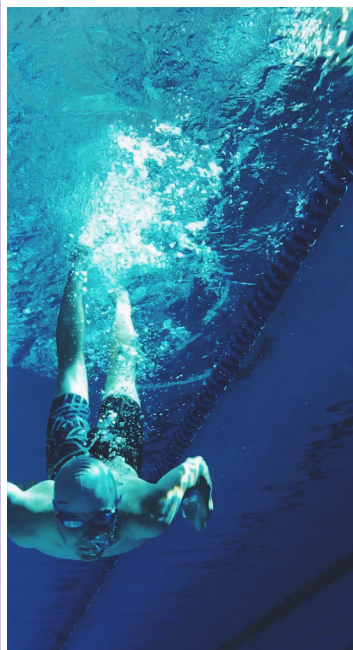




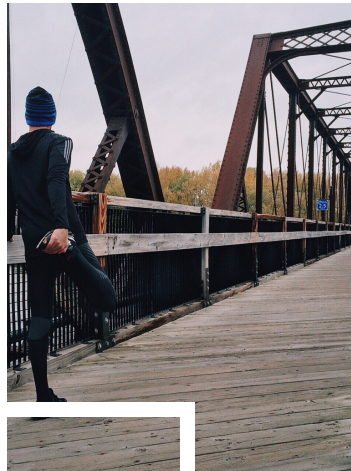
**Safe
Exercise**



guideline



**at Every
Stage**



Safe Exercise at Every Stage (SEES)

A Guideline for Managing Exercise in Eating Disorder Treatment

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Acronyms

ACSM: American College of Sports Medicine	FSH: Follicle stimulating hormone
AGREE-II: Appraisal of Guidelines for Research and Evaluation	FX: Flexibility training
AHA: American Heart Association	GH: Growth hormone
AN: Anorexia nervosa	GnRH: Gonadotropin-releasing hormone
AN-P: Anorexia nervosa - Purging subtype	HR: Heart rate
AN-R: Anorexia nervosa - Restrictive subtype	HRQOL: Health Related Quality of Life
ANS: Autonomic nervous system	IBW: Ideal body weight
BED: Binge eating disorder	IGF-1: Insulin-like growth factor 1
BF: Body fat	ISCD: International Society for Clinical Densitometry
BMD: Bone mineral density	Kcal/kg: Kilocalories per kilogram
BMI: Body mass index	LEA: Low energy availability
BN: Bulimia nervosa	LEAP: Loughborough Eating disorders Activity Therapy
BPM: Beats per minute	LH: Luteinizing hormone
BUN: Blood urea nitrogen	IBW: Ideal body weight
CBT: Cognitive behavioural therapy	MET: Metabolic equivalent
CBT-A: Cognitive behavioural therapy for Anorexia nervosa	mL: Millilitre
CBT-B: Cognitive behavioural therapy for Bulimia nervosa	mg/dL: Milligrams per decilitre
CBT-E: Enhanced cognitive behavioural therapy	mmHg: Millimetres of mercury
CET: Compulsive Exercise Test	mmol/L: Millimoles per litre
CRT: Cognitive remediation therapy	mEq/L: Milliequivalents of solute per litre
CVD: Cardiovascular disease	MANTRA: Maudsley Model of Anorexia Nervosa Treatment for Adults
DBT: Dialectical behavior therapy	n: Number of participants in the sample
DSM-IV: Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition)	NEDA: National Eating Disorder Association
DSM-5: Diagnostic and Statistical Manual of Mental Disorders (Fifth Edition)	NHRMC: National Health Research Medical Council
EA: Energy adequacy	OH: Orthostatic hypotension
ECG: Electrocardiogram	OSFED: Other Specified Feeding and Eating Disorders
ED(s): Eating disorder(s)	PT: Postural tachycardia
EDNOS: Eating Disorder Not Otherwise Specified	RANZCP: Royal Australian and New Zealand College of Psychiatrists
FBT: Family based therapy	RCT: Randomized controlled trial
FBT-AN: Family based therapy for Anorexia nervosa	RED-S: Relative Energy Deficiency in Sport
FBT-BN: Family based therapy for Bulimia nervosa	RMR: Resting metabolic rate
FFM: Fat-free mass	RT: Resistance training
FHA: Functional hypothalamic amenorrhea	SEES: Safe Exercise at Every Stage guideline
	SMA: Superior mesenteric artery syndrome
	USG: Urine specific gravity

Background

Eating disorders (EDs) are a complex set of psychiatric diagnoses comprising anorexia nervosa (AN), bulimia nervosa (BN), other specified feeding or eating disorder (OSFED) (previously recognised as eating disorder not otherwise specified), and binge eating disorder (BED). ED diagnoses are associated with a high economic burden, poorer quality of life, and increased likelihood of mortality from both medical complications and suicide (Bulik, Reba, Siega-Riz & Reichborn-Kjennerud, 2005; Crow, 2014). AN, in particular, has been recognised as having the highest mortality rate of any psychiatric diagnosis (Reel, 2013). Treatment of these chronic and severe disorders typically incorporates psychotherapy, often in combination with pharmacological treatment and nutrition counselling (Geller, Goodrich, Chan, Cockell, & Srikameswaran, 2012; Grilo & Mitchell, 2011). The full recovery rate of individuals treated with these protocols, however, is estimated to be no greater than 50%, supporting the need for additional adjunct treatment methods (Reel, 2013).

These sequelae are particularly concerning in the context of the rising incidence of these illnesses over the past decade, as well as increased reports of their prevalence in younger and more diverse populations (Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2005). Furthermore, individuals with an ED are less likely to seek treatment; males, in particular, exhibit low help-seeking behaviours and are more likely to develop medical complications earlier in their disorder than their female counterparts (Harvey & Robinson, 2003). One factor impacting the rate of relapse, chronicity and general prognosis in ED populations is engagement in dysfunctional exercise (DE) behaviours (also referred to as compulsive exercise, exercise dependence, exercise addiction or excessive exercise; Carter, Blackmore, Sutandar-Pinnock, & Woodside, 2004). DE is reported to be present in 20-81% of individuals with an ED (Dalle Grave, Calugi & Marchesini, 2008; Moola, Gairdner, & Amara, 2013; Schroff et al., 2006). Continued engagement in DE has been postulated as a predictor of relapse and illness chronicity (Carter et al., 2004; Strober, Freeman, & Morrell, 1997).

Despite the impact of exercise on treatment outcomes, many treatment protocols do not formally address exercise during the course of treatment. Currently, there is no standard practice to support health professionals manage and reintegrate exercise into ED treatment. Consequently, many health professionals have adopted the practice of recommending abstinence

from exercise during ED treatment (Davies, 2015; Hechler, Beumont, Marks, & Touyz, 2005; Thien, Thomas, Markin, & Biringham, 2000; Zunker, Mitchell, & Wonderlich, 2011). Over recent decades, both research and clinical health professionals working in the eating disorder field have questioned the practice of recommending that individuals with EDs abstain from exercise (Beumont, Arthur, Russell, & Touyz, 1994; Ng, Ng, & Wong, 2013; Quesnel et al., 2017). Concerns about DE management have continued to escalate given that research has proposed that nutritionally supported, supervised, and prescribed exercise is beneficial to both the physical and mental health of individuals with an ED (Chantler, Szabo, & Green, 2006; Duesund & Skarderud, 2003; Fernandez-del-Valle et al., 2010; Fernandez-del-Valle et al., 2014; Hausenblas, Cook, & Chittester, 2008; Ng et al., 2013; Moola et al., 2013; Vancampfort et al., 2014). Consequently, the Safe Exercise at Every Stage (SEES) guideline has been developed to better facilitate the prescription of exercise in eating disorder populations. This simple and graded process aims to support clinicians in determining the level of exercise and education appropriate for each individual based upon their current level of physical and psychological well-being. The guideline was created based upon results from systematic reviews of the literature, focus groups with professionals in the field, and feedback from relevant stakeholders worldwide (including clinicians, researchers and individuals with lived experience). It is our hope to disseminate the guidelines as an international standard (for further details see <https://www.safeexerciseateverystage.com/access-sees-guideline/>).

Statement of Intent

The Safe Exercise at Every Stage (SEES) guideline aims to provide evidence-based information to support clinicians in the management of dysfunctional exercise during eating disorder treatment. This guideline serves as a general tool for use under a practitioner's expert judgement, not as a standard for medical care. Standards of Medical Care are based on the clinical data of the individual as well as the scientific knowledge available at the time (Yager et al., 2005). The inclusion of the SEES guideline as a component within an evidence-based treatment approach does not ensure improved treatment outcomes, nor should it be interpreted as including all methods of integrating exercise into the treatment of ED. Exercise may not be healthful or safe for all individuals with eating disorders. As such, the SEES guideline is intended to be administered by a trained medical or exercise professional (see glossary, pp.63) in conjunction with a multidisciplinary team (see glossary, pp.63). The development of the SEES guideline was undertaken in line with both the Appraisal of Guidelines for Research and Evaluation guidelines (AGREE II; AGREE Next Steps Consortium, 2009) and recommendations from the National Health and Medical Research Council Guidelines (National Health and Medical Research Council (NHMRC) 2011). In addition to the contribution by our lead investigators, our final guideline has been developed based on a consensus by our steering committee (comprising an international panel of experts in the ED field; see pp.2) and consultation committee (comprising clinicians, researchers and those with lived experience with a thorough understanding of EDs; see pp.3). The information contained within this document reflects the knowledge and evidence at the time of its publication. Efforts will continue to evolve and amend the SEES guideline as new information and stakeholder feedback emerges.

Executive Summary

Dysfunctional exercise is commonly the first presenting and last remaining symptom of EDs (Davis, Kennedy, Ravelski, & Dionne, 1994). As such, it is imperative that exercise engagement is addressed throughout all stages of ED treatment and recovery. Addressing exercise engagement throughout treatment requires a step-up/step-down approach, tailoring the recommendations for exercise engagement according to the individual's current physical and psychological well-being (Cook & Leininger, 2017). The SEES guideline provides a framework for thoroughly assessing each individual's current physical and psychological health status. Furthermore, the SEES guideline augments clinical decision-making in utilising these results to select safe exercise interventions. The incorporation of the SEES guideline as a component of evidence-based treatment for individuals with ED is anticipated to promote an ethical standard level of care and greater consistency in the management of dysfunctional exercise.

