparisons (runners vs. controls) within each sex for most variables were performed with a one-way ANOVA with statistical significance set at $p \leq 0.05$, and for comparison of prevalence of low bone mineral density (Tables 2 and 3), a Chi-square analysis with Fisher's exact test was utilized. All statistical computations were performed with SPSS version 20.

Ethical statement

All ethical approaches and procedures were carried out according to the World Medical Association Declaration of Helsinki. The study procedures and protocols were approved by the Institutional Review Board of School of Medicine, Moi University, Kenya and Research Ethics Committee of the University of Tartu, Estonia.

Results

The participant's physical characteristics and IAAF performance scores and VO_{2max} values (runners only) are reported in Table 1. Male athletes were slightly older ($p = 0.001$), and had lower mass ($p = 0.030$) and a lower BMI ($p = 0.024$) compared to the male controls. Female athletes had lower body mass ($p = 0.001$) and BMI ($p = 0.001$) compared to the female controls. BMI was in the normal range for all study groups (i.e., importantly, no controls were obese). The mean IAAF performance scores of the runners confirmed them as being high-level performers (i.e., 10,000 M [< 28:30, < 34:00] or marathon [< 2:14:00, < 2:40:00] equivalences, for the men and women, respectively).

Table 2 for males and Table 3 for females present the prevalence of *Z* score values in three categories of bone measurements (LS-, RF-, and TB-BMD) and the *p* values for Fisher's exact test from the Chi-square analysis. There were no significant differences in the prevalence of low LS-, RF-, or TB-BMD values between male runners and male controls nor between the female runners and female controls.

Tables 4 and 5 present the blood work findings for each of the study groups, specifically, the complete blood count (CBC, select values) and hormonal values and corresponding reference ranges. All the hormonal and CBC values measured were in the expected range for the specific study group. Male athletes did have higher cortisol ($p = 0.005$) and lower red blood cells ($p = 0.003$), hematocrit ($p = 0.014$), and estradiol ($p = 0.049$) values compared to male controls. For

Table 1 Physical characteristics of male and female participants, IAAF scores, and maximal oxygen consumption of athletes

	Male athletes		Male controls		*p*	Female athletes		Female controls		*p*
	N	Mean ± SD	*N*	Mean ± SD		*N*	Mean ± SD	*N*	Mean ± SD	
Age (years)	30	28.0 ± 3.75	29	24.1 ± 3.83	0.001*	26	28.6 ± 6.34	29	24.97 ± 5.74	0.311
Mass (kg)	30	57.7 ± 6.07	29	62.5 ± 10.11	0.030*	26	51.7 ± 5.04	29	63.37 ± 9.12	0.001*
BMI (kg m^{-2})	30	19.5 ± 1.84	29	21.82 ± 4.97	0.024*	26	19.5 ± 1.98	29	23.28 ± 3.19	0.001*
IAAF score (*points*)	28	1096.8 ± 62.3		–		23	1037.3 ± 106.8		–	
VO_{2max} ml kg^{-1} min^{-1}	26	67.0 ± 4.5		–		20	52.1 ± 5.8		–	

BMI body mass index, *IAAF* International Association of Athletics Federations (rebranded as World Athletics since June 2019)

*$p < 0.05$ difference between athletes and controls for each sex

Table 2 Prevalence of male participants bone mineral density values

	Male athletes	Male controls	Total	*p* value for the Fisher's exact test
LS-BMD				
Z > − 1	14	20	34	1.000
Z − 1 to − 2	5	7	12	
Z < − 2	1	2	3	
Total	20	29	49	
RF-BMD				
Z > − 1	18	24	42	0.820
Z − 1 to − 2	2	3	5	
Z < − 2	0	2	2	
Total	20	29	49	
TB-BMD				
Z > − 1	29	26	55	0.293
Z − 1 to − 2	1	3	4	
Z < − 2	0	0	0	
Total	30	29	59	

LS-BMD lumbar spine bone mineral density, *RF-BMD* right femur bone mineral density, *TB-BMD* total body bone mineral density

