BMJ Open Sport & Exercise Medicine

Exploring the relationship between low energy availability, depression and eating disorders in female athletes: a cross-sectional study

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To cite: Halioua R, Wasserfurth P, Toepffer D, *et al*. Exploring the relationship between low energy availability, depression and eating disorders in female athletes: a cross-sectional study. *BMJ Open Sport & Exercise Medicine* 2024;**10**:e002035. doi:10.1136/bmjsem-2024-002035

Accepted 18 July 2024

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ABSTRACT

Objective This cross-sectional study aimed to investigate the role of low energy availability (LEA) in the interplay between depression and disordered eating/eating disorders (DE/EDs) among female athletes. The International Olympic Committee consensus statement on Relative Energy Deficiency in Sport (REDs) identified depression as both an outcome of LEA and a secondary risk factor for REDs. However, the direct link between LEA and depression has yet to be fully established.

Methods We assessed 57 female athletes participating in weight-sensitive sports at different levels of competition training at least four times a week. Assessment was conducted using laboratory analyses, clinical interviews and the Patient Health Questionnaire-9 questionnaire. Participants were recruited through various channels, including German sports clubs, Olympic training centres, social media platforms and the distribution of flyers at competitions. Indicators of LEA were defined if at least two of the following three physiological indicators were present: menstrual disturbances, suppressed resting metabolic rate and suppressed thyroid hormones. Logistic and linear regression analysis were used to examine the relationship between LEA, depression and DE/ED.

Results The lifetime prevalence of depressive disorders was 29.6%. 19% of the participants were diagnosed with an ED, and an additional 22.6% exhibited DE.
LEA was not significantly associated with either lifetime prevalence of depressive disorders or current depressive symptoms. However, a significant association was found between depression and DE/ED in terms of both lifetime prevalence and current depressive symptoms. DE/ED increased the probability of lifetime prevalence of depressive disorders by 34% (19%–49%) compared with normal eating behaviour.

Conclusion We found no evidence that LEA is an independent factor for depression in female athletes. Its association with LEA and REDs appears to occur primarily in the presence of DE/ED.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Depression is recognised as both a mental health outcome and a secondary risk factor of Relative Energy Deficiency in Sport (REDs). However, the relationship between low energy availability (LEA) and depression has not been fully established, as existing research on depression has neither been based on clinical data nor accounted for potential comorbid eating disorders (EDs).

WHAT THIS STUDY ADDS

⇒ We found no evidence that LEA is associated with the lifetime prevalence of depressive disorders or current depressive symptoms in participants with or without disordered eating/EDs (DE/EDs).

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ The association between LEA and depression appears to occur primarily in the presence of DE/ED. Therefore, further investigation is needed before conclusively classifying depression as a mental health outcome and a secondary risk factor of REDs.

INTRODUCTION

Low energy availability (LEA) refers to a state where energy intake is inadequate to sustain optimal functioning of all physiological systems after accounting for exercise-related energy expenditure.[1] Problematic LEA represents a maladaptive response to severe and/or prolonged LEA that disrupts various body systems[1] and is understood as the underlying cause of Relative Energy Deficit in Sports (REDs), a syndrome of impaired physiological and/or psychological functioning experienced by female and male athletes.[2–4]

The impact of problematic LEA on mental health and the influence of psychological factors are not as well understood as the physiological consequences. Eating disorders (EDs) and disordered eating (DE) behaviour are considered significant risk factors for LEA and REDs, as they can be both a cause and consequence and, thus, are considered a primary indicator of REDs.[4 5] The International Olympic Committee (IOC) consensus statement also identifies depressive symptoms

and affective disorders as adverse mental health outcomes associated with problematic LEA and REDs, considering clinically diagnosed depression a secondary indicator of REDs.[4] A narrative review by a subgroup of the IOC consensus on REDs discussed the temporal relationship between exposure to LEA and mental health outcomes.[5] It was suggested that mood changes, fatigue and psychological conflict may be expected within days and weeks, whereas the development of depressive symptoms may require longer exposure to LEA.[5]

