Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating

Esther N. Muia, Hattie H. Wright, Vincent O. Onywera & Elizabeth N. Kuria

To cite this article: Esther N. Muia, Hattie H. Wright, Vincent O. Onywera & Elizabeth N. Kuria (2015): Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating, Journal of Sports Sciences, DOI: 10.1080/02640414.2015.1065340

To link to this article: http://dx.doi.org/10.1080/02640414.2015.1065340

Published online: 08 Jul 2015.

Article views: 150

View related articles

View Crossmark data
Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating

ESTHER N. MUIA¹, HATTIE H. WRIGHT²,³, VINCENT O. ONYWERA⁴ & ELIZABETH N. KURIA¹

¹Department of Food, Nutrition and Dietetics, School of Applied Human Sciences, Kenyatta University, Nairobi, Kenya, ²Centre of Excellence in Nutrition, North-west University, Potchefstroom, South Africa, ³School of Health and Sport Sciences, Faculty of Health Science, University of the Sunshine Coast, Sippy Downs, Queensland, Australia and ⁴Department of Recreation Management and Exercise Science, School of Applied Human Sciences, Kenyatta University, Nairobi, Kenya

(Accepted 18 June 2015)

Abstract
Limited data are available on the female athlete triad (Triad) in athletes from minority groups. We explored subclinical and clinical Triad components amongst adolescent elite Kenyan athletes (n = 61) and non-athletes (n = 49). Participants completed demographic, health, sport and menstrual history questionnaires as well as a 5-day weighed dietary record and exercise log to calculate energy availability (EA). Ultrasound assessed calcaneus bone mineral density (BMD). Eating Disorder Inventory subscales and the Three-Factor Eating Questionnaire’s cognitive dietary restraint subscale measured disordered eating (DE). EA was lower in athletes than non-athletes (36.5 ± 4.5 vs. 39.5 ± 5.7 kcal ⋅ kg FFM⁻¹ ⋅ d⁻¹, P = 0.003). More athletes were identified with clinical low EA (17.9% vs. 2.2%, OR = 9.5, 95% CI 1.17–77, P = 0.021) and clinical menstrual dysfunction (32.7% vs. 18.3%, χ² = 7.1, P = 0.02). Subclinical (75.4% vs. 71.4%) and clinical DE (4.9% vs. 10.2%, P = 0.56) as well as BMD were similar between athletes and non-athletes. More athletes had two Triad components than non-athletes (8.9% vs. 0%, OR = 0.6, 95% CI 0.5–6.9, P = 0.05). Kenyan adolescent participants presented with one or more subclinical and/or clinical Triad component. It is essential that athletes and their entourage be educated on their energy needs including health and performance consequences of an energy deficiency.

Keywords: young athlete, bone mineral density, energy availability, menstrual function

Introduction
Physical activity promotes good health and is recommended for women of all ages (Loucks & Nattiv, 2005). Excessive physical activity without a concurrent increase in appropriate dietary intake is, however, associated with energy deficiency. Energy deficiency may result in health and performance declines over the long term (Mountjoy et al., 2014). The female athlete triad (Triad) refers to the interrelation between energy status, bone health and menstrual function (Nattiv et al., 2007) which moves along a continuum from a healthy to a diseased state. The clinical outcomes of the Triad are low-energy availability (with or without an eating disorder), osteoporosis and functional hypothalamic amenorrhea (Nattiv et al., 2007). Each disorder on its own is of significant medical concern, but when all three components are present the effects have greater potential for serious and prolonged impacts on health and performance. Female athletes with insufficient energy intake to support their exercise energy expenditure are at risk of low-energy availability which is the cornerstone of the Triad. Female athletes in sports, which emphasise a thin body, or those competing in sports, where low body weight is a prerequisite such as gymnastics, ballet, figure skating, swimming and distance running, may be at an increased risk for energy availability (Torstveit & Sundgot-Borgen, 2005).