Key Principles

The primary authors identified five key principles as underpinning their intentions in developing the SEES guideline. These principles have guided the development of this guideline and should be employed when applying its recommendations. The key principles include: non-abstinence, safety, holistic, intuitive and mindful movement, and collaboration.

1. Non-abstinence

This principle was central to our motivation to develop the SEES guideline. The clinical practice of recommending exercise abstinence for individuals with ED is common and understandable considering the high rates of complex medical complications (Davies, 2015; Hechler et al., 2005; Thien et al., 2000; Zunker et al., 2011). The safety of this recommendation is further supported by an absence of standard practice in managing dysfunctional exercise in ED treatment. However, we equate recommending exercise abstinence with simply encouraging an individual with AN to 'just eat,' without further intervention to guide the process of renourishing or supporting their return to a healthy relationship with food.

Exercise abstinence has been associated with an increased risk of relapse (Carter et al., 2004), poorer treatment outcomes (Bratland-Sanda & Vrabel, 2018), more severe psychopathologies, and worsened illness chronicity (Davis et al., 1994; Dalle Grave et al., 2008). Furthermore, promoting complete exercise abstinence before providing new and healthy ways to cope may remove a vital and significant coping mechanism for an individual and may also cause

negative affect withdrawal symptoms (Geller, Cockell, & Goldner, 2000; Morris, Steinberg, Sykes, & Salmon, 1990). Conversely, healthy exercise engagement and education during treatment has been associated with improved quality of life, body composition, central health markers of the illnesses (e.g., drive for thinness, weight and shape concerns and eating restraint), and improved comorbid physical and psychological symptomatology (e.g., anxiety, depression, muscle degradation, body esteem issues, sleep disturbances, perceived stress and osteoporosis) in those with AN, BN and EDNOS (Hausenblas et al., 2008; Ng et al., 2013; Moola et al., 2013; Vancampfort et al., 2014).

2. Safe and Healthful

EDs (AN in particular) are associated with high morbidity and mortality rates (Bulik et al., 2005; Crow, 2014; Reel, 2013). Furthermore, individuals with an ED who engage in dysfunctional exercise are more likely to have a chronic ED, severe psychopathologies, and a higher risk of relapse (Dalle Grave et al., 2008). Considering these deleterious outcomes, it is crucial that exercise prescription in ED treatment promotes the safety of clients. Safe exercise prescription is currently limited; due, in part, to gaps in knowledge among health professionals (Quesnel et al., 2017). This limited knowledge has likely contributed to uncertainty and confusion related to the safety of clients engaging in any exercise during ED. The SEES guideline has subsequently been developed to provide a summary of the current evidence with the hope that this will address concerns related to safety and improve upon clinical knowledge of exercise engagement during ED treatment. Furthermore, the SEES guideline was developed to promote physical and psychological safety as paramount for any exercise intervention. We hope that by providing a safety-focused guideline, SEES may help alleviate both the lack of knowledge as well as concerns of health professionals regarding the prescription and management of exercise during ED treatment.

3. Holistic

The relationship with exercise is multifaceted; comprising physical, emotional, social, cognitive, and sociocultural components (Calogero & Pedrotty, 2007). Engagement in exercise, consequently, influences and is influenced by each of these individual components (Calogero & Pedrotty, 2003). While clinical management of exercise in ED treatment commonly considers the physical risk of exercise engagement, this can be at the cost of addressing the psychological relationship with exercise. As highlighted above, safe and healthful exercise has been paramount in developing the SEES guideline; however, we also further define this relationship with exercise

to incorporate these socio-emotional aspects of exercise engagement. Addressing these dimensions through a holistic lens is integral to supporting individuals in developing a healthy relationship with exercise. Our prioritisation of a holistic approach underpins the combination of medical, cognitive, emotional, and behavioural benchmarks in the SEES guideline. This decision-making process aims to guide clinicians in supporting their clients to begin and continue safe exercise engagement, contributing to this multifaceted construct of well-being.

4. Mindful and Intuitive Movement

A dysfunctional relationship with exercise is often characterised by rigid inflexibility, punitive attitudes, and guilt (Meyer, Taranis, Goodwin, & Haycraft, 2011). Clinicians supporting clients to reduce dysfunctional exercise must, consequently, address each of these mechanisms to promote a healthy relationship with exercise. Building skills in mindful and intuitive movement can be a key strategy to facilitate this transition. Mindful and intuitive movement is defined as, “movement that is done with attention, purpose, self-compassion, acceptance, awareness, and joy... focused on the process of becoming more connected, healthier, and stronger” (Calogero & Pedrotty, 2010 p.434). Clinicians can support their clients to begin mindful movement through providing opportunities to learn to listen to their physiological and psychological cues prior to, during, and after exercise engagement and use these internal signals to choose and adjust their own health-enhancing movement (Calogero and Pedrotty, 2007). This process aims to support individuals to foster trust in their bodies’ preferences and needs related to exercise contributing to the likelihood of positive, rather than destructive, health outcomes over time (Calogero & Pedrotty, 2003). According to Calogero and Pedrotty (2007, p.184), mindful and intuitive movement should:

1. Rejuvenate the body, not exhaust or deplete it.
2. Enhance mind–body connection, not allow or induce disconnection.
3. Alleviate mental and physical stress, not produce more.
4. Provide genuine enjoyment and pleasure, not pain and dread.

5. Collaborative

Developing a strong therapeutic alliance in treatment is a well-established predictor of positive treatment outcomes (Graves et al., 2017). Collaborative client-practitioner relationships have been identified as more likely to prevent dropout, reduce client ambivalence toward change, and increase treatment acceptability by both clients and clinicians (Geller et al., 2003). The SEES guideline has, consequently, been designed to promote collaboration between the

individual, their loved ones, and a team of experienced eating disorder professionals to help guide safe exercise engagement at all stages of ED treatment and recovery. This approach differs from directive approaches in that it emphasises listening to the individual's needs and preferences, addressing client motivation and ambivalence, and fostering client autonomy. It should be noted that collaborative approaches are not wholly client-directed. Rather, collaborative approaches view clients as an active member in clinical decision-making and, consequently, these decisions are still made within the context of clinicians prioritising the client's engagement in safe and healthful exercise. We aim that this approach will facilitate open and honest conversations with clients about exercise, identifying their needs and concerns about exercise engagement.

Overview of the SEES guideline

Objective

Dysfunctional exercise is associated with a range of deleterious outcomes for individuals with ED symptomatology; yet, conversely, safe exercise engagement can predict positive physical and mental health outcomes in these populations. As individuals with ED symptomatology often exhibit an array of medical health complications, it can be challenging for clinicians in this field to determine safe prescriptions for exercise engagement. The SEES guideline was developed to address the management of exercise in adult ED populations. Recommendations in this guideline are intended to provide guidance for clinicians to better facilitate clinical decision-making related to exercise prescription for individuals with ED symptomatology. We aim that the use of the SEES guideline will reduce the likelihood of over- and under-prescribing exercise interventions relative to the client's individual physical and psychological health status. Consequently, this is intended to support individuals with ED symptomatology to engage in safe levels of exercise and exercise education throughout their treatment, contributing to improved psychological and physical outcomes.

Target users and population

We developed the SEES guideline for use by trained exercise and medical professionals working with individuals with ED symptomatology. This guideline has been developed for use across all levels of care including inpatient and medical settings as well as outpatient settings. The SEES guideline is intended to augment the clinical and medical knowledge of clinicians working with these populations. The application of the guideline to prescribe exercise should only occur within an individual clinician's scope of practice.

We specifically intended that the SEES guideline apply to individuals at all stages of their ED and to not determine health status based upon an individual's specific diagnosis (for example, bulimia nervosa or anorexia nervosa). Consequently, this guideline was developed for use with individuals over the age of 18 exhibiting ED symptomatology (e.g. dietary restriction, purging, and compensatory behaviours). The SEES guideline may require adaptation by a trained medical team or accredited exercise professional (pp.63) for special populations such as: athletes, children/adolescents, and individuals with diabetes, osteopenia/osteoporosis, or other existing cardiovascular/respiratory, metabolic, neurological, psychological or musculoskeletal complications. This guideline may be incorporated by clinicians working with these populations

for individuals exhibiting either under- or over-exercising behaviours to support a healthy relationship with exercise. We actively recommend against determining medical risk solely based upon weight or BMI and have, consequently, developed the guideline to apply to individuals with ED symptomatology regardless of weight or BMI.

Scope

- The SEES guideline is a tool for supporting clinical decision-making regarding exercise interventions for health professionals working with clients with eating disorder symptomatology.
- The SEES guideline is intended for use in all levels of care for individuals experiencing an ED or ED symptomatology.
- The SEES guideline applies to individuals at every stage of their eating disorder, regardless of their weight, size, or individual diagnosis.
- The SEES guideline was developed for use in adult populations and is not suitable for use with children or adolescents.
- The SEES guideline is not intended to replace clinical or medical judgment and recommendations should be made with consideration of the clients' individual needs.
- The SEES guideline may need to be adapted or not be appropriate for individuals with comorbid conditions.

Methodology

The guideline has been developed using a thorough review of the best available research in conjunction with input by experts in the field of eating disorders and related conditions. The AGREE-II instrument (AGREE Next Steps Consortium, 2009) was used to support the development of the guideline in conjunction with the NHMRC (2011) instrument to ensure that SEES meets high standards.

The authors completed the following procedure to support the development of SEES:

- Review of existing guideline.
- Two focus groups with both steering and consultation committees.
- Meta-analytic review of exercise interventions in ED populations.
- Consultation with steering committee experts.

- Review of final guideline by consultation committee (including clinicians and researchers from multiple relevant disciplines, as well as individuals with lived experience of an ED)
- Approval of SEES final document by steering committee

Method of consultation process

Our initial consultation process was undertaken in a sample of members from both our consultation committee and steering committee (pp.3-4). Participants were able to provide their feedback during two online focus groups or via online submission of information. Following completion of the SEES guideline, the consultation committee was again invited to provide feedback related to the guideline.