Table 3 Prevalence of female participants bone mineral density values

	Female athletes	Female controls	Total	*p* value for the Fisher's exact test
LS-BMD				
Z > − 1	16	21	37	0.634
Z − 1 to − 2	7	6	13	
Z < − 2	3	2	5	
Total	26	29	55	
RF-BMD				
Z > − 1	19	23	42	0.820
Z − 1 to − 2	5	3	8	
Z < − 2	2	3	5	
Total	26	29	55	
TB-BMD				
Z > − 1	23	25	48	1.000
Z − 1 to − 2	3	3	6	
Z < − 2	0	1	1	
Total	26	29	55	

LS-BMD lumbar spine bone mineral density, *RF-BMD* right femur bone mineral density, *TB-BMD* total body bone mineral density

females, the athletes had higher neutrophils ($p = 0.015$), but lower lymphocytes ($p = 0.036$) and insulin ($p = 0.001$) levels compared to female controls.

Dietary intake values, absolute and relative to body mass (uncorrected and corrected) are presented in Table 6. We found that male athletes had higher calcium ($p = 0.05$) intake compared to the control group. Female athletes had higher carbohydrates ($p = 0.035$) and calcium ($p = 0.039$) intake compared to the control group. No other runner vs. control differences were noted for assessed measures.

Discussion

The purpose of this study was to examine and compare the prevalence of select Triad–RED–S components/risk factors in Kenyan male and female distance runners to corresponding control groups with special interest on bone indices, hormonal markers, and energy intake commonly associated with RED-S. We hypothesized that our key finding would be Kenyan male and female distance runners would present lower bone mineral indices compared to ethnically similar control groups. The data from the present study did not support this hypothesis.

BMD

We found that there was a remarkably similar prevalence of low BMD (*Z* score < − 1) in the male and female Kenyans, but no difference in prevalence between male runners and male controls and female runners and female controls. It has been shown previously that Kenyan elite runners have low BMD (Tam et al. 2017). However,

Table 4 Male (M) and female (F) participants' complete blood count (CBC) values

	M athletes $N=28$		M controls $N=29$		p	Reference range
	Mean ± SD	Range	Mean ± SD	Range		
WBC (10^3 μL^{-1})	5.24 ± 1.45	3.30–9.96	5.46 ± 1.55	3.29–5.46	0.597	4.5–11
RBC (10^6 μL^{-1})	5.51 ± 0.33	4.77–6.17	5.84 ± 0.46	5.19–6.84	0.003*	4.5–5.90
HGB (g dL^{-1})	16.29 ± 0.99	13.90–18.86	16.65 ± 1.64	10.20–19.40	0.339	13.5–17.5
HCT (%)	47.55 ± 2.74	41.32–54.57	49.82 ± 3.89	36.70–57.30	0.014*	41.0–53.0
Neutrophils (%)	49.92 ± 14.07	24.20–80.50	44.98 ± 10.10	27.20–76.40	0.132	40–70
Lymphocytes (%)	39.24 ± 12.44	8.90–61.90	43.88 ± 8.76	16.60–60.50	0.109	22–44
Monocytes (%)	6.86 ± 1.42	4.30–9.60	6.92 ± 1.55	3.70–9.60	0.894	4–11
Eosinophils (%)	2.80 ± 1.86	0.60–8.30	3.20 ± 2.35	0.80–11.10	0.484	0–8
Basophils (%)	1.15 ± 1.08	0.40–6.20	1.02 ± 0.44	0.50–2.50	0.523	0–3
	F athletes $N=24$		F controls $N=29$		p	Reference range
	Mean ± SD	Range	Mean ± SD	Range		
WBC (10^3 μL^{-1})	5.98 ± 2.05	2.58–10.17	5.65 ± 1.46	3.43–10.22	0.845	4.5–11.0
RBC (10^6 μL^{-1})	5.01 ± 0.47	3.73–5.75	4.99 ± 0.56	2.79–5.77	0.869	4.00–5.20
HGB (g dL^{-1})	14.01 ± 1.65	9.49–17.10	13.98 ± 2.08	7.01–16.76	0.951	12.5–16.0
HCT (%)	42.05 ± 4.23	31.68–49.00	40.74–9.36	2.57–50.83	0.530	36.0–46.0
Neutrophils (%)	49.39 ± 10.02	28.90–67.10	43.52 ± 6.96	26.20–55.40	0.015*	40–70
Lymphocytes (%)	40.30 ± 9.94	25.10–62.80	45.26 ± 6.71	35.30–64.00	0.036*	22–44
Monocytes (%)	6.98 ± 1.69	3.80–10.10	7.43 ± 2.09	0.60–12.60	0.404	4–11
Eosinophils (%)	2.15 ± 1.06	0.70–4.00	2.78 ± 1.66	0.90–7.80	0.119	0–8
Basophils (%)	1.16 ± 0.99	0.40–4.00	0.86 ± 2.73	0.30–1.70	0.125	0–3