There are limited data on the presence of the components (subclinical and clinical) of the Triad in African athletes as most studies to date have focused on those of Caucasian descent. Recently, an appeal was made for research on the components of the Triad and energy availability in non-Caucasian athletes (Mountjoy et al., 2014). Race has the potential to influence many of the Triad components, for example, due to sociocultural factors black women in general are more tolerant of a
bigger body size which may reduce their risk for disordered eating and low-energy availability (Puoane, Fourie, Shapiro, & Tshaka, 2005). In fact, Hulley, Currie, Njenga, and Hill (2007) reported a low risk for disordered eating among adult Kenyan runners (11.1%) compared to Caucasian runners (48.1%) from the United Kingdom. Due to acculturation, the risk for disordered eating may increase in black African women and adolescents (Bennett, Sharpe, Freeman, & Carson, 2004; Fawzi, Hashim, Fouad, & Abdel-Fattah, 2010). Race also plays a role in bone mineral density (BMD) as African blacks have greater BMD than Caucasians (Micklefield, Norris, & Pettifor, 2011). This may reduce the risk for low BMD and stress fractures in African athletes.

From the early 1990s, Athletics Kenya, a member of the International Athletics Association Federation, put programmes in place for competitive sport due to the successes of elite Kenyan distance runners internationally (Saltin et al., 1995). This has resulted in an increased number of young Kenyan athletes competing in sport and striving to become elite athletes. Most of these young athletes are in boarding schools and receive similar meals to non-athletes which may increase their risk for a relative energy deficiency. As a contribution to the ongoing research on the Triad among black athletes, this study identifies and evaluates subclinical and clinical components of the Triad for the first time in Kenyan adolescent female athletes.

**Methods**

**Participants and study design**

Volunteer middle and long-distance (≥1500 m) athletes (n = 61) with a median age of 16 (interquartile range = 16–17) years and non-athletes (n = 49) of 17 (interquartile range = 16–17) years were recruited from four boarding schools and two secondary schools in Iten, Rift valley Province, Kenya. Previous research shows that many Kenyan athletes, and particularly those involved in international competition, are Kalenjin from the Rift Valley province (Onywera, Scott, Boit, & Pitsiladis, 2006). The study was approved by the Kenyatta University’s Ethical Research Committee and the District Education Officer. Participants and head teachers of schools (the legal guardians of the participants) gave written informed consent. Inclusion criteria were athletes that had competed at regional or higher level in the previous year (2012) or were scheduled to compete in 2013. The control group were students not involved in any organised sport and are referred to as non-athletes. Participants completed a series of questionnaires under supervision of the researcher. A socio-demographic questionnaire included information on training hours, history of medically diagnosed stress fractures, type of school attended, number of siblings and parents’ educational level.

**Menstrual patterns**

Self-reported menstrual function gathered information on age of menarche, frequency and characteristics of menses during the preceding 12 months. Eumenorrhoea was defined as a menstrual cycle every 21–45 days; primary amenorrhea as the absence of menstrual cycle by age 15; secondary amenorrhea as no menstrual cycle for more than 90 continuous days after menarche; and oligomenorrhea as cycles longer than 45 days (American College of Obstetricians and Gynaecologist, 2006). Participants were classified with clinical menstrual dysfunction if they reported primary, secondary or oligomenorrhea (Gibbs, Williams, & De Souza, 2013).

**Disordered eating**

Disordered eating was assessed using the Eating Disorder Inventory-3 (EDI-3) drive for thinness, bulimia and body dissatisfaction subscales (Garner, 1983), as well as the cognitive dietary restraint subscale of the Three-Factor Eating Questionnaire (TFEQ) (Stunkard & Messick, 1985). These subscales have been used to assess disordered eating among athletes in a non-clinical setting (Vescovi, Scheid, Hontscharuk, & De Souza, 2008). The Eating Disorder Inventory has also been shown to be a valid indicator of disordered eating in black African women (Fawzi et al., 2010). Participants were categorised with subclinical disordered eating if they scored ≥7 for the EDI-3 drive for thinness and/or ≥9 TFEQ cognitive dietary restraint CDR subscales and/or had a BMI <17.5 kg m⁻². Clinical disordered eating was identified as scoring ≥15 for the EDI-3 drive for thinness and/or ≥14 for the EDI-3 body dissatisfaction subscales (De Souza et al., 2014; Gibbs, Williams, & De Souza, 2013).

**Weight, height and body composition**

Body weight and height were measured according to the International Society for the Advancement of Kinanthropometry’s (ISAK) International Standards for Anthropometric Assessment Criteria (Marfell-Jones, Olds, Stewart, & Carter, 2006). Weight was recorded with a calibrated digital beam scale (Seca 874 digital medical scale, USA) and height with stadiometer (Seca 213 portable scale, USA) to the
meaning daily exercise energy expenditure was not adjusted for resting energy expenditure. Heart rate and activity monitors were not available due to limited resources.