Results of initial consultation process

Feedback from initial consultation was as follows:

Potential contraindications to exercise. Recommendations regarding minimum BMI was controversial in the group, with current practices ranging from 14-18kg/m² and other individuals suggesting that BMI was not as important as other physiological measurements. Common potential contraindications included electrolyte imbalances and cardiovascular abnormalities (e.g. bradycardia, postural tachycardia and a prolonged QT interval). Only one participant highlighted psychological factors that may contraindicate exercise.

Factors that may support increased exercise engagement. These included an increased BMI, treatment compliance, improvements in cardiac health, medical stability, and reduced ED pathology and exercise compulsions. Suggestions were varied regarding how rigid these steps should be as well as the stage that exercise could be safely increased.

Exercise interventions. Exercise practices for higher levels of medical instability ranged from exercise abstinence to interventions tailored and monitored to the individual, stretching, gentle movement such as Tai Chi or Yoga, and body weight exercises. As health improved, clinicians recommended engaging in moderate levels of exercise such as dancing, swimming, walking, and stretching, progressing to higher levels of strength/resistance training and cardiovascular training. Appropriate durations of exercise were suggested to range from 10-60 minutes. Commonly used intensity ratings included rate of perceived exertion, talk test, and heart rate monitoring, whilst safe exercise frequency suggestions ranged from once per week to daily. Multiple respondents recommended beginning with supervised and transitioning to unsupervised exercise.

Strategies for reducing harm related to exercise. Here, the consultation committee recommended strategies that related directly to exercise (e.g. promoting diversity, non-repetitive, social and reduced duration/intensity/frequency exercise), the utilisation of exercise professionals for monitoring and prescription of activity as well as the inclusion of medical professionals for medical monitoring, and adjunct treatments (e.g. CBT, psychoeducation, LEAP, DBT, FBT, ACT and medication) to help promote client insight, healthy exercise beliefs, and intuitive movement.

Strategies for preventing exercise relapse. Respondents most commonly noted psychotherapeutic approaches, particularly CBT, as well as education, relapse planning, increasing awareness of one's physiological cues, challenging unhelpful exercise beliefs, symptom monitoring, goal setting and identifying triggers which cause dysfunctional exercise thoughts or engagement.

Important components of the exercise guideline. Recommendations for the future guideline were opposing, with some professionals asking for clearer guidance, cut offs, checklists, and rationales, whilst others encouraged a less black-and-white approach, more client-centred collaboration, less rule-based, and more individualised guideline. Other responses included guidance related to nutrition, increased awareness of physical cues, recognition and validation of the functions of exercise, strategies for supporting clients to reduce exercise and educational tips for clients.

Concerns about the guideline. Aligned with the above important comments, respondents were split as to concerns about the guideline being overly rule-based and restrictive for individuals, or, opposingly, were cautionary that the guideline must be clear to follow and indisputable. Other key concerns highlighted were the need for client education, professionals using guideline outside of their scope of practice, improper use of the guideline, the capacity to implement recommendations in an outpatient setting, as well as the potential for the guideline to contribute to beliefs that are overly permissive of client exercise or increase clinician fear related to exercise engagement.

Key knowledge for clinicians. Respondents encouraged clinicians to be aware of the role of exercise in the ED, as well as the need for individualised approaches, benefits and risks of exercise, and the importance of a collaborative relationship with clients.

Concerning comorbidities/medications. These included supplement use in athletes, as well as individuals with depression, physical injuries, medical instability, or comorbid diagnoses including diabetes, celiac disease, osteopenia, osteoporosis and pre-existing cardiovascular/pulmonary complications.

Recommendations for exercise assessments. Weekly at least initially was nearly universally recommended. Other recommendations included once every two to three months.

Literature search strategy and data extraction

The two first authors (AD and MC) conducted comprehensive systematic searches of the literature during the period of January to March 2018. We searched for articles published prior to these dates on the following electronic databases: Medline, PubMed and PsychArticles. Search terms included: “eating disorder* OR anorexi* OR bulimi*” AND “exercise OR physical therapy OR physical activity” AND “cardiorespiratory OR mental OR metabolic OR neurological OR musculoskeletal OR mortality OR adverse events OR injury.” A supplementary examination of reference sections for additional papers was also conducted. Inclusion criteria were as follows, studies must meet the following:

1. Be original research reports only;
2. Be from a peer-reviewed journal;
3. Include either a participant/group of participants identified as experiencing an eating disorder;
4. Have physical activity or exercise as a main antecedent or exposure variable;
5. Report on a health outcome variable or risk factor is clearly described and fits into one of our health outcome categories (as described in search terms); and
6. Be written in English.

The two authors initially screened all titles, duplications, and publication types to remove articles that could immediately be excluded. A further review of abstracts and full text manuscripts was then conducted independently to determine eligibility for inclusion. Any differing opinions between the raters were discussed to reach a consensus decision. MC, subsequently, extracted data related to: study design, setting, client diagnosis, sample characteristics, exercise intervention details, reported outcomes, and contraindications. Further

details regarding the search strategy and data analysis will be published in an upcoming meta-analyses.

Results of search process

This review process resulted in a total of 191 studies that were included in resultant systematic reviews and meta-analyses (separated by children/adolescent and adults). Core findings were presented at the 2018 Australian and New Zealand Academy for Eating Disorders conference and will be published in upcoming meta-analyses.

Monitoring and Audit Process

We intend that the SEES guideline is reviewed for updated information in 2020; although, this review may occur earlier should key information relevant become available. The primary investigators will review updates to the literature at this time and, where applicable, update aspects of the guideline based upon the latest scientific evidence. Any modifications to the SEES guideline will be conducted under the review of our steering committee. Users of the guideline are encouraged to forward recommendations for adaptations and relevant literature to the SEES guideline through our website (<https://www.safeexerciseateverystage.com/>).

Limitations

It is important to highlight the following limitations to the SEES guideline:

- This guideline was developed based upon clinical and research understanding of exercise intervention and ED treatment at the time it was written. Throughout this development process, we identified highly divergent opinions on safe exercise interventions from our consultation committee clinicians working in this field. To rectify these contrasting opinions within our guideline, we relied on both the current research evidence as well as the expert opinions of our steering committee.
- We recognise that there is currently a limited evidence base to support the safe use of exercise in ED populations, particularly in outpatient settings. Consequently, the SEES guideline will require updating as research into this area evolves.
- This guideline was not developed as a manual for exercise intervention. It is a review of current literature and practices of exercise engagement in adult ED populations. Consequently, it is intended that the SEES guideline will support clinicians in their prescription of safe exercise and exercise education adjunctive to their implementation of an evidence-based treatment approach.

- This guideline does not purport to train clinicians in the physiology and psychology of exercise in individuals with ED symptomatology. Instead, it provides recommendations for clinicians already trained in these areas to support clinical decision-making. We also recognise that there are situations where access to specialised ED clinicians is not possible, often due to geographic or economic resources. Consequently, we aim that the SEES guideline will support trained medical and exercise professionals (pp.63) in these situations to augment their current clinical judgement and knowledge.
- The SEES guideline was not developed to address the specific needs of diverse cultural or special populations. We recommend that clinicians applying the SEES guideline should tailor interventions to each individual's needs.

Application of Guidelines

The SEES guideline is a summary of current evidence and clinical practices for the management of dysfunctional exercise in ED treatment. It is a tool to augment clinical judgment and aims to provide a standard of practice for exercise interventions. In creating this clinical tool, we will review evidence-based strategies to support the implementation of the SEES guideline and recognise potential barriers in the application.

Facilitating the implementation of SEES

The following are encouraged strategies for supporting the implementation of the SEES guideline when working with individuals with an ED.

In vivo tasks in session. Calogero and Pedrotty (2007) emphasise that dysfunctional exercise is defined by the quality, not simply the quantity, of activity that individuals are engaging in. These qualitative aspects can include high rigidity, inflexibility, and compulsivity (Meyer et al., 2011). In vivo tasks during treatment provide an approach that may facilitate connection with the quality of exercise. Calogero and Pedrotty (2007) describe these tasks as a form of “debriefing,” in which clinicians encourage clients to become mindful before, during, and after exercise sessions. They note that debriefing consists of reflecting on sensations, emotions, and thoughts evoked by the exercise session and that these reflections may occur in session as well as over time with an exercise journal. For a further review of potentially helpful in-session activities, see Calogero and Pedrotty (2010) and Taranis and colleagues (2011). We also recommend that clinicians and clients collaboratively determine exercise types that foster fun and sociability, particularly when in Level B and C of the guideline (Quesnel et al., 2017).