However, the current literature on the association between LEA and depressive symptoms and/or clinically diagnosed depression is limited. To our knowledge, only three studies have investigated the relationship between LEA and depressive symptoms.[6–8] Although these studies provide valuable insights, their findings may be subject to interpretation due to methodological limitations. Ackerman *et al* (2019) reported a positive association between LEA and depressive symptoms in a large-scale (n=1000) cross-sectional study. However, LEA assessment relied on DE/ED screening questionnaires, which may have primarily captured the association between DE/ED and depressive symptoms. Furthermore, diagnoses of ED and depression were based solely on questionnaires without clinical confirmation from diagnostic interviews. In the study by Rogers *et al* (2021), LEA was established through objective laboratory analyses and defined as a low resting metabolic rate (RMR). And although clinical interviews were conducted, findings from these were not included in the final analysis, which instead solely relied on questionnaire responses that considered depressive symptoms only. Mathisen *et al* (2019) conducted a longitudinal study and found an increase in depressive symptoms in female fitness athletes preparing for a competition.[8] However, the relationship between LEA and depressive symptoms was not directly evaluated, and the assessment of depressive symptoms relied solely on questionnaires. Furthermore, the increased depressive symptoms remained well below clinical significance. Neither study accounted for potential comorbid DE/ED, which could have influenced the observed relationship.

Investigating the association between LEA and depression requires consideration of comorbid DE/ED, which are both a possible cause of LEA and share a well-established comorbidity with depression in non-athletic populations.[9] Preliminary findings suggest this comorbidity may also exist in the athletic context.[10] In response to the IOC's call for more studies to explore the reciprocal function of psychological variables with LEA,[2 5] we aimed to examine the role of LEA in the interplay between DE/ED and depression more closely. As the study was exploratory in nature, no specific hypotheses were formulated. This allowed for an open investigation into the association between LEA and depression in athletes accounting for DE/ED, as well as an exploration of whether LEA increases the risk of depression in those with ED/DE.

METHODS

Study design, setting and participants

This cross-sectional study examined physiological indicators of problematic LEA, depression and DE/ED in female athletes using state-of-the-art assessments: laboratory analyses, clinical interviews and questionnaires. Laboratory analyses were performed at our core facility at the Technical University of Munich, and clinical interviews were conducted via telemedicine by a trained psychiatrist. Participants were recruited through various channels, including German sports clubs, Olympic training centres, social media platforms and the distribution of flyers at competitions. The study was approved by the Technical University Munich's Ethical Review Board (registration number: 347/21 S) and adhered to the Declaration of Helsinki. All participants provided written informed consent. Recruitment and data collection lasted from November 2021 until April 2023.

Eligibility criteria

The eligibility criteria required participants to be female athletes aged 18–39 years engaged in weight-sensitive sports (cycling, triathlon, long-distance running, swimming, cross-country skiing, biathlon and ballet) who trained ≥4 times weekly. Exclusion criteria encompassed the use of hormonal contraceptives, pregnancy, breastfeeding or chronic illness or acute injury at the time of potential inclusion.

Clinical assessment of depression and eating disorders

The clinical assessment comprised clinical interviews conducted by a board-certified psychiatrist via a secure online video platform, along with the completion of questionnaires. All interviews were conducted following the laboratory analysis and lasted approximately 45 min.

Depression

The clinical interview was conducted using Module A of the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), Clinician Version (SCID-5-CV). The SCID-5-CV is a semistructured interview for diagnosing mental disorders according to DSM-5. Each DSM-5 criterion is associated with specific interview questions to assist the interviewer in assessing the criterion. Both current and past depressive symptoms were assessed. This allowed for an evaluation of both current depressive symptomatology and the lifetime prevalence of depressive disorder.