Energy availability was calculated with the following formula:

\[
\text{Energy availability} = \frac{\text{Mean daily energy intake, kcal} \cdot \text{d}^{-1} - \text{mean daily exercise energy expenditure, kcal} \cdot \text{d}^{-1}}{\text{Fat-free mass (FFM, kg)}^{-1}} \times \text{Fat-free mass (kg)}
\]

Participants with energy availability <30 kcal · kg FFM\(^{-1} \cdot \text{d}^{-1}\) were categorised as having clinically low-energy availability, 30–45 kcal · kg FFM\(^{-1} \cdot \text{d}^{-1}\) with subclinical low-energy availability and ≥45 kcal · kg FFM\(^{-1} \cdot \text{d}^{-1}\) with optimal energy availability, as suggested for sedentary women (Gibbs, Williams, & De Souza, 2013; Loucks, 2007).

**Triad components**

Table I summarises the criteria used to classify participants with subclinical and/or clinical energy availability, disordered eating and menstrual dysfunction.

**Statistical analysis**

Data were analysed for normality using the Statistical Package for Social Sciences®, version 21 for Windows software program, and normally distributed data are expressed as mean and standard deviation. Non-parametric data (age, age of menarche, number of siblings, training hours per week, daily energy intake, EDI-3 bulimia and body dissatisfaction subscales) are reported as median and
Tables and figures

Table I. Clinical and subclinical definitions of energy availability, disordered eating and menstrual dysfunction.

<table>
<thead>
<tr>
<th>Components</th>
<th>Subclinical definition</th>
<th>Clinical definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-energy availability (EA)</td>
<td>EA ≥ 30 – ≤ 45 kcal · kg FFM⁻¹ · d⁻¹</td>
<td>EA &lt; 30 kcal · kg FFM⁻¹ · d⁻¹</td>
</tr>
<tr>
<td>Disordered eating behaviour</td>
<td>EDI-3 DT ≥ 7 and/or EDI-3 BD ≥ 9, TFEQ CDR ≥ 9</td>
<td>EDI-3 DT ≥ 15, EDI-3 BD ≥ 14</td>
</tr>
<tr>
<td>Menstrual dysfunction</td>
<td>Not measured</td>
<td>Primary amenorrhea: no menses by ≥ 15 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Secondary amenorrhea: Absence of menstrual cycle &gt; 90 continuous days after menarche</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oligomenorrhea: menstrual cycles &gt; 45 days</td>
</tr>
</tbody>
</table>

Notes: As classified by Gibbs, Williams, and De Souza (2013). FFM, fat-free mass; EDI, Eating Disorder Inventory; DT, drive for thinness; BD, body dissatisfaction; TFEQ, Three-Factor Eating Questionnaire; CDR, cognitive dietary restraint; BMI, body mass index.

Interquartile ranges. Categorical data are expressed as percentage of the total group. Differences between athletes and non-athletes were determined with χ² analysis for categorical data. If more than 20% of the contingency cells had a value < 5, a Fisher’s exact test was used to compare categorical data between groups. Data from Fisher’s exact tests are presented as odds ratio and 95% confidence intervals (CIs). T-tests compared parametric continuous variables and Mann-Whitney U-tests compared non-parametric continuous variables between groups. Effect sizes were calculated for all continuous variables and reported when differences between groups were non-significant but had a medium (r = 0.3) or large (r = 0.5) effect size. Statistical significance was set at P < 0.05.

Results

Participant characteristics

Of the participants, 75% (n = 82/110) were enrolled in boarding school. Athletes had more siblings (5 (interquartile range = 3.5–6)) vs. 4 (interquartile range = 3–5.5), P = 0.03) than non-athletes. Fewer mothers from athletes had tertiary education than non-athletes (16% vs. 38%, χ² = 9.16, P = 0.010). There was a strong trend that fewer fathers of athletes had tertiary education than those of non-athletes (28% vs. 50%, χ² = 11.8, P = 0.06) and less mothers from athletes had formal employment than non-athletes (13% vs. 54%, χ² = 22, P = 0.000). On average, the median weekly hours exercised by athletes were 4 (interquartile range = 3–8). A typical training day consisted out of stretching before and after running sessions, a 1 h run at moderate to high intensity in the morning and up to a 2 h run at a very easy to moderate intensity in the evening for at least 4 days a week.