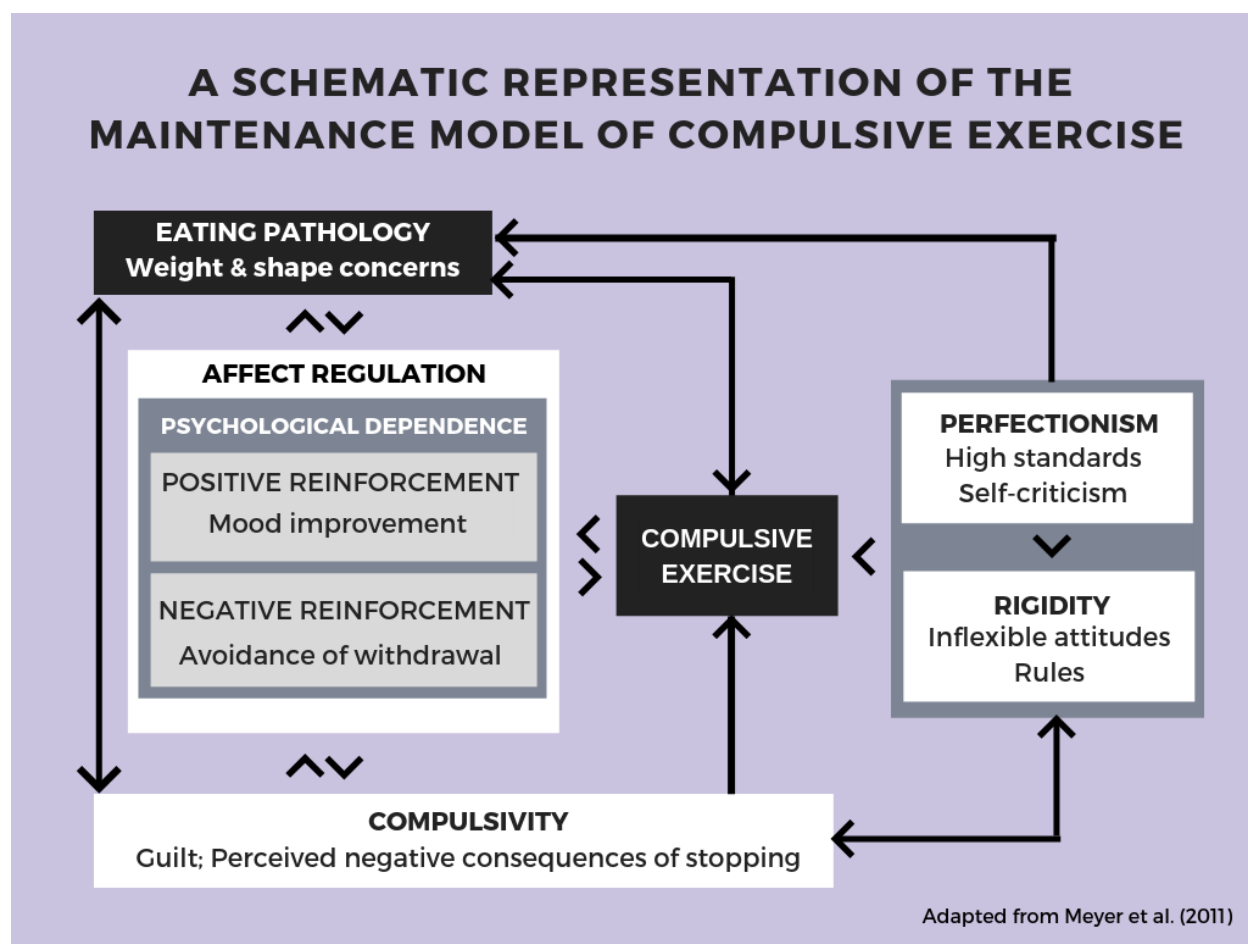
Involve social support. There are multiple ways in which support people and carers can be involved to help achieve optimal outcomes for individuals with EDs returning to exercise. These are suggested to include: improved assessment procedures, helping with behaviour monitoring, supporting behaviour change, and provision of emotional support (National Institute for Clinical Excellence, 2017). The NICE guideline (2017) highlights the importance of the following factors when involving carers in ED treatment: psychoeducation regarding ED and the impact of mental health issues on carers; information regarding available treatments; clinician awareness of carers potential feelings of guilt or responsibility for the ED; communicating with empathy, compassion and, respect; and note risks of carer burden.

Motivational and collaborative approach. The importance of a motivational approach in ED treatment is recognised by both clients and clinicians alike (Geller et al., 2003). This approach is suggested to reduce client ambivalence for change and increase treatment acceptability. Further to these benefits, Geller and Srikameswaran (2006) recommend that this approach may be helpful in establishing treatment non-negotiables, upholding the values of client safety, autonomy, and respect. Clear therapeutic boundaries continue to be paramount in collaborative settings to ensure medical and psychiatric safety, efficient use of treatment resources, and a level of client and clinician accountability (Geller, Goodrich, Chan, & Srikameswaran, 2012). For a full review of developing non-negotiable treatment components, see Geller & Srikameswaran.

Promote intuitive movement and its benefits. Intuitive movement involves learning to evaluate and appropriately respond to physiological and psychological cues prior to, during, and after exercise engagement (Calogero & Pedrotty, 2007; 2010). Calogero and Pedrotty (2003) suggest that with increased awareness of the impact of exercise, individuals can use this knowledge to adjust the frequency, intensity, duration, type, rest, and recovery of exercise engagement. This is built on by Douillard (2001), recommending that intuitive movement emphasises the process of movement as opposed to exercise outcomes.

Increase client awareness of the function of exercise in their ED. Conceptualising the role of exercise within the ED is helpful for both the clinician aiming to address dysfunctional exercise as well as for the client wishing to gain a further understanding of their relationship with exercise. Meyer and colleagues' (2011) have presented an evidence-based model that clinicians may find helpful in supporting the development of this conceptualisation (Box 1). They postulate that eating pathology (including weight and shape concerns) is a core aspect in the development and maintenance of compulsive exercise; with compulsive exercise subsequently serving to reinforce eating pathology. The authors also build upon Fairburn's transdiagnostic theory of ED (Fairburn, Cooper, & Shafran, 2003) and addiction models, highlighting the important role of exercise in not only managing negative affect, but also increasing positive affect and possessing negative withdrawal symptoms (Meyer et al., 2011). Meyer and colleagues (2011) draw further similarities to obsessive compulsive symptomatology, noting the reinforcing roles of perfectionism (including self-criticism), compulsivity (and fear/guilt of ceasing exercise) and rigidity. Additionally, the authors present the Exercise Profile tool as a tool to determine the strongest maintaining factors (Taranis et al., 2011). Calogero and Pedrotty (2010) suggest that

debunking maintaining factors may help shift the dysfunctional exercise cognitions and behaviours toward a healthy exercise relationship.



Box 1. A schematic representation of the maintenance model of compulsive exercise.

Psychological strategies for managing dysfunctional exercise. Psychological interventions for addressing dysfunctional exercise have commonly adopted a CBT approach. As SEES is a clinical guideline, not a treatment approach, it is recommended that it be utilised in conjunction with evidence-based interventions. Psychological interventions for addressing exercise have included strategies such as: understanding exercise motivations, psychoeducation, exercise logging, increasing body awareness, self-reflection, diaphragmatic breathing, and cognitive restructuring (Calegoro & Pedrotty, 2004; Schlegel, Hartmann, Fuchs, & Zeek, 2015; Taranis et al., 2011).

Create a written exercise contract. Cook and colleagues' (2016) highlight that the development of a collaboratively written contract may be useful in ED treatment. They encourage that this

contract may include goals, outcomes, expectations, rules and contingencies to facilitate transparency and clarity in treatment (Cook et al., 2016). The key components of behavioural contracting highlighted are: collaboration, relevant goal setting, clear monitoring of exercise within the parameters of the guidelines, and setting appropriate consequences. Finally, it is noted that behavioural contracts should be flexible and collaborative with other treating team members (Yager, 1992).

Multidisciplinary approach. The importance of a multidisciplinary team in ED treatment is commonly cited as a key component for recovery (Wonderlich et al., 2012). Clinical guidelines for eating disorders recommend that this team should minimally include professionals administering medical, dietetic, and psychological interventions (Hay et al., 2014). Trained exercise professionals may also be helpful in the presence of dysfunctional exercise. Further, physical therapists as part of the rehabilitative team can help assist with an often overlooked heightened falls risk (during ambulation, and standing from lying or sitting), and later, to complement nutritional rehabilitation during the medical stabilisation process (Laging, Brinton, Sabel, L. Gaudiani & Mehler, 2017).

Barriers to implementing SEES

We recognise several potential barriers to the implementation of the SEES guideline as well as those related to exercise engagement in ED treatment.

Access to specialist ED teams. Geographic and financial factors are commonly recognised as a barrier to treatment access. In particular, research has recognised that public health care systems may be limited in their funding and resources including both specialised staff (pp.63) and program resources including equipment and program development (Quesnel et al., 2017).

Clinician scope of practice. The SEES guideline is not intended to replace clinical training or education. Consequently, its effective implementation is limited by the skills and knowledge of the clinicians utilising it. The SEES guideline should only be used by trained medical and accredited exercise professionals (pp.63) with expert knowledge in the physiology of ED.

Professional attitudes and perceptions of exercise and eating disorders. Health professionals' attitudes and perceptions regarding the inclusion of exercise into eating disorder treatment have

been identified as one of the most prominent barriers to incorporating exercise in ED treatment (Quesnel et al., 2017). The hesitancy around incorporating exercise into ED treatment has been explained to stem from the historical beliefs describing patients engaging in exercise as a “pathological calorie wasting mechanism employed to limit energy reserves” (Davis, Kapstein, Kaplan, Olmstead, & Woodside, 1998, p.3). This notion has been perpetuated by an absence of clear guidance and fear of negative consequences resulting from prescribing exercise to patients during treatment. Despite this, there has recently been a shift in perspectives of health profession regarding exercise and EDs. Emerging attitudes and perspectives suggest that the intention is now to have safe exercise prescription become part of treatment (Quesnel et al., 2017). The SEES guideline aims to alleviate some of these historical tensions and help facilitate the process of formalising safe exercise prescription into ED treatment.

Resistance to change. Although we have used the term ‘dysfunctional exercise,’ throughout this document, we recognise that even this mode of exercise serves a function, commonly as a coping mechanism. Consequently, clients can be resistant to the concept of reducing their exercise engagement (Meyer et al., 2011). Both the development of a collaborative client-practitioner relationship (Geller et al., 2003), as well as the psychological interventions highlighted above (Calegoro & Pedrotty, 2004; Schlegel et al., 2015; Taranis et al., 2011), may be helpful in addressing resistance to change.

The Safe Exercise at Every Stage Guideline

Instructions for use

The Safe Exercise at Every Stage (SEES) guideline was developed to better facilitate clinical decision-making related to safe exercise for individuals with eating disorder symptomatology. This step-up/-down model involves two key components:

1. **Risk assessment:** Reviews key markers of psychological and physical health requiring assessment to facilitate safe exercise prescription.
2. **Exercise recommendations:** Describes exercise interventions as related to the level of risk identified in the risk assessment.

This guideline does not replace clinical judgment, but rather augments the ethical and clinical decision-making process. It is expected that clinicians review clients' medical and psychological progress in accordance with the guideline's risk levels regularly (i.e. weekly in Level A – highest risk, decreasing in relation to risk). We also recommend that psychological intervention occurs concurrently to exercise and nutrition interventions to best support clients. These should include addressing factors contributing to the development and maintenance of dysfunctional exercise behaviours as well as building healthy coping strategies.

Importance of Safe Exercise at Every Stage

Graded exercise can be safely undertaken during eating disorder treatment to achieve positive outcomes such as improved eating disorder symptomatology, general psychological well being, cardiac functioning and musculoskeletal health, as well as increased adherence to meal plans and treatment. However, the exact exercise recommendation for each client will differ depending on their level of physical and mental health risk in relation to exercise.