WBC white blood cells, RBC red blood cells, HGB hemoglobin, HCT hematocrit

* $p < 0.05$ difference between athletes and controls

similarly, to our study, Goodwin et al. (2014) found that there were no differences in bone mineral density by region in their study with females between athletes and controls.

In our study, there were some participants (runners and controls) with very low BMD (Z score < 2), but most did not have excessively low BMD values across our various measurement sites. It is known that long periods of inadequate energy consumption (low energy availability) can result in a low BMD status (Mountjoy et al. 2014). We speculate that our lack of substantial numbers of participants having low BMD could be the resultant of the combination of factors: (1) osteogenic effects of running (2) adequate intake of carbohydrates, which has been shown to acutely effects bone turnover (Hammond et al. 2019; Langan-Evans et al. 2021). That said, the overall EI of our participants (runners vs. controls) seems relatively low (see subsequent discussion). These findings suggest that further research is warranted on this topic with our unique sample population of runners. Finally, statistic testing for differences in prevalence of bone health indices between the sexes as that comparison was not conducted as it was not a focus of our work.

CBC and hormones

Our intent in examining the complete blood counts (CBC) was to assure ourselves that our male and female samples (athlete and control) were healthy and not suffering any illness; and, or displaying signs of dehydration. Furthermore, some evidence points to athletes with Triad–RED–S displaying immune systems dysfunctions as a consequence of their energy availability imbalance (Sangenis et al. 2006). While we did see some significant differences between athletes vs. controls within each sex, these were slight in magnitude and within normal clinical ranges (Kratz et al 2004; Feingold et al. 2000). Overall, these data suggest that our samples were normal in immune function and not dehydrated at the time of the study.

The hormonal values (see Table 5) we observed were variable, but relatively unremarkable as all values for athletes and controls were within clinical norms (Feingold et al. 2000). We did find that cortisol ($p = 0.005$) levels were somewhat higher, and estradiol ($p = 0.049$) levels lower in male athletes compared to male controls; and, insulin ($p = 0.001$) levels were lower in female athletes compared to female controls, but as noted, all within