Body composition and energy availability

Nine participants (five athletes, four non-athletes) did not present themselves for anthropometric measurements. Athletes were taller (P < 0.05), had a lower body weight, BMI and FFM compared to non-athletes (Table II). Energy availability and energy intake was significantly lower in athletes

Table II. Body composition and energy status of athletes and non-athletes.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Athletes (n = 56)</th>
<th>Non-athletes (n = 45)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>159.2 ± 5.8</td>
<td>157.2 ± 4.7</td>
<td>0.018</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>48.5 ± 6.1</td>
<td>52.1 ± 6.4</td>
<td>0.004</td>
</tr>
<tr>
<td>Body mass index (kg · m⁻²)</td>
<td>19.9 ± 2.0</td>
<td>21.8 ± 2.3</td>
<td>0.000</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>41.0 ± 4.7</td>
<td>43.2 ± 4.8</td>
<td>0.018</td>
</tr>
<tr>
<td>Energy availability (kcal · kg FFM⁻¹ · d⁻¹)</td>
<td>36.5 ± 4.5</td>
<td>39.5 ± 5.7</td>
<td>0.003</td>
</tr>
<tr>
<td>Mean energy intake (kcal · d⁻¹)</td>
<td>2028 (2090–2155)</td>
<td>2080 (2150–2212)</td>
<td>0.002</td>
</tr>
<tr>
<td>Mean total daily energy expenditure (kcal)</td>
<td>1427 ± 238</td>
<td>1211 ± 168</td>
<td>0.000</td>
</tr>
<tr>
<td>Mean exercise energy expenditure (kcal · d⁻¹)</td>
<td>591 ± 94.5</td>
<td>490 ± 85.6</td>
<td>0.000</td>
</tr>
<tr>
<td>Predicted basal metabolic rate (kcal · d⁻¹)</td>
<td>1396 ± 79</td>
<td>1496 ± 96</td>
<td>0.000</td>
</tr>
<tr>
<td>Goldberg cut-off</td>
<td>1.5 ± 0.1</td>
<td>1.4 ± 0.1</td>
<td>0.202</td>
</tr>
</tbody>
</table>

Notes: Parametric variables are reported as means and standard deviation. Basal metabolic rate calculated with the Schofield equation (Schofield, 1985).

*Non-parametric variable reported as median and interquartile ranges (25th–75th percentile).

FFM, fat-free mass.
Table III. A typical daily menu for athletes and non-athletes.

<table>
<thead>
<tr>
<th>Meal time</th>
<th>Menu</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast (07:00 am)</td>
<td>A cup of tea (full-cream milk and sugar) and buttered white bread</td>
</tr>
<tr>
<td>Morning snack (10:00 am)</td>
<td>A cup of tea (full-cream milk and sugar) (white buttered bread optional student has to buy own)</td>
</tr>
<tr>
<td>Lunch (12:45 pm)</td>
<td>Ugali (stiff cooked maize flour) served with fried cabbage or kale OR Fried githeri (mixture of boiled maize and beans) OR Boiled rice served with meat stew (has potatoes and carrots added) or bean stew (with or without potatoes, peas and carrots) and fried cabbage or kale</td>
</tr>
<tr>
<td>Dinner (06:30 pm)</td>
<td>Fried bean stew (occasionally the stews have potatoes, peas and carrots added to them)</td>
</tr>
<tr>
<td>Bed time drink (09:00 pm)</td>
<td>A cup of Cocoa/Milo (made with boiled water without milk)</td>
</tr>
</tbody>
</table>

Note: The items for lunch and dinner are interchanged on a daily basis. Meat is served not more than twice weekly. Occasionally, a fruit in season is given at lunch and dinner. Common fruits are oranges, mangoes, bananas and passion fruits.

than non-athletes and exercise energy expenditure higher ($P < 0.05$). Clinical low-energy availability was identified among 17.9% ($n = 10/56$) of the athletes and 2.2% ($n = 1/45$) of non-athletes (odds ratio = 9.5, 95% CI 1.17–77, $P = 0.021$). Subclinical low-energy availability was identified in 76% participants. No participants were identified as over- or under-reporters. Estimated calcaneus BMD tended to be higher in athletes than non-athletes ($0.629 ± 0.1$ vs. $0.592 ± 0.1$ g · cm$^{-2}$, $P = 0.06$). The presence of clinically diagnosed stress fractures reported by athletes and non-athletes was similar (16% vs. 10%, odds ratio = 1.14, 95% CI 0.1–6.0, $P = 0.91$).