The Patient Health Questionnaire-9 (PHQ-9) questionnaire was administered in addition to the clinical interview. The PHQ-9 corresponds to the Depression Module of PHQ (PHQ-D) and consists of nine questions related to depressive symptoms. It was developed as a screening instrument for diagnosing depression for routine use in somatic medical settings. To minimise potential bias, the interviewer was kept blinded to the participants' questionnaire outcomes throughout the data collection process.

Eating disorders and disordered eating

The clinical interview was conducted using the German version of the Eating Disorder Examination by Fairburn and Cooper, a structured interview for assessing the specific psychopathology of ED.[11 12] It assesses eating behaviour across four scales: 'Restraint', 'Eating Concern', 'Weight Concern' and 'Shape Concern'. In addition to these scales, 14 additional diagnostic items enable clinical diagnosis of ED.

DE was only identified in cases where clear pathological behaviours and/or distorted body image were present, without meeting the diagnostic criteria for an ED. The assessment took into account the context of sports and the resulting demands on eating behaviour.[10] The study set a high threshold for DE, placing it closer to ED than to normal, functional eating behaviour.

Assessment of LEA

Classification of menstrual status

Participant menstrual status was determined retrospectively via phone interviews, with information collected on menstruation within the last 3 months. Based on their cycle lengths, participants were classified as eumenorrheic (28±7 days), oligomenorrheic (cycle length >35 days), primary amenorrheic (no menarche after 15 years of age) or secondary amenorrheic (absence of ≥3 consecutive periods or no menses for ≥90 days).[13]

Laboratory examinations

For participants with regular or irregular periods, laboratory examinations were scheduled during the early follicular phase (days 3–5 after period onset), while for those with amenorrhoea, the examination was conducted at the earliest opportunity. Prior to measurement, participants refrained from exercise, caffeine and alcohol for 24 hours and arrived at the laboratory after a 12-hour overnight fast in a rested state.

Anthropometry and body composition

Participants wore only underwear or tightly fitting swimwear for measurements. Height was measured to the nearest 0.1 cm using a stadiometer (seca 217, SECA, Hamburg, Germany). Body composition was assessed using air displacement plethysmography (ADP; BOD POD, COSMED, Rome, Italy), with body mass being measured using a built-in scale. The device was calibrated before each measurement session. Participants wore a swim cap and remained still during the measurement. Fat mass and fat-free mass were determined using the Siri equation based on ADP-measured body density.[14]

Resting metabolic rate

RMR was assessed using open-circuit indirect calorimetry with a canopy (Q-NRG, COSMED, Rome, Italy) at room temperature (20–25 °C).[15] The device was calibrated with external gas and room air prior to each measurement, and ethanol burning tests were conducted monthly. Participants rested supine for ~10 min before data collection, which lasted at least 30 min. RMR (kcal/day) was calculated using the Weir equation from steady-state data, which was defined as a coefficient of variation ≤10% in steady-state oxygen consumption and carbon dioxide production.[16]

Blood samples

Blood samples were drawn from the antecubital vein and free triiodothyronine (fT3) was analysed by an external laboratory (Labor Becker MVZ GbR, Munich, Germany).

Classification of LEA

Following the 2023 IOC REDs consensus statement,[4] cases of LEA were operationally defined if at least two of the following three physiological indicators were present: menstrual disturbances (amenorrhoea and oligomenorrhoea), suppressed RMR (<30 kcal/kg/fat-free mass (FFM)) and suppressed thyroid hormones (fT3 below the IOC-recommended laboratory reference ranges: within or below the lowest quartile). Suppressed RMR was selected as an indicator due to its association with LEA,[17] without being directly related to DE/ED.

Statistical analysis

Sample size

Based on the existing literature on LEA prevalence,[7] a sample size of 50 was determined, allowing for approximately 30% of LEA cases. To account for a 15%–20% dropout rate between measurements, we aimed for a total of 60 participants.

Descriptive analysis

We calculated frequencies, means and 95% CIs for demographic data and assessment instruments.