Table 5 Male (M) and female (F) participants' hormonal values

	M athletes			M controls			*p*	Reference range
	N	Mean ± SD	Range	*N*	Mean ± SD	Range		
TSH (mU L^{-1})	27	2.46 ± 0.83	0.73–4.96	23	1.72 ± .0.73	0.56–3.40	0.255	0.5–4.7
T_3 (nmol L^{-1})	27	1.81 ± 0.24	1.25–2.26	22	1.83 ± 0.02	1.46–2.14	0.733	0.92–2.78
T_4 (nmol L^{-1})	27	87.44 ± 15.33	53.50–123.80	23	86.48 ± 10.99	62.80–113.30	0.790	58–140
Cort (nmol L^{-1})	27	303.05 ± 97.35	170.90–595.20	23	225.36 ± 101.98	81.00–458.20	0.005*	138–690
Ins (mU L^{-1})	27	5.88 ± 4.14	2.00–17.80	22	7.53 ± 13.03	1.10–73.20	0.531	2–20
FSH (U L^{-1})	27	2.67 ± 1.16	0.20–7.10	23	2.23 ± 1.00	0.50–4.70	0.134	1.0–12.0
LH (U L^{-1})	27	5.23 ± 2.29	1.66–10.27	23	4.62 ± 1.66	2.37–8.03	0.248	2.0–12.0
Prol (mU L^{-1})	26	230.65 ± 82.22	137.00–518.00	22	242.66 ± 102.48	115.00–486.00	0.636	0–15
Testo (nmol L^{-1})	28	25.25 ± 6.91	12.10–43.86	23	25.13 ± 5.96	10.34–44.58	0.944	9.36–37.10
E_2 (pmol L^{-1})	27	112.06 ± 56.44	29.10–232.50	22	136.78 ± 33.12	78.90–222.30	0.049*	184
GH (mU L^{-1})	28	4.11 ± 6.72	0.15–26.04	24	1.96 ± 2.60	0.00–9.22	0.115	0.5–17.0
IGF-1 (μg L^{-1})	28	143.49 ± 37.76	85.70–283.00	24	156.47 ± 39.87	91.60–255.90	0.213	182–780
	F athletes			F controls			*p*	Reference range
	N	Mean ± SD	Range	*N*	Mean ± SD	Range		
TSH (mU L^{-1})	23	1.90 ± 1.02	0.27–5.11	29	1.68 ± 0.86	0.65–4.16	0.402	0.5–4.7
T_3 (nmol L^{-1})	22	1.97 ± 0.58	1.47–4.21	29	1.99 ± 0.29	1.40–2.54	0.861	0.92–2.78
T_4 (nmol L^{-1})	23	100.15 ± 17.52	64.20–136.20	29	96.87 ± 17.77	52.00–122.20	0.509	58–140
Cort (nmol L^{-1})	23	242.98 ± 77.65	127.00–489.90	28	265.60 ± 93.44	144.0–547.30	0.358	138–690
Ins (mU L^{-1})	22	5.47 ± 2.78	2.00–12.30	28	12.69 ± 6.81	4.40–34.50	0.001*	2–20
FSH (U L^{-1})	23	10.40 ± 29.42	0.10–144.80	28	8.23 ± 18.20	0.00–100.40	0.749	3.0–26.0
LH (U L^{-1})	23	7.50 ± 14.25	0.16–71.49	28	8.86 ± 13.84	0.00–75.66	0.733	2.0–64.0
Prol (mU L^{-1})	22	336.55 ± 231.36	129.00–981.00	29	397.29 ± 269.53	92.0–1240.00	0.833	0–20
Testo (nmol L^{-1})	23	0.78 ± 0.95	0.00–4.09	28	0.78 ± 781	0.00–4.23	0.977	0.21–2.98
E_2 (pmol L^{-1})	22	497.39 ± 751.24	18.40–3677.00	29	402.03 ± 712.36	0.00–3780.00	0.251	184–1626
GH (mU L^{-1})	24	8.13 ± 9.38	0.52–30.37	29	6.02 ± 8.20	0.34–37.68	0.386	0.5–17.0
IGF-1 (μg L^{-1})	24	185.52 ± 71.19	84.20–326.40	29	208.87 ± 59.20	66.80–346.30	0.198	182–780

TSH thyroid-stimulating hormone, T_3 triiodothyronine, T_4 thyroxine, *Cort* cortisol, *Ins* insulin, *FSH* follicle-stimulating hormone, *LH* luteinizing hormone, *Prol* prolactin, *Testo* testosterone, E_2 estradiol, *GH* growth hormone, *IGF-1* insulin-like growth factor-1

*$p < 0.05$ difference between athletes and controls

the normal expected range for healthy men and women within each of the respective hormonal measures (Feingold et al. 2000). This was a somewhat surprising finding as we had surmised that based on prior literature (specifically in women), there would be anomalies in hormones levels. Why none were seen is unclear, but as our BMD values were relatively normal, it could be expected that our hormones values would follow suit. Alternative possible explanations could also relate to: (1) Ethnic-race considerations—that is, could hormonal norms for African's differ somewhat from those based on Caucasian populations? (2) Temporal factors—while low EI occurred in our participants, perhaps, it was not manifested long enough to result in hormonal disturbances. Further work is warranted to pursue this speculation on our part and determine the underlying factor(s) for these findings.