Using the SEES guideline

This guideline was developed to support clinicians in making evidence-based decisions when recommending safe exercise for their clients. It is important that exercise sessions be supervised in the initial stages with increasing autonomy permitted as treatment and recovery progress. Please note that it is not a requirement, nor always possible, for supervision to be conducted by a treatment team member. As such, we recommend that a trusted friend or partner with knowledge of the individual's exercise plan and limitations be present for these sessions. Regular medical reviews are required to decide whether the current exercise is maintained, progressed, or regressed, depending on client symptomatology and physiological results.

1. **Assessment:** Use psychological and physical results (as per page 28) to determine your client's level of risk when engaging in exercise. Always begin the assessment using the markers from the highest-risk category, Level A (as outlined on page 28). If no risk factors from Level A are identified, assess the measures in Level B, and so on. Note the risk category your client falls within.
2. **Recommendations:** Once the level of exercise risk has been identified (e.g. Level A), match with the corresponding exercise recommendation on page 29. Please note that even once an individual positively progresses past Level A (highest risk), clinicians are still recommended

to continue interventions from this level as they include important education regardless of health status. This continues to apply at Level D (lowest risk), whereby interventions from Level A, B and C should continue to be implemented.

3. **Step up/step down:** Individuals may step up (into the lower risk categories) and down (higher risk) on the guideline throughout treatment due to the non-linear nature of recovery. Specific reviews (e.g. weekly, monthly) are recommended in each level for this reason. Stepping up requires not only the clearance of all risk markers up to and including their current level, but individuals must also be adhering to treatment, increasing nutritional consumption and exhibiting improvements in health status. Conversely, an individual will step down to previous level/s if they exhibit any of the higher risk markers. Individuals must also step down a level with treatment/meal plan non-compliance, return to exercise compulsions, or a worsening of ED behaviours.

Intensity, Duration and Type of Exercise

The SEES guideline provides recommendations regarding the intensity, duration and type of exercise, however, deliberately does not specify the frequency of exercise sessions per week. This must be collaboratively determined by both the clinician and individual to prioritise safety, optimal treatment outcomes, and minimise harm.

Exercise is a positively indicated treatment component but is not compulsory and boundaries are important to prevent returning to dysfunctional exercise. Clinicians should work with clients to help them listen to their body signals prior to, during and after exercise sessions. This knowledge can then be incorporated into learning to match exercise type, intensity, and amount to their energy levels, creating exercise autonomy. Supervising professionals must be aware of each individual's limitations and any changes in energy and/or symptomatology to adjust exercise accordingly. This includes incidental physical activity (such as walking to appointments/work, cleaning/gardening, carrying groceries), which the clinician must discuss with their client and consider in addition to recommended exercise to determine an individual's total daily physical activity.

Limitations

This guideline does not replace clinical judgement by the treatment team. It has been developed for the use of trained medical and exercise professionals with expert knowledge in the physiology of eating disorders when working with the general adult population (aged 18 and over). Some special populations will need further support and must be assessed by a medical team and, where accessible, an accredited exercise professional (see glossary) before recommending an appropriate supervised exercise plan. Please note, this does not preclude these special populations from engaging in exercise; however, we encourage that adaptations to the SEES guideline for these populations must be done under the supervision of medical advice specific to their individual requirements. These populations may include (but are not limited to): Athletes, children/adolescents, and individuals with diabetes, osteopenia/osteoporosis, or other existing cardiovascular/respiratory, metabolic, neurological, psychological or musculoskeletal complications. Finally, whilst purging as a behaviour has not been included as a contraindication to exercise, we encourage practitioners to ensure a thorough and frequent assessment for individuals engaged in vomiting, laxative, or diuretic use and exercise due to the compromising nature of these behaviours (see *Purging and Hypovolemia*).

Increasing progression with nutritional plan

Level A Review weekly	Level B Review fortnightly	Level C Review monthly	Level D Review as required
<p>Cardiovascular profile: HR<44bpm or >120bpm Postural tachycardia >20bpm Orthostatic hypotension >20mmHg systole (independent of symptoms) Systolic BP <90mmHg Prolonged QT/c interval >450msec Arrhythmias</p> <p>Biochemical profile: Hypokalemia <3.0mmol/L Hypophosphatemia <0.8mmol/L Hypomagnesemia <0.7mmol/L Hypercarbia>32mmol/L Hyponatremia <130mmol/L Hypoglycaemia <4mmol/L</p> <p>Psychological profile: Dependent category in Exercise Dependence Scale</p> <p>Other: Temperature <35°</p>	<p><i>Individual has cleared all prior risk markers and is also adhering to:</i></p> <p>Individuals with AN: Positive weight gain trajectory in line with treatment goals</p> <p>Weight-restored individuals: Weight stabilisation/mobilisation in line with treatment goals</p> <p>Recommended to assess BMD if: (i) underweight for > 6mths (ii) amenorrhea for > 6mths (iii) low testosterone in males (iv) history of stress or fragility fractures</p>	<p><i>Individual has cleared all prior risk markers and is also adhering to:</i></p> <p>Weight stabilisation or gain if still required</p> <p>Level A markers related to ED are completely normalised as per medical recommendation</p> <p>Managing ED behaviours (e.g. self-induced vomiting, restriction/ bingeing, fear of becoming fat, & laxative use)</p> <p>Normalised sex hormones without exogenous replacement (return to menses & normalized oestrogen for females; testosterone for males)</p>	<p><i>Individual has cleared all prior risk markers and is also adhering to:</i></p> <p>Weight progression >90% of IBW (considering individual weight history & family characteristics)</p>

Symptom regression, treatment/meal plan noncompliance, return to exercise compulsion

Exercise Components:	SEES Recommendations: Level A	Level B	Level C	Level D
Intensity	Max Talk Test level: 2 METs: <3	Max Talk Test level: 5 METs: 3-5	Max Talk Test level: 8 METs: 6-8	Individualised
Duration	30min max	30min max	60min max (30min max cardio; 30min max resistance)	Individualised
Stretching	Static (without orthostatic compromise)	Dynamic warm up; static cool down		
Cardiovascular/ respiratory exercise	Nil	Low impact; social/games focus (excluding return to sport) (e.g. gentle Yoga and Pilates, table tennis, walking, swimming)	Moderate impact (excluding return to sport) (e.g. cardio classes, jogging)	High impact; return to sport (e.g. rugby, football, martial arts, basketball, hockey); individualised; or may return to previously dysfunctional cardio exercise
Resistance exercise	Nil	Social, functional body weight (e.g. circuit)	All resistance exercise (e.g. weight lifting, weights classes)	All resistance exercise; may return to previously dysfunctional resistance exercises
Setting	Indoor or outdoor			
Supervision	Medical supervision required	Medical OR friend/family	Flexible (social partner encouraged)	Flexible, progressing to unsupervised
Education	Identify unhealthy exercise beliefs Nutrition support Ambulation assessment & injury prevention in daily living tasks (e.g. correct bending technique) Breathing & body awareness tasks Introduction to intuitive movement Assessment of exercise habits prior to treatment & long-term exercise goals Physiological education Consider suggestions in <i>Facilitating the implementation of SEES</i> section	Continue relevant/outstanding interventions and: Further challenge unhealthy exercise beliefs Continue exploring & practicing intuitive movement	Continue relevant/outstanding interventions and: Increase exercise intensity in conjunction with body awareness Set future exercise goals	Continue relevant/outstanding interventions and: Address remaining unhealthy aspects of exercise relationship, renormalising & increasing autonomy Develop future exercise plan in accordance with treatment plan & activity goals including focus on relapse prevention

MEDICAL COMPLICATIONS OF EATING DISORDERS AND EXERCISE

EATING DISORDER (IRRESPECTIVE OF WEIGHT, SHAPE OR SIZE)

↓ ENERGY & FLUID AVAILABILITY, MALNUTRITION, STARVATION, PURGING

HEALTH CONSEQUENCES OF ED

<p>PSYCHOLOGICAL</p> <ul style="list-style-type: none"> Exercise dependence Anxiety Depression Irritability Dysfunctional attitudes Emotional distress ↓ stress tolerance Interpersonal dependence Pain medication reliance & side effects ↓ health related quality of life Withdrawals from exercise Neglect of life for exercise 	<ul style="list-style-type: none"> Angina Palpitations Heart attack Mitral valve disease Torsade de pointes Organ damage & failure Abnormal blood oxygen saturation Aortic obstruction Pericardial effusion ↓ stroke volume ↓ left ventricular mass Peripheral blood pooling 	<p>Rhabdomyolysis</p> <ul style="list-style-type: none"> Tetany Catabolism <p>SKELETAL</p> <ul style="list-style-type: none"> Uncoupling of bone ↓ bone mineral density & geometry ↓ lying down of lifetime bone Difficulty reacquiring bone Osteoporosis & osteopenia Cortical thinning ↓ trabecular number & density ↓ space between trabeculae ↓ bone calcium regulation Permanent postural damage 	<ul style="list-style-type: none"> Hypophosphatemia Hypomagnesemia Hypercarbia Hyponatremia <p>HYDRATION</p> <ul style="list-style-type: none"> Hypohydration Dehydration Hypovolemia <p>TEMPERATURE</p> <ul style="list-style-type: none"> Hypothermia Hyperthermia General coldness 	<ul style="list-style-type: none"> ↑ transaminase Hypoglycemia <p>SEXUAL</p> <ul style="list-style-type: none"> ↓ estrogen ↓ testosterone ↓ luteinising hormone ↓ follicle stimulating hormone Risk of menstrual disturbance & dysfunction Functional hypothalamic amenorrhea <p>COMORBID ILLNESS</p> <ul style="list-style-type: none"> ↑ risk of developing an illness 	<p>OTHER SYMPTOMS</p> <ul style="list-style-type: none"> Fainting Unconsciousness Dizziness Fatigue Confusion Weakness Light headedness Visual blurring or tunnel vision Poor concentration Nausea Headaches Early satiety Pallor Cold, clammy skin Constipation Delirium Coma Death Seizures Cyanosis Vomiting Abdominal pain Rectal prolapse Hospitalization risk Poor long term outcomes
<p>CARDIOVASCULAR</p> <ul style="list-style-type: none"> Bradycardia Tachycardia Postural tachycardia Orthostatic hypotension Hypotension Prolonged QTc interval Arrhythmias Superior mesenteric artery syndrome Cardiac arrest Heart failure 	<p>RESPIRATORY</p> <ul style="list-style-type: none"> Shortness of breath Rapid, shallow breathing Hyperventilation Respiratory compromise Respiratory paralysis <p>MUSCULAR</p> <ul style="list-style-type: none"> Muscular dysfunction Weakness Cramping Tremors & fasciculations Pain 	<p>NEUROLOGICAL</p> <ul style="list-style-type: none"> Autonomic nervous system dysfunction Neuralgia Ataxia Vertigo Dysphagia Requirement of pain medication Irreversible brain damage <p>ELECTROLYTES</p> <ul style="list-style-type: none"> Hypokalemia 	<p>METABOLIC</p> <ul style="list-style-type: none"> ↓ resting metabolic rate ↓ glycogen ↓ leptin ↓ triiodothyronine ↓ growth hormone ↓ insulin-like growth factor 1 ↓ urine specific gravity ↑ blood urea nitrogen ↑ ghrelin ↑ peptide tyrosine tyrosine ↑ cortisol 	<p>ANTHROPOMETRY</p> <ul style="list-style-type: none"> ↓/↑ body fat percentage ↓/↑ body mass index ↓/↑ ideal body weight ↓/↑ fat mass <p>HAEMATOLOGICAL</p> <ul style="list-style-type: none"> Anemia <p>IMMUNOLOGICAL</p> <ul style="list-style-type: none"> ↑ risk of infection 	