Main analysis

To examine the role of LEA in the interplay between depression and DE/ED, logistic regression and linear regression analyses were conducted. The lifetime prevalence of diagnosed depressive disorder was chosen as the outcome for logistic regression, while the outcome for linear regression was current depressive symptoms according to PHQ-9 scores. In both analyses, LEA was used as a predictor variable, with DE/ED serving as a control variable. Given the high threshold applied for DE in this study, DE and ED were examined as a combined variable. In both regression analyses, DE and ED were also inserted as separately coded variables, aiming to explore the potential differences in outcomes compared with analyses combining DE/ED. Interactions between LEA and DE/ED were examined by the likelihood ratio test.

The OR and Average Marginal Effects (AME) were calculated to interpret the effect of LEA and DE/ED. In this case, AMEs reflect the average change in predicted probability for each observation in factorial variables while holding the other variable constant.[18] Predicted probabilities for lifetime depressive disorder and predicted margins for depressive symptoms were also calculated.

Missing data

We conducted Multiple Imputation by Chained Equations to analyse 10 multiply-imputed datasets, addressing incomplete variables using the *mi impute* command in Stata. Rubin's combination rules were applied to adjust estimates and their standard errors for imputation uncertainty. Additionally, we performed a complete case analysis for comparison.

Statistical software

Stata Statistical Software: Release V.16.1. College Station, Texas was used to analyse the data.

Patient and public involvement

As our study focuses on female athletes in weight-sensitive sports, no patients were involved. Participants were first engaged in the study during the recruitment phase. Some participants subsequently recruited additional participants from their own circles. Results of laboratory measurements and clinical interviews were shared with participants. Assistance was provided to one participant in arranging therapy for her ED. Results and new insights will be provided to the participants.

Equity, diversity and inclusion statement

Our study is on female athletes in weight-sensitive sports. The author group consists of junior, mid-career and senior researchers from different disciplines in Switzerland and Germany. Participants were recruited through various channels to ensure a broad spectrum of socioeconomic backgrounds. Nevertheless, our patient cohort predominantly represents the middle to upper socioeconomic strata. Clinical interviews were conducted via telemedicine to enhance accessibility.

RESULTS

Participants

A total of 93 athletes provided written informed consent to participate in the study, with 57 completing on-site measurements. Demographics, laboratory assessments and clinical diagnoses of depressive disorder and ED are presented in table 1.

Missing data

The assessment and clinical diagnosis of depression and DE/ED were completed for 53 (92.9%) participants. Four participants did not participate in the clinical interviews, one of whom did not provide a blood sample. Three participants reported taking thyroid medication, which was treated as missing values for low fT3 and RMR <30 kcal/kg/FFM. Non-participation in clinical interviews was treated as missing values for lifetime depressive disorder and DE/ED. The missing blood analysis was treated as a missing value for low fT3. There were a total of five missing values for LEA/Controls, four for lifetime depressive disorders and four for DE/ED. Multiple imputation and complete case analysis lead to similar results; therefore, we present only the former.

Main results

Logistic and linear regressions

Table 2 summarises the results of the logistic regression analysis on lifetime prevalence of depressive disorders and the linear regression analysis on current depressive symptoms according to the PHQ-9. LEA was not statistically associated with either lifetime prevalence of depressive disorders or current depressive symptoms. DE/ED showed statistical significance in both regression analyses. There was no significant interaction between LEA and DE/ED.

DE/ED increased the probability of lifetime prevalence of depressive disorders by 34% (19%–49%) compared with normal eating behaviour. The predicted probabilities for female athletes with DE/ED and concurrent LEA were 57.9% (25.3%–90.5%) and 51.5% (27.7%–75.3%) for those without concurrent LEA. The difference of 6.4% was statistically not significant (p=0.73). Those with a DE/ED and concurrent LEA yielded a predicted PHQ-9 score of 7.5 (5.5–9.4), and those without concurrent LEA yielded a score of 7.7 (6.2–9.2). The difference was statistically not significant (p=0.71). Figure 1 presents the predicted probabilities for lifetime prevalence of depressive disorders, while figure 2 displays the predicted scores of the PHQ-9.