Diet records revealed the consumption of a monotonous high carbohydrate diet consisting mainly of ugali (a dish made of maize flour and cooked with water) served with fried vegetable (cabbage or kale) and githeri (a mixture of boiled beans and maize). Meat was consumed twice weekly and a seasonal fruit was eaten sometimes with main meals (Table III). Athletes and non-athletes had similar carbohydrate intake, the average intake for participants was $6.7 ± 1.7$ g · kg body weight$^{-1}$ contributing 70.5% of total daily energy intake.

Disordered eating

No differences were found between athletes and non-athletes in prevalence of subclinical (75.4% vs. 71.4%) and clinical disordered eating (4.9% vs. 10.2%, respectively, $x^2 = 1.1, P = 0.56$ (Table IV). A medium effect size ($r = 0.3$) was, however, found in the bulimia subscale between groups. More athletes (16.1%) than non-athletes (0%) had a BMI < 17.5 kg · m$^{-2}$ (odds ratio = 0.8, 95% CI 0.7–0.9, $P = 0.004$).

Prevalence of Triad components

The prevalence of subclinical and clinical components of the Triad is shown in Table V. A similar percentage of athletes and non-athletes had subclinical energy availability and disordered eating. Significantly more athletes had a combination of clinically low-energy availability and menstrual dysfunction (odds ratio = 0.6, 95% CI 0.5–6.9, $P = 0.05$) than did the non-athletes.

Discussion

The primary findings of this study were that adolescent Kenyan runners and non-athletes present with clinical and subclinical components of the Triad. Athletes and non-athletes had a relative energy deficiency but more athletes had a clinical low-energy availability. To our knowledge, this is the first study to report on the energy status of adolescent Kenyan female high school athletes. Two earlier studies on elite male Kenyan runners reported low-energy intakes and a negative energy balance in these athletes (Mukeshi & Thairu, 1993; Onywera, Kiplamai, Tuitoek, Boit, & Pitsiladis, 2004). Similarly, we found most participants had subclinical low-energy availability (≥30 to <45 kcal · kg FFM$^{-1}$ · d$^{-1}$) and around 21% athletes had clinical low-energy availability (<30 kcal · kg FFM$^{-1}$ · d$^{-1}$), indicating a
relative energy deficiency (Manore et al., 2007; Mountjoy et al., 2014). Additionally, mean daily energy intake of athletes was below the recommended 2300 kcal · d⁻¹ for female athletes to maintain body weight (Manore, Barr, & Butterfield, 2000). Others have also reported insufficient energy intake in adult female endurance athletes (Loucks, 2000; Tomten & Hostmark, 2006). It is noticeable despite the significant difference in energy availability between athletes and non-athletes the average for both groups were below the recommended ≥45 kcal · kg FFM⁻¹ · d⁻¹. This indicates that both groups were operating at suboptimal energy levels that place them at risk of energy deficiency. Limitations associated with the use of weighed food records and subjective exercise logs are recognised and need to be considered when interpreting the results on energy status.

Various factors might have contributed to the insufficient energy intake of these participants. In Kenya, staple foods, such as ugali and githeri, are carbohydrate-rich and high in soluble fibre. These staple foods are often the main meal served in Kenyan boarding schools. It is known that elite Kenyan male runners follow a high carbohydrate diet (76.5% total energy) (Onywera et al., 2004). A high carbohydrate diet intake (70.5% total energy) was also reported in the current study. High carbohydrate diets may increase fullness and satiety but, at the same time, reduce total daily energy intake. Stubbs et al. (2002) found a high carbohydrate diet (62% total energy) reduced ad libitum energy intake in untrained men with and without exercise. Additionally, the suppressive effects on energy intake of prolonged exercise and high carbohydrate diet were additive. It is therefore possible that the suboptimal energy intake of athletes in the current study may due to the combined effect of their high carbohydrate diet and training sessions. Low socio-economic status has been indicated as a driver

Table IV. Eating Disorder Inventory subscales and the Three-Factor Eating Questionnaire subscale scores.