CONSEQUENCES OF EXERCISING WITH AN ED WITHOUT APPROPRIATE MODIFICATION

<p>PSYCHOLOGICAL</p> <ul style="list-style-type: none"> Further exercise dependence ↓ psychological capacity 	<ul style="list-style-type: none"> Worsened long-term bone health ↑ amino acid catabolism ↑ stress fracture risk & prevalence ↑ muscle pain due to circulatory lactate ↓ oxygen perfusion, uptake & utilization ↓ muscle strength ↑ injury risk ↓ concentration in sport ↓ judgement in sport ↓ coordination ↓ training adaptations & responses 	<p>ELECTROLYTES</p> <ul style="list-style-type: none"> ↑ electrolytes lost in sweat <p>HYDRATION</p> <ul style="list-style-type: none"> Hypohydration Hypovolemia <p>TEMPERATURE</p> <ul style="list-style-type: none"> ↑ risk of heat illness & heat stroke 	<p>METABOLIC</p> <ul style="list-style-type: none"> Induce or worsen hypoglycemia Adrenal dysfunction ↑ blood urea nitrogen (indirectly) <p>SEXUAL</p> <ul style="list-style-type: none"> Induce or worsen FHA & its associated risks <p>COMORBID ILLNESS</p> <ul style="list-style-type: none"> ↑ risk of negative outcomes with comorbid condition 	<ul style="list-style-type: none"> Organ dysfunction <p>OTHER</p> <ul style="list-style-type: none"> Exercise intolerance Fatigue Time away from sport Relative Energy Deficiency in Sport ENERGY AVAILABILITY FURTHER DECREASED
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Health Complications: Unmodified Exercise with an Eating Disorder

The following section reviews *health complications* related to eating disorders which may contraindicate or detrimentally impact unmodified exercise engagement. In addition, Table 1 (pp. 61) lists *signs and symptoms* that may contraindicate or impact exercise engagement:

Low energy availability (LEA)

LEA occurs when a person's energy availability required for living falls below 30 kilocalories per kilogram of fat-free mass per day (kcal/kg FFM/day; 125 kilojoules per kilogram of fat-free mass per day) (Mountjoy et al, 2018). LEA can occur via two pathways; either as a direct result of insufficient dietary intake to support the vital functions of the human body (after removing the energy cost of exercise relative to metabolically active fat-free mass) (Mountjoy et al, 2018), or when exercise alone is increased without an appropriate increase in food consumption (Woods et al., 2017). Optimal energy availability (OEA) is estimated as 45kcal/kg FFM/day (188kJ/kg; 45 kcal/kg FFM/day or 188 kJ/kg FFM/day) and is suggested to provide appropriate energy availability to thrive, even in the presence of exercise engagement (Loucks & Heath, 1994; Loucks & Thuma, 2003). It is important to note that the detrimental effects of LEA can occur irrespective of body size and shape (Gaudiani, 2018). These effects are outlined in detail throughout the section to highlight the detrimental outcomes of LEA on both health and performance outcomes.

Energy availability explained:

Energy Availability (EA) = [Energy intake (EI) (kcal) - Exercise Energy Expenditure (EEE) (kcal)] / fat-free mass (FFM) (kg)

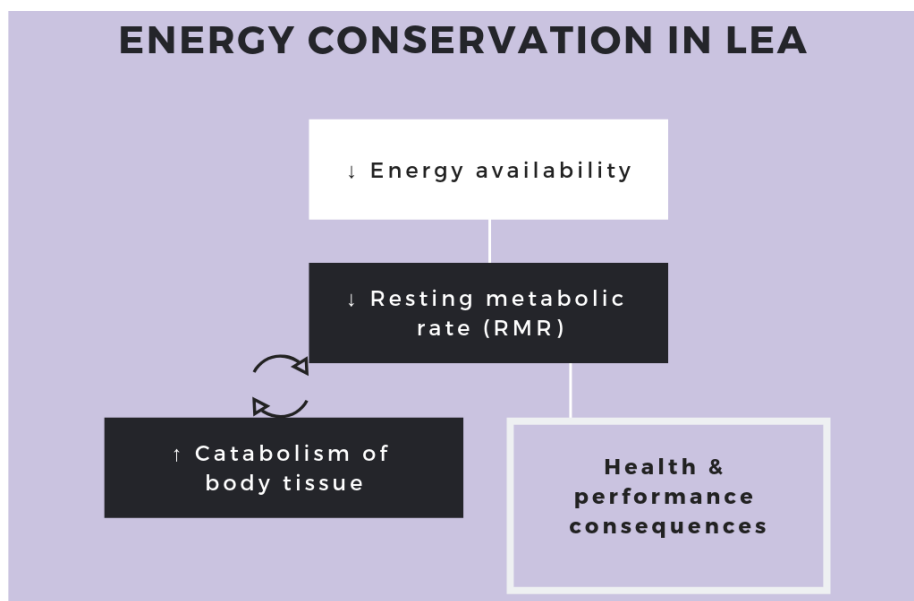
WHERE:

- **Optimal EA (OEA):** 45 kcal/kg FFM/day (188 kJ/kg FFM/day)
- **Low EA (LEA):** 30 kcal/kg FFM/day (125 kJ/kg FFM/day)

LEA can occur irrespective of body size and shape.

Box 2. Energy availability explained

LEA impacts the body's ability to engage in physical activity by hindering its ability to adequately store glycogen and synthesize protein (Tarnopolsky et al., 2001). A further consequence of LEA is that the body may adapt to the LEA state by decreasing both the absolute and relative resting metabolic rates (RMR), in an effort to protect the unfavourable breakdown of fat free mass (FFM), such as muscles, bones and organs (Woods et al., 2017). This reduction in RMR due to LEA can negatively impact both health and performance, as noted above (Mountjoy et al., 2018). If energy adequacy (EA) is extremely low, the breakdown of metabolically-active FFM can result, leading to an even greater decrease in RMR via the loss of metabolically-active tissue (Mountjoy et al., 2018) In a state of LEA, the body will reduce its absolute and relative resting metabolic rate (RMR) to help conserve energy and to ultimately avoid the breakdown of its own tissue (Gaudiani, 2018; Woods et al, 2017). This reduction in RMR, as can be seen in Box 3, can negatively impact both health and performance outcomes, as outlined in Mountjoy and colleagues (2018). If energy availability (EA) becomes extremely low, also referred to as 'starvation' or 'famine', the body may ultimately be forced to initiate catabolism of its own metabolically-active tissue (NEDA, 2018). This breakdown of course causes an even greater decrease in RMR than originally experienced in a higher state of EA. Muscles, including the heart, are generally utilised as energy (NEDA, 2018) while many other negative consequences, such as psychological distress (Gaudiani, 2018), can simultaneously occur. A more detailed explanation of the effects of starvation can be found in the *Starvation* section, below.



Box 3. Energy conservation in a low energy availability state

Unfortunately, despite the consequences of inadequate EA, measuring it can prove challenging for many reasons (Mountjoy et al, 2018). For example, a standardised, reliable and valid measurement protocol for measuring EA does not yet exist, and measuring one's exact EA can require specialised equipment, an expert to operate the equipment and assess the results, and significant buy-in from the individual by keeping an extremely accurate food and exercise log (Mountjoy et al, 2018). Furthering the complexity of evaluating EA is that it may also change with training and/or competition phases (Mountjoy et al, 2018). Despite the difficulty in its measurement, not ensuring adequate EA contributes to many of the upcoming conditions that may contradict exercise, worsen performance and endanger health.

1. Cardiovascular health

Potential cardiovascular contraindications to exercise

The following conditions may contraindicate exercise engagement. These conditions may be applied throughout each section of 'Cardiovascular health'.

- Tachyarrhythmias with uncontrolled ventricular rates
- Uncontrolled cardiac arrhythmias with hemodynamic compromise
- “Noticeable changes in heart rhythm by palpation or auscultation”
- “Sustained ventricular tachycardia or other arrhythmia... that interferes with normal maintenance of cardiac output during exercise... (and) that (has) the potential to become more complex or to interfere with hemodynamic stability”
- “Uncorrected medical conditions, such as... important electrolyte imbalance...”

Sources: ACSM, 2018; Fletcher et al., 2013; Gibbons et al., 2002; McCallum et al., 2006; Mountjoy et al., 2018.

Modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favour improved energy and nutritional availability and the improvement of LEA-related cardiac consequences (Mountjoy et al., 2018).