DISCUSSION

The aim of this study was to examine the role of LEA in the interplay between depression and DE/ED in an exploratory investigation. Our findings indicate that the previously established association between LEA and depression may be mediated by comorbid DE/ED. We found no evidence that indicators of LEA are associated with the lifetime prevalence of depressive disorders or current depressive symptoms in participants regardless of DE/ED status. However, a strong association was found between DE/ED and depression, in terms of both lifetime prevalence and current symptoms.

Energy availability

LEA alone did not increase the prevalence of lifetime depressive disorder or current depressive symptoms in those with or without DE/ED. Our results indicate that DE/ED as an underlying cause of LEA has a more significant impact on depression than LEA itself. LEA may result from intentional or unintentional undereating.[19] In the case of intentional undereating, it is important to distinguish DE/ED from controlled eating for performance enhancement and other functional reasons. Although both can result in LEA, they are driven by different motivational factors and seem to play a decisive role in whether LEA is associated with depression or not. Given that DE/EDs typically have a long course and that LEA was not associated with depression in those with DE/EDs, it seems unlikely that there is a temporal relationship between prolonged exposure to LEAs and the development of depressive symptoms. Taken together, depression as a direct mental health outcome resulting

Table 1 Descriptive characteristics of the study sample

Variables	Controls (39)	LEA (14)	Total (57)
Age (years), mean±SD	28.4±5.7	27.3±4.2	27.9±5.3
Height (cm), mean±SD	168.7±5.8	169.5±5.5	168.8±5.5
Weight (kg), mean±SD	62.0±6.5	55.8±5.3	60.5±7.1
BMI (kg/m²), mean±SD	21.7±1.7	19.4±1.3	21.2±2.1
Education (%)			
Academic	61.5	100	70.2
High school	25.6	0	21.1
Non-academic	2.6	0	1.8
Secondary school	10.3	0	7
Training level (%)			
National team	7.7	0	5.3
Professional	5.1	14.3	7
Club	41.0	21.4	36.8
Individual with competition	38.5	57.1	43.9
Individual without competition	7.7	7.1	7
Menstrual status (%)			
Amenorrhoea	7.7	64.3	21.1
Oligomenorrhoea	20.5	21.4	21.1
Eumenorrhoea	71.8	14.3	57.9
RMR (kcal/kg FFM), mean±SD	30.9±2.6	27.4±1.7	30.0±2.8
fT3 (ng/L), mean±SD	2.7±0.3	2.2±0.5	2.6±0.4
Depressive disorder (%)			
No depressive disorder	71.1	61.5	68.5
Mild depressive episode	m	m	1.9
History of one depressive episode in the past	10.5	15.4	11.1
Recurrent depressive disorder, current episode in remission	15.8	15.4	14.8
Recurrent depressive disorder, current episode mild	2.6	7.7	3.7
Lifetime prevalence	28.9	38.5	29.6
PHQ-9 (0–28), mean±SD	5.8±3.6	5.8±3.6	5.8±3.5
Eating disorder (%)			
Normal eating behaviour	60.5	46.2	58.5
Disordered eating	23.7	23.1	22.6
Bulimia nervosa	2.6	7.7	3.8
Anorexia nervosa	0.0	7.7	1.9
Bulimia nervosa of low frequency/limited duration	7.9	0.0	5.7
Atypical anorexia nervosa	2.6	7.7	3.8
Purging disorder	2.6	7.7	3.8
Eating disorder or disordered eating (%)	39.5	53.8	41.5

Unless otherwise specified, mean and SD are reported for continuous variables, and proportions are reported for categorical data.
*Depression Module of the Patient Health Questionnaire.
BMI, body mass index; fT3, free triiodothyronine; LEA, Low energy availability; m, missing; PHQ-9, Patient Health Questionnaire-9; RMR, resting metabolic rate.

from a physiological causal chain involving exposure to LEA seems unlikely.