Dietary energy intake

There were no differences in overall EI between runners and controls in our study; although, we found extremely low EI values across all study groups in examining either the uncorrected or corrected results. We had anticipated potential errors in the dietary data based on previous reports (Mertz et al. 1991; Fudge et al. 2006; Scagliusi et al. 2006; Pfrimer et al. 2015; Orcholski et al 2015) and as such applied a correction factor to adjust; nonetheless, we were surprised by the relatively low caloric consumption by all our participants. That said, Christensen et al. (2002) found somewhat similar findings in male adolescent Kenyan runners; although, other studies have reported higher EI than we found (Fudge et al. 2006). There are multiple factors that could potentially contribute to this difference in study

Table 6 Dietary intake of three highest energy intake days for male and female athletes and respective controls (respective relative value below each measurement)

	Male athletes $N=27$ Mean ± SD	Male controls $N=26$ Mean ± SD	p	Female athletes $N=21$ Mean ± SD	Female controls $N=26$ Mean ± SD	p
Energy intake (kcal day^{-1})	1581.46 ± 439.6 (2313.1 ± 642.7)	1453.5 ± 378.2 (2125.0 ± 553.0)	0.262	1446.9 ± 402.8 (2115.3 ± 588.9)	1341.9 ± 245.54 (1961.9 ± 359.0)	0.277
Relative value (kcal day^{-1} kg^{-1})	27.8 ± 8.5 (40.6 ± 12.4)	23.9 ± 6.9 (34.9 ± 10.1)	0.073	28.1 ± 7.4 (41.1 ± 10.8)	21.7 ± 5.9 (31.7 ± 8.6)	0.002*
Protein (g)	67.4 ± 19.0 (98.5 ± 27.8)	67.4 ± 19.7 (98.6 ± 28.9)	0.991	58.1 ± 22.6 (84.9 ± 33.1)	58.4 ± 14.2 (85.3 ± 20.8)	0.959
Relative value (g kg^{-1})	1.2 ± .4 (1.8 ± 0.6)	1.1 ± .4 (1.6 ± 0.6)	0.455	1.1 ± .4 (1.6 ± 0.6)	1.0 ± .3 (1.5 ± 0.4)	0.123
Fat (g)	36.9 ± 19.3 (53.9 ± 28.3)	39.3 ± 23.2 (57.4 ± 33.9)	0.684	37.7 ± 26.6 (55.0 ± 38.8)	39.1 ± 18.3 (57.22 ± 26.8)	0.882
Relative value (g kg^{-1})	0.7 ± .4 (1.0 ± 0.6)	0.6 ± .4 (.9 ± 0.6)	0.970	0.7 ± .5 (1.0 ± 0.7)	0.6 ± .4 (0.9 ± 0.6)	0.546
Carbohydrates (g)	263.6 ± 83.3 (385.4 ± 121.8)	238.7 ± 62.3 (349.0 ± 91.0)	0.226	243.8 ± 63.6 (356.5 ± 93.0)	209.9 ± 43.4 (306.9 ± 63.4)	0.035*
Relative value (g kg^{-1})	4.6 ± 1.5 (6.7 ± 2.2)	3.9 ± 1.1 (5.7 ± 1.6)	0.058	4.7 ± 1.2 (6.9 ± 1.8)	3.4 ± .7 (5.0 ± 1.0)	0.001*
Dietary fiber (g)	34.6 ± 10.1 (50.6 ± 14.8)	32.6 ± 6.7 (47.6 ± 9.8)	0.400	29.9 ± 7.6 (43.7 ± 11.2)	28.5 ± 10.6 (41.7 ± 15.5)	0.625
Relative value (g kg^{-1})	0.6 ± .2 (0.9 ± 0.3)	0.5 ± .1 (0.7 ± 0.1)	0.096	0.6 ± .1 (0.9 ± 0.1)	0.5 ± .2 (0.7 ± 0.2)	0.013*
Sodium (mg)	1221.3 ± 447.7 (1785.5 ± 654.5)	1173.9 ± 422.9 (1716.2 ± 618.3)	0.694	1018.1 ± 418.6 (1488.5 ± 612.1)	1153.2 ± 460.1 (1686.0 ± 672.6)	0.303
Relative value (mg kg^{-1})	21.5 ± 8.6 (31.4 ± 12.6)	19.3 ± 7.6 (28.2 ± 11.1)	0.325	19.9 ± 8.1 (29.1 ± 11.8)	18.8 ± 9.2 (27.5 ± 13.5)	0.672
Potassium (mg)	1669.6 ± 723.9 (2441.0 ± 1058.3)	1332.2 ± 538.2 (1947.7 ± 786.9)	0.061	1649.8 ± 635.9 (2411.9 ± 929.6)	1400.9 ± 544.2 (2048.1 ± 795.6)	0.155
Relative value (mg kg^{-1})	29.3 ± 13.0 (42.8 ± 19.0)	22.0 ± 10.0 (32.2 ± 14.6)	0.027*	31.9 ± 11.6 (46.6 ± 16.2)	22.6 ± 9.8 (33.0 ± 14.3)	0.005*
Calcium (mg)	258.4 ± 150.2 (377.8 ± 219.6)	161.9 ± 75.2 (236.7 ± 110.0)	0.005*	263.8 ± 110.4 (385.7 ± 161.3)	191.6 ± 120.1 (280.1 ± 175.6)	0.039*
Relative value (mg kg^{-1})	4.5 ± 2.7 (6.6 ± 3.9)	2.6 ± 1.3 (3.8 ± 1.9)	0.003*	5.1 ± 2.1 (7.5 ± 3.0)	3.1 ± 2.0 (4.5 ± 2.9)	0.002*