Box 4. Potential cardiovascular contraindications to exercise

Bradycardia

Bradycardia is defined as a low resting heart rate (<60 beats per minute, where a normal resting heart rate occurs between 60-100bpm) (American Heart Association, 2015; American Heart Association, 2016) and is a subtype of arrhythmia (American Heart Association, 2016). This arrhythmia may be influenced by the effects of low energy availability (Spaulding-Barclay,

Stern & Mehler, 2016) and advanced malnutrition (Sachs, Harnke, Mehler & Krantz, 2016). Despite the relationship between bradycardia and LEA, bradycardia can present and remain irrespective of weight status (Spaulding-Barclay, Stern & Mehler, 2016). The exact physiological cause of bradycardia, as well as its influence on cardiac function in individuals with ED, is not yet clear and is an area of ongoing study (Mehler & Anderson, 2017). Symptoms of bradycardia may include fainting, dizziness, fatigue, confusion, shortness of breath, and difficulty tolerating exercise (American Heart Association, 2016). In extreme cases, bradycardia can result in cardiac arrest (American Heart Association, 2016), heart failure, fainting, chest pain and blood pressure issues (American Heart Association, 2016).

Bradycardia: Fitness or ED?

- Both athletes and individuals with ED commonly exhibit a ↓ HR at rest.
- However, upon minimal exertion (such as sitting to standing), an athlete's HR will barely increase and quickly return to normal.
- Conversely, upon minimal exertion, an individual with ED's HR may increase excessively and take extensive time to return to normal.

Box 5. Bradycardia: Fitness or ED?

Consequently, the presence of bradycardia should be considered carefully in the prescription of exercise. Gibbons and colleagues (2002) warn that sustained arrhythmia can interfere with normal maintenance of cardiac output when exercising and, in particular, that bradyarrhythmias with the potential to become more complex or to impede hemodynamic stability may contraindicate exercise. This is further supported by Fletcher and colleagues (2013) who remark that uncontrolled cardiac arrhythmias with hemodynamic compromise may also contraindicate exercise engagement. It should be noted that the American College of Sports Medicine (ACSM, 2018) are considerably more conservative, recommending that any noticeable change in heart rhythm by palpitation or auscultation may contraindicate exercise.

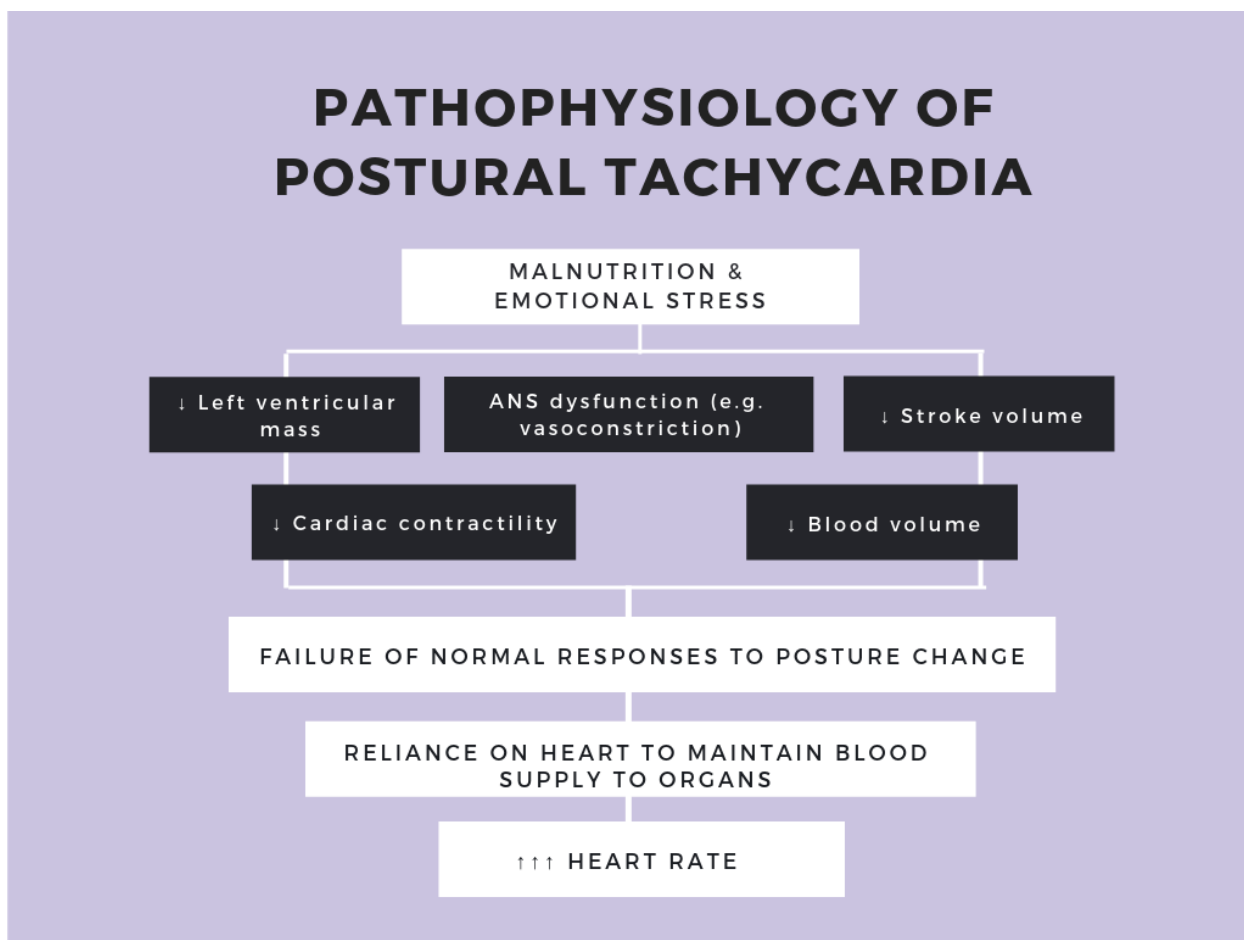
Tachycardia:

Tachycardia is defined as a high resting heart rate (>100bpm) and is a second subtype of arrhythmia (American Heart Association, 2016). It is suggested that individuals with AN who experience tachycardia are affected by more severe autonomic nervous dysfunction than those with bradycardia; consequently, exhibiting an increased likelihood of poorer long-term outcomes (Tokumura et al., 2012). Similar to bradycardia, tachycardia in ED may be associated with a state of LEA. Symptoms of tachycardia may include fainting, light-headedness, dizziness, palpitations, chest pressure, tightness or pain, shortness of breath and fatigue (American Heart Association, 2016). Tachycardia consists of a number of subtypes, some of which represent a natural response to situations such as emotional distress, which may not harm the body, while others signify abnormal and dangerous responses as a result of faults in the electrical system of the heart (American Heart Association, 2016). The latter subtypes can involve the heart beating faster than the chambers can replenish with blood, ultimately compromising vital blood flow to the rest of the body and potentially death (American Heart Association, 2016). Other serious consequences of tachycardia can include a life-threatening cardiac arrest (American Heart Association, 2016), heart attack (American Heart Association, 2016). With consideration of these consequences, it is important to carefully investigate tachycardia prior to any exercise engagement.

Postural Tachycardia

Postural tachycardia (PT) can present as an excessive heart rate increase of at least 30bpm within the first 10 minutes of standing or upright tilt from lying (Grubb, Kanjwal & Kosinski, 2006; Grubb & Karabin, 2008; Mehler & Andersen, 2017). Typically, upright posture changes include the tightening of the peripheral blood vessels and an increase in heart contractility (Grubb & Karabin, 2008); however, when these changes do not occur blood is left to pool in the peripheral parts of the body while upright, causing the heart to beat at an abnormally fast rate in an effort to return sufficient blood back to the brain (Grubb et al, 2006) (Box 6). While this compensatory effort is crucial to ensure oxygenation supply to the brain remains constant (Grubb et al., 2006), PT, particularly in the presence of excess, can lead to minor CNS symptoms or in some cases sudden cardiac death. PT can arise as a result of a range of physiological issues, including malnutrition (Gaudiani, 2018), autonomic nervous dysfunction

(Jacob et al., 2000), decreased heart muscle mass (particularly the left ventricle), decreased stroke volume, decreased blood volume (Fu et al., 2010) and emotional stress (Sheldon et al., 2015).



Box 6. Pathophysiology of postural tachycardia

Symptoms of PT may include palpitations, light-headedness, visual blurring or tunnel vision, tremors, weakness, fatigue, exercise intolerance, hyperventilation, shortness of breath, anxiety, chest pain, nausea, peripheral coldness or pain, concentration issues and headaches (Freeman et al., 2011). Furthermore, a complete loss of consciousness can ultimately occur if too much blood remains pooled in the extremities, as the brain is starved of oxygen and nutrients (Grubb & Karabin, 2008). It should be noted that while PT can mimic the cardiac symptoms of Postural Orthostatic Tachycardic Syndrome (POTS); however, POTS should not be diagnosed in the presence of malnutrition (Gaudiani, 2018).

It is evident that PT presents as a response to abnormal physiological or psychological influences whereby even a small postural change can cause the heart to overwork and symptoms to present (Freeman et al., 2011; Grubb & Karabin, 2008; Grubb et al., 2006).

Orthostatic Hypotension

Orthostatic hypotension (OH) is defined as a sustained drop in systolic blood pressure of ≥ 20 mmHg or diastolic pressure of ≥ 10 mmHg after three minutes of standing or head up tilt from 60° on a tilt table, from either a sitting or lying position (Freeman et al., 2011; Lanier, Mote & Clay, 2011). Similar to PT, OH manifests as a result of inadequate cardiovascular and neural responses to standing, which allows gravity to pool blood in the lower extremities and the abdominal organs (Freeman et al., 2011). A sustained drop in blood pressure indicates the blood is not exerting the necessary amount of pressure on the artery walls, preventing the brain and other organs from receiving adequate oxygen and nutrients (Grubb & Karabin, 2008; National Heart, Lung and Blood Institute, n.d.). Symptoms of OH may include fainting, palpitations, dizziness, lightheadedness, blurred vision, weakness, fatigue, nausea, headaches, shortness of breath, and pain in the chest, shoulder or neck (Lanier, Mote & Clay, 2011). Although OH and PT can present with similar symptoms, OH is caused by orthostatic stress only, whereas PT can be caused by both orthostatic and emotional stress (Sheldon et al, 2015).