This contradicts the findings of previous studies by Ackermann[6] and Rogers,[7] who found a positive association between LEA and depression. The difference in results with Ackermann's study can be attributed to their use of the DE/ED screens as a marker for LEA, essentially confirming the association between LEA and DE/ED observed in our study. Our study's more precise operationalisation of LEA, which incorporates several

Table 2 Results from the logistic and linear regressions

Logistic regression on lifetime prevalence of depressive disorders				
Variable	**OR**	**(95% CI)**	**SE**	**P value**
LEA*	1.3	(0.29 to 5.75)	0.99	0.73
DE/ED†	7.59	(1.93 to 29.82)	5.29	<0.01
Constant	0.14	(0.05 to 0.43)	0.08	<0.01
Linear regression on current depressive symptoms according to the PHQ-9				
Variable	**β**	**(95% CI)**	**SE**	**P value**
LEA*	−0.23	(−2.33 to 1.87)	1.04	0.83
DE/ED†	3.09	(1.33 to 4.85)	0.88	<0.01
Intercept	4.62	(3.42 to 5.82)	0.6	<0.001

*Low energy availability.
†Disordered Eating / Eating disorder.
DE/ED, disordered eating/eating disorder; LEA, low energy availability; PHQ-9, Patient Health Questionnaire-9.

physiological indicators such as low RMR, low T3, and menstrual dysfunction, may explain the difference from Rogers' findings, which identified LEA only using low RMR. Furthermore, Rogers' study did not consider potential comorbid DE/ED in their analysis. Our results are in line with those in the non-athletic context, which found no correlation between nutritional status and depressive symptoms in individuals with anorexia nervosa.[20 21]

Depression

When compared with the general population, participants in the present study showed higher levels of depression, both in terms of lifetime prevalence and current symptoms. The overall lifetime prevalence was 29.6%, surpassing the 10%–15% range observed in the general population.[22 23] Additionally, the average PHQ-9 score was elevated at 5.8, exceeding the normative value of 3.6[24] and aligning with scores observed in representative patient samples from general hospitals in Germany.[25] The findings are consistent with Schaal *et al* (2001),[26] who found a lifetime prevalence of 30% in women who participate in aesthetic sports. Only 5.6% of the participants in our sample exhibited clinically diagnosed depression during the clinical interviews. Therefore, the score from the PHQ-9 should be interpreted as mainly subclinical. However, in light of the higher lifetime prevalence, it may have clinical significance as it may indicate a higher overall burden of depressive symptoms.

DE/ED

The prevalence of ED (19%) and DE (22.6%) in our sample was higher than that of the general population,[27] which is consistent with the findings of similar studies.[28 29] The majority (13.3%) classified as Other Specified Feeding and Eating Disorder. Participants with LEA primarily displayed atypical anorexia nervosa and purging disorder, whereas those with adequate EA showed bulimia of low frequency/limited duration. Notably, individuals with LEA were twice as likely to have an ED (30.8%) when compared with those with