<table>
<thead>
<tr>
<th>Subscales</th>
<th>Athletes (n = 61)</th>
<th>Non-athletes (n = 49)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eating Disorder Inventory subscales</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulimia score</td>
<td>3 (0–6)</td>
<td>2 (0–5)</td>
<td>0.210</td>
</tr>
<tr>
<td>Percentage of participants scoring above the cut-off (≥5)</td>
<td>30</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Body dissatisfaction score</td>
<td>5 (3–8)</td>
<td>5 (4–7.5)</td>
<td>0.549</td>
</tr>
<tr>
<td>Percentage of participants scoring above the cut-off (≥14)*</td>
<td>5</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Percentage of participants scoring above the cut-off (≥9)**</td>
<td>13</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Drive for thinness score</td>
<td>5.6 ± 3.2</td>
<td>5.4 ± 3.9</td>
<td>0.26</td>
</tr>
<tr>
<td>Percentage of participants scoring above the cut-off (≥15)*</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Percentage of participants scoring above the cut-off (≥7)**</td>
<td>46</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td><strong>Three-Factor Eating questionnaire</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cognitive dietary restraint score</td>
<td>10.6 ± 3.7</td>
<td>9.5 ± 3.7</td>
<td>0.30</td>
</tr>
<tr>
<td>Percentage of participants scoring above the cut-off (≥9)**</td>
<td>71</td>
<td>63</td>
<td></td>
</tr>
</tbody>
</table>

Notes: Parametric variables are reported as means and standard deviation and non-parametric variables as median and interquartile ranges (25th–75th percentile). Categorical data are reported as a percentage of the total group.

*Cut-off for clinical disordered eating.

Table V. Prevalence of any one or any two Triad components among athletes and non-athletes.

<table>
<thead>
<tr>
<th>Triad components</th>
<th>Athletes (n = 61)</th>
<th>Non-athletes (n = 49)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subclinical components</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EA &lt; 45 kcal · kg FFM⁻¹ · d⁻¹ with or without subclinical DE behaviour</td>
<td>56</td>
<td>69.6 (39)</td>
<td></td>
</tr>
<tr>
<td>Subclinical DE behaviour only</td>
<td>61</td>
<td>7.1 (4)</td>
<td></td>
</tr>
<tr>
<td><strong>Clinical components</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EA &lt; 30 kcal · kg FFM⁻¹ · d⁻¹ with or without clinical DE</td>
<td>56</td>
<td>17.9 (10)</td>
<td></td>
</tr>
<tr>
<td>Clinical DE only</td>
<td>56</td>
<td>3.6 (3)</td>
<td></td>
</tr>
<tr>
<td>EA &lt; 30 kcal · kg FFM⁻¹ · d⁻¹ and MD</td>
<td>56</td>
<td>8.9 (5)</td>
<td></td>
</tr>
<tr>
<td>Clinical DE and MD</td>
<td>61</td>
<td>3.3 (2)</td>
<td></td>
</tr>
</tbody>
</table>

Notes: *Significant difference between athletes and non-athletes.
*Odds ratio = 9.5, 95% CI = 1.17–77, P = 0.021.
**Odds ratio = 0.6, 95% CI = 0.5–6.9, P = 0.05.
EA, energy availability; DE, disordered eating; MD, menstrual dysfunction.
for elite Kenyan athletes to excel in order to break the cycle of poverty (Saltin et al., 1995). In our study, parents’ education, employment status and family size were used as indicators of socio-economic status. It seemed that athletes came from lower socio-economic households than non-athletes, and this may have impacted food availability due to financial constraints. Other possible contributing factors to low-energy intake include school meals that do not meet the athletes’ increased energy requirements; ignorance of athletes and parents regarding their energy needs; and/or deliberate undereating to attain a lean physique (Hulley & Hill, 2001). More than three-quarters of athletes with subclinical low-energy availability were identified with disordered eating in the current study. This finding supports those of Cobb and colleagues (2003) that a chronic energy deficiency among many exercising women is a result of disordered eating (Cobb et al., 2003). Additionally, a high percentage of our participants reported cognitive dietary restraint and had a high drive for thinness which has been associated with an energy deficiency in exercising women (De Souza, Hontscharuk, Olmsted, Kerr, & Williams, 2007; Gibbs, Williams, Mallinson, et al., 2013; Robbeson, Kruger, & Wright, 2015).