Corrected values in brackets; Relative values = per kg body weight

*$p < 0.05$ difference between athletes and controls within each sex

findings. First, in our study, the participants had a free living diet, whereas in some studies, dietary food was provided by the schools where participants were enrolled and as such could be consumed ad libitum. Second, Christensen and colleagues followed up with a 24 h recall interview which could have ensured a more accurate and detailed food consumption record. We too attempted to improve the accuracy of our dietary data by insuring explanations of diet records procedures were given by native Kenyan language(s) speakers.

Macronutrients

We found that the protein intake was elevated and composed 16.7% of male runners, 15.2% of female runners, 17.3% of male controls, and 16.7% of female controls' dietary intake. This is in accordance with the studies done with Kenyan male runners by Fudge et al. (2006) and Christensen et al. (2002) where protein composed 15.3% and 13% of dietary intake, respectively. In addition, the carbohydrate intake in our study was also in accordance with these previous studies—64.4% for male runners, 65.6% for female runners, 62.0% for male control, and 60.8% for female controls from the total EI as compared to 71% in Christensen (2002) and colleagues study and 67.3% in Fudge et al.'s study (2006). Our findings do not shed light on this issue, but do show macro- and micro-nutrient intake among Kenyan athletes is remarkably similar to their non-athletic country men-women.

Limitations and strengths

As with any study, there were short comings within our research. A key limitation of our study was the self-reported aspects of our dietary records which could have affected the accuracy of the information. Due to the observational nature of this study, we cannot demonstrate a

causative linkage between EI and metabolic or endocrine alterations. Also, we did not collect hormonal contraception nor menstrual cycle data. Finally, we had a varying number of subjects across several of our outcome measures, due to technical issues, causing incomplete data sets and as such affecting the robustness of our findings.

One of the strengths of our study is the overall high athletic level of the participants. To give perspective as to the level of our participants, we translated the average IAAF scores into marathon running times, which yielded a 2:39:57 for women and 2:13:41 for men in this study according to the IAAF Scoring Tables of Athletics. To add even more perspective, these marathon times would have given 8th and 13th places, respectively, at the 2019 World Athletics championship. Furthermore, our participants represent not just high-level athletes, but a racial group that has been understudied in the sport science community. Another strength of our study was the usage of DXA for BMD measurements, especially the segmental data of lumbar spine and right femur in addition to total body BMD as the latter may underestimate the prevalence and presence of low BMD (Tam et al. 2017).

Conclusions

Our current findings do not show a statistical linkage between EI and high prevalence of low BMD in male and female Kenyan runners. We did find that high-level Kenyan male and female runners do have, in addition to low overall EI, some occurrences of low BMD, which if were to be of a chronic nature could increase their risk for developing low EA. Nevertheless, even with the low EI observed, there were no major Triad—RED-S risk factors detected in our athletes, which might suggest that the current criteria for Triad–RED–S diagnosis may not be entirely applicable for athletes of African ethnicity. This latter point is in need of future investigation.

Author contributions MM and ACH conceived and designed research. MM, SS, DWH, RO, ACH and ARL conducted experiments. ACH and LÕ analyzed data and wrote the manuscript. All authors read and approved the manuscript.

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Availability of data and materials The data that support the findings of this study are available from the corresponding author, upon reasonable request.

Code availability Not applicable.

Declarations

Conflict of interest Authors' have no conflict of interests to declare.

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