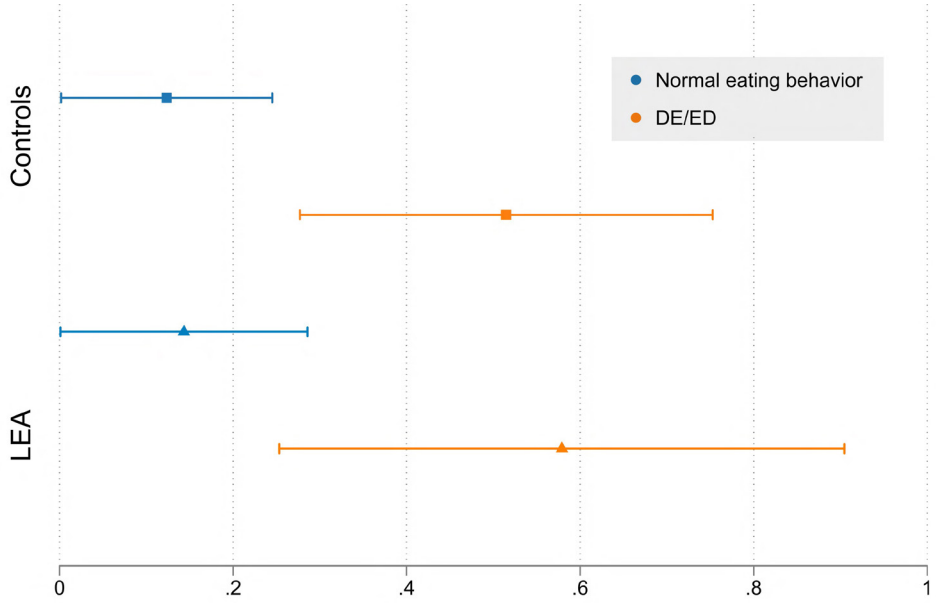

Figure 1 Predicted probabilities for lifetime prevalence of depressive disorders. For those without LEA (controls), the predicted probabilities for lifetime prevalence of depressive disorders were 12.4% (0.2%–24.5%) with normal eating behaviour and 51.5% (27.7%–75.3%) with DE/ED. For those with LEA and normal eating behaviour, the probability was 14.3% (0%–29%) compared with 57.9% (25.3%–90.5%) with concurrent DE/ED. Whiskers represent the 95% CI. DE/ED, disordered eating/eating disorder; LEA, low energy availability.

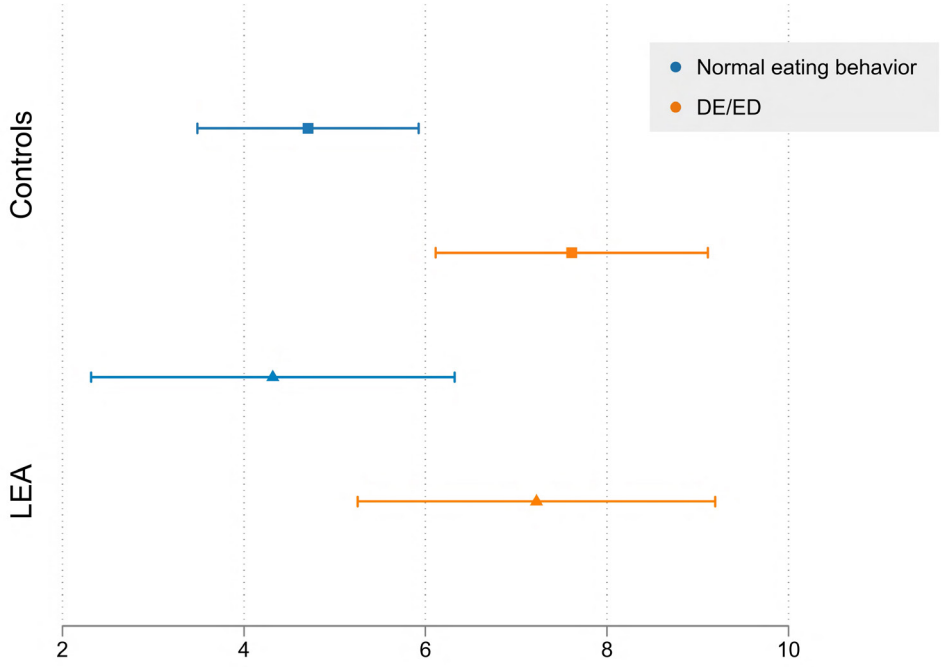

Figure 2 Predicted scores of the PHQ-9. For those without LEA (controls), the predicted PHQ-9 score was 4.6 (3.4–5.8) with normal eating behaviour and 7.7 (6.2–9.2) with DE/ED. For those with LEA and normal eating behaviour, the predicted score was 4.4 (2.4–6.4) compared with 7.5 (5.5–9.4) with concurrent DE/ED. Whiskers represent 95% CI. DE/ED, disordered eating/eating disorder; LEA, low energy availability; PHQ-9, Patient Health Questionnaire-9.