There has been an increase in the number of eating disorders and disordered eating behaviours among collegiate and high school students in Africa (Bennett et al., 2004; Fawzi et al., 2010). We found many students presented with subclinical disordered eating and a few with clinical disordered eating supporting this notion. Traditionally, the cultural norm of the ideal female body image is larger in African women (Puoane et al., 2005). This ideal body image seems to be changing and may be due to Western media influences, which portray thin images as attractive as opposed to the traditional “big is beautiful” (Puoane et al., 2005). Young female endurance runners are exposed to the lean body images of elite female athletes portrayed in the media and widely available in international television coverage of athletic events. These exposures may have influenced our athletes’ eating behaviour. Almost a third of athletes and non-athletes scored above the cut-off for the EDI-3 bulimia subscale, indicating binge-eating behaviour. Possible causes for the binge-eating behaviour are not clear. Hulley et al. (2007) reported a higher prevalence of binge eating in young adult non-athlete Kenyans (43.3%) compared to athletes (11.1%) and found that Kenyan women identified with Eating Disorder Not Otherwise Specified reported binge eating as a key behaviour. Pernick et al. (2006), on the other hand, found a lower prevalence of binge eating in African American and Caucasian adolescent athletes compared to Latinas. Whether age, cultural and/or ethnicity played a role in these observed differences is not clear. Research is needed to explore motivators and risk factors for disordered eating behaviour and eating disorders in adolescent Kenyan girls.

Consistent with other studies (O’Donnel & De Souza, 2004; Torstveit & Sundgot-Borgen, 2005), more athletes than non-athletes reported menstrual dysfunction, including primary and secondary amenorrhea. No participants reported oligomenorrhoea and both athletes and non-athletes had the same age of menarche in contrast to that reported for Caucasians athletes (Malina, 1994). Nichols, Rauh, and Lawson (2006) reported menstrual dysfunction in 23.5% of high school athletes. The most prevalent form of menstrual dysfunction was oligomenorrhoea (17.1%) followed by secondary amenorrhea and primary amenorrhea (5.3% and 1.2%, respectively). The difference in prevalence of menstrual dysfunction found between our study and that of Nichols et al. (2006) may be attributed to the differences in defining primary amenorrhea and oligomenorrhoea. In the current study, half of the athletes identified with clinical low-energy availability also reported menstrual dysfunction. Energy deficiency and/or a high drive for thinness are associated with a disruption in menstrual function (Gibbs, Williams, Scheid, Toombs, & De Souza, 2011; Nattiv et al., 2007). Often exercising women are not aware of subtle menstrual disturbances such as anovulation or luteal phase deficiency (De Souza et al., 2010). It is possible that athletes who reported regular cycles with clinical and subclinical low-energy availability might have presented with subtle (subclinical) menstrual disturbances. In fact, 45% athletes reported menstrual cycle changes during competition season. Due to limited resources, subclinical menstrual abnormalities were not measured and should be explored further in subsequent studies in this population group.

A wide range of clinical consequences associated with menstrual disturbances in exercising women, such as decreased BMD and increased risk of stress fractures, have been reported (Nattiv et al., 2007). Furthermore, disordered eating may result in low-energy availability and consequently menstrual dysfunction which impairs bone health by indirectly or directly suppressing hormones promoting bone formation (Nattiv et al., 2007). Due to the limitations associated with ultrasound, as described in the methodology, relationships between menstrual dysfunction, energy availability and BMD was not explored. As expected, BMD of athletes was approximately 6% higher compared to non-athletes probably as a result of force load sustained in the heel during running (Nichols, Bonnick, & Sanborn, 2000). Furthermore, the effect of an energy deficiency and/or menstrual dysfunction on BMD has
most likely not yet transpired in these adolescent participants. Noteworthy is that a small group of participants reported a history of stress fractures placing them at risk for low BMD.

To conclude, insufficient energy intake and disordered eating were identified as major concerns in this group of African adolescent students. Further understanding of the possible motivators and risk factors is required to identify suitable intervention programmes for prevention or treatment of eating disorders and relative energy deficiency in adolescent Kenyan girls. It is recommended that coaches, parents, athletes and carers are educated on athletes’ energy needs and boarding school menus are reviewed to reduce the risk of energy and nutrient deficiencies and its associated health and performance consequences.

Acknowledgements
We acknowledge Getz Pharmaceuticals for their support in conducting BMD measures. We thank the students for participating in this research study, their coaches and school principals for supporting our research endeavours. We would like to thank Alice Wambui, Meda and Loice Chepchumba for their help with data collection.

Disclosure statement
No potential conflict of interest was reported by the authors.

References