adequate EA (15.7%); however, DE occurred at the same frequency (23 %) in both groups. This underscores the necessity to consider LEA and DE/ED as distinct entities. While DE/ED is a prevalent underlying cause of LEA, a notable proportion (46.2%) of those with LEA did not exhibit an associated DE/ED. Furthermore, 40% of individuals without LEA still exhibited a DE/ED.

Interplay between depression and DE/ED

A strong association was found between DE/ED and depression, in terms of both lifetime prevalence and current depressive symptoms. The presence of DE/ED increased the likelihood of experiencing depression at least once in one's lifetime by 35% and current depressive symptoms by three points on the PHQ-9 scale. Participants with DE/ED had a lifetime prevalence of depressive disorder ranging from 51.5% (without concurrent LEA) to 57.9% (with concurrent LEA). This is in line with findings in the general and athletic population, which indicate a high rate of comorbidity between depression and ED, suggesting a bidirectional relationship.[10 27 30] Our findings indicate that the previously established association between LEA and depression may be mediated by comorbid DE/ED. Additionally, this comorbidity may explain the higher prevalence of depression in weight-sensitive samples compared with other sports.[26 31]

Clinical implications and future research

REDs is conceptualised as an etiological syndrome based on problematic LEA. Within this framework, depression is both considered a mental health outcome and a secondary risk factor of REDs.[4] From a theoretical perspective, there is insufficient evidence to conclude that LEA is in fact a causative factor for depression. Our data demonstrate that the association between LEA and depression appears to occur primarily in the presence of DE/ED. Therefore, further investigation is needed before conclusively classifying depression as a mental health outcome and a secondary risk factor of REDs.

Limitations

First, while ensuring that participants represented the primary target audience of REDs, our inclusion criteria of a minimum of four training sessions per week may have excluded participants with severe depressive symptoms, as they may not have been able to maintain such a sports regimen at this frequency. Second, despite our efforts to represent a broad socioeconomic spectrum, the generalisability is limited by the fact that the participants are predominantly from middle to upper socioeconomic strata with educational backgrounds. Third, the use of state-of-the-art methodology involving lab measurements and clinical interviews resulted in a relatively small sample size. As such, caution is necessary when interpreting effect sizes as they may be susceptible to inflation. Fourth, by using cross-sectional data, we are precluded from drawing causal conclusions from the associations we found.

Acknowledgements We would like to thank Mona Saller, Lena Fladerer, Josephine Ruf and Vanessa Corrêa Mascarenhas for their assistance. We would further like to thank all volunteers who participated in the study.

Contributors Conceptualisation: KK and RH. Methodology: KK, RH and PW. Investigation: RH, PW and DT. Formal analysis: RH. Data curation: RH, PW and DT. Writing—original draft preparation: RH. Writing—review and editing: PW, KK, MCC and DT. Funding acquisition: KK, RH and MC. Resources: MCC and KK. Supervision: KK and MCC. RH is the guarantor. Since English is not the first author's first language, AI was used to make linguistic corrections. The use of AI was strictly confined to correcting language errors. No content interpretations or statistical analyses were conducted using AI.

Funding This study was funded by the Robert-Enke-Foundation. PW is supported by a fellowship by the Bavarian Equal Opportunity Scholarship Program.

Disclaimer AI use statement: since English is not the first author's first language, AI was used to make linguistic corrections. The use of AI was strictly confined to correcting language errors. No content interpretations or statistical analyses were conducted using AI.

Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, conduct, reporting or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Consent obtained directly from patient(s).

Ethics approval This study involves human participants and was approved by the Ethical Review Board of the Technical University Munich (registration number: 347/21 S). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. The datasets used and/or analysed during the current study are available from the first author on reasonable request.

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