Hypotension

Hypotension is defined as a low blood pressure ($< 90/60$ mmHg; National Heart, Lung and Blood Institute, n.d.). The presence of a low blood pressure indicates the blood is not exerting the necessary amount of pressure on the artery walls (National Heart, Lung and Blood Institute, n.d.). Because a normal pressure is required to supply the vital organs with sufficient oxygen and nutrients to work and survive, a low blood pressure can decrease this availability and, therefore, compromise vital supply to the organs (National Heart, Lung and Blood Institute, n.d.). Low blood pressure in individuals with ED can occur as a result of starvation, blood volume depletion due to insufficient food and fluid intake, as well as due to vomiting and laxatives (McCallum et al., 2006). A low blood pressure in individuals with ED can also occur as a result of autonomic dysfunction due to the disturbance of key hormones and other mechanisms regulated by the central nervous system (Oswiecimska et al, 2007). It should also be noted that even mild

dehydration (1-2% of body weight) can cause an individual to become symptomatic (American Heart Association, 2016). Symptoms of hypotension may include dizziness, light-headedness, nausea, fainting, dehydration, lack of concentration, blurred vision, cold, clammy and pale skin, rapid and shallow breathing, fatigue and depression (American Heart Association, 2016). It has been suggested to modify exercise engagement until hypotension has resolved (McCallum et al., 2006).

Prolonged QTc interval

Electrocardiograph (ECG or EKG) abnormalities can identify electrical and structural abnormalities of the heart (American Heart Association, 2015). One reading provided by an ECG is the QT interval, which is the time calculated from the beginning of the Q wave to the end of the T wave (Garson, 1993). The QT interval is an important component of the heart's electrical activity and represents part of the vital contraction and relaxation process (Garson, 1993). If the QT interval is prolonged (such as in the presence of low potassium, calcium and magnesium) it can cause a life-threatening ventricular arrhythmia called torsade de pointes, which can ultimately result in ventricular fibrillation, preventing the heart from pumping vital blood supply to the body (American Heart Association, 2016; Mehler & Andersen, 2017).

The QTc interval, more specifically, is a measure of the same time interval as the QT, however, has been corrected to account for heart rate (Drew et al, 2010). The QTc is measured most accurately by the Fridericia and Framingham formulae (Vandenberk et al, 2016). Abnormal QTc prolongation is defined as >460ms in women and >450ms in men (Rautaharju, Surawicz & Gettes, 2009). Severe QTc prolongation is seen in durations greater than 480ms and can lead to serious ventricular arrhythmias (Mehler & Andersen, 2017). Although QTc prolongation is an important and common consideration in AN, it may not be an exclusive marker of AN itself (Krantz et al, 2012). Rather, QTc prolongation may be associated with independent and reversible factors such as low potassium, psychotropic medications (Krantz et al, 2012) and hypoglycaemia (Laitinen et al, 2008).

Arrhythmias

The term *arrhythmia* refers to any change from the normal sequence of electrical impulses within the heart which therefore causes an abnormal heartbeat rhythm (American Heart

Association, 2016). Unusual electrical impulses may cause the heart to beat too fast (also known as tachycardia), too slow (bradycardia), or irregularly (fibrillation) (American Heart Association, 2016). Arrhythmias can prevent the vital organs from receiving normal, steady amounts of blood which can cause organ damage or failure (American Heart Association, 2016). Arrhythmias in ED may occur as a result of effects of LEA (Spaulding-Barclay, Stern & Mehler, 2016) and advanced malnutrition (Sachs, Harnke, Mehler & Krantz, 2016), whereby a mismatch between dietary intake, vital function and exercise occurs relative to metabolically active fat-free mass (Mountjoy et al, 2018). Importantly, arrhythmias can present and remain irrespective of weight status (Spaulding-Barclay, Stern & Mehler, 2016). Symptoms of arrhythmia may include fatigue, weakness, dizziness, light-headedness, fainting or near fainting, rapid heartbeat, shortness of breath, anxiety, chest pain or pressure, and in extreme cases, collapse and cardiac arrest (American Heart Association, 2014).

2. Electrolyte abnormalities

Electrolytes

Electrolytes:

- Electrically-charged particles or ions within the body
- Key regulators of fluid, nutrients and waste products
- Commonly depleted with purging and in sweat
- Detrimentally, both electrolytes and water are often lost in unison.

Box 7. Electrolyte facts

Electrolytes, or electrically-charged particles or ions, are key regulators of the exchange of fluids, nutrients and waste products inside the body (McArdle, Katch & Katch, 2015, pp. 67). Electrolytes can be lost in sweat during exercise and are regularly affected in purging-related eating disorders (Lindsay, 2017; Mehler, 2001; Mehler & Andersen, 2017). Although we will review a range of electrolyte-specific symptoms in upcoming paragraphs, it should be mentioned that more severe symptoms can include rhabdomyolysis (the breakdown of muscle with

strenuous exercise), significant cardiac arrhythmias and sudden death (Mehler & Andersen, 2017).

Some of the most common electrolytes affected in ED are potassium, sodium and bicarbonate, with symptoms often presenting as dizziness, weakness, fatigue, constipation, muscle pain, abnormal skin sensations and depression (Mehler & Andersen, 2017). Importantly, these negative health and performance consequences also present in individuals engaging in subclinical rates of purging (Raj, Keane-Miller, & Golden, 2012). In fact, a history of purging may be a more sensitive indicator of complications than lifetime BN diagnosis (Raj et al., 2012).

Potential electrolyte contraindications to exercise

The following conditions may contraindicate exercise engagement. These conditions may be applied throughout each section of 'Electrolyte Abnormalities'.

- Electrolyte imbalance and/or a hypohydrated state

Sources: Fletcher et al., 2013 and Thomas, Erdman & Burke, 2016.

Modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favour improved energy and nutritional availability and the improvement of LEA-related electrolyte consequences.

Box 8. Potential electrolyte contraindications to exercise

Hypokalemia

Hypokalemia is defined as a serum potassium level below 3.6 mmol/L, which can be caused by purging in ED (Gaudiani, 2018; Mehler & Andersen, 2017). Potassium is a key electrolyte involved in normal cellular function, especially in muscles and nerves (Stone, Martyn & Weaver, 2016). The heart muscle is particularly sensitive to changes in potassium concentration (American Heart Association, 2005). Symptoms of hypokalemia may include muscle weakness, cramping, abnormal skin sensations, constipation, heart palpitations, fatigue, respiratory difficulty and paralysis (American Heart Association, 2005; Mehler & Andersen, 2017). Severe hypokalemia (<2.5mmol/L) can cause rhabdomyolysis, significant cardiac arrhythmias and sudden death (Mehler & Andersen, 2017). ECG changes due to hypokalemia

can include “flat or inverted T waves, ST segment depression, and prominent U waves, which can exceed the amplitude of the T waves” (Mehler & Andersen, 2017).

Hypophosphatemia

Hypophosphatemia is defined as a serum phosphate level below 0.8mmol/L (2.5 mg/dL) (Agency for Clinical Innovation, 2017). Causes of low phosphate may include, but are not limited to, refeeding, malnutrition, chronic diarrhea, and the chronic use of diuretics and alcohol (Agency for Clinical Innovation, 2017). Symptoms of low phosphate can include muscular dysfunction and weakness, which can affect the heart, eye, respiratory and throat muscles, abnormal blood-oxygen levels, and irregular heartbeats (Agency for Clinical Innovation, 2017). Furthermore, hypophosphatemia may induce a state of confusion and delirium, anaemia and an increased severity of infections (Agency for Clinical Innovation, 2017). Lastly, rhabdomyolysis, coma and even death may result (Agency for Clinical Innovation, 2017).

Hypomagnesemia

Hypomagnesemia, or low serum levels of magnesium (<0.7mmol/L) can present as a result of diarrhea, the use of diuretics, hormone dysfunction, alcohol intake and some medications (American Heart Association, 2005). Low levels of magnesium can cause disturbances to other vital hormones and electrolytes, and can cause muscle tremors and fasciculations, tetany, vertigo, eye problems, altered mental state, respiratory compromise, cardiac arrhythmias, ataxia, seizures, dysphagia and sudden cardiac death (American Heart Association, 2005; Mehler & Andersen, 2017).

Hypercarbia

Hypercarbia, also known as metabolic alkalosis, is one of the most common clinical abnormalities in people with ED (Mehler & Andersen, 2017). Similar to other electrolyte disturbances, it can be caused by purging, and is defined as a higher-than-normal serum bicarbonate level (30-40mmol/L; Mehler & Andersen, 2017). Usually, metabolic alkalosis is asymptomatic, however, when bicarbonate increases above 35mmol/L, short and shallow breaths may present contributing to respiratory complications (Mehler & Andersen, 2017). Indicators of abnormal respiration, such as shortness of breath and signs of poor perfusion including light-

headedness, cyanosis, confusion, nausea, ataxia, pallor or cold and clammy skin (American College of Sports Medicine, 2018, pp. 84) may contraindicate exercise engagement.

Hyponatremia

Hyponatremia, or a low concentration of serum sodium, can present as mild to moderate, 120-130mmol/L, or severe, <120mmol/L (Mehler & Andersen, 2017). Hyponatremia can prevail in response to two main physiological disturbances. Firstly, a decreased concentration of serum sodium may present during a low volume status, or a state where the body is left with lower than required levels of salt and water due to behaviours such as purging or responses such as excessive sweating (Gaudiani, 2018; Mehler & Andersen, 2017). Secondly, a low serum sodium concentration may occur when the body is holding too much water as a result of malnutrition, (which prevents the kidneys from working normally to excrete unneeded water in urine, leading to an excessive water build-up inside the body), or due to extreme water intake (Gaudiani, 2018; Mehler & Andersen, 2017). Symptoms of hyponatremia can range from less severe, including nausea, confusion, headaches and vomiting to more severe, including delirium, impaired consciousness, seizures, and, in some cases, cardiorespiratory arrest (Henry, 2015). Considering these risks, it may be reasonable to suggest exercise in some individuals with hyponatremia may be contraindicated or require modification.

1. Sex Hormones and Relative Energy Deficiency in Sport

Relative Energy Deficiency in Sport

The term “Relative Energy Deficiency in Sport” (RED-S) has in recent years replaced the less inclusive “Female Athlete Triad” (Mountjoy et al, 2014). RED-S is defined by the International Olympic Committee Consensus group as, “a syndrome resulting from relative energy deficiency that affects many aspects of physiological function including metabolic rate, menstrual function, bone health, immunity, protein synthesis, cardiovascular and psychological health” (Mountjoy et al, 2014, pp. 1). RED-S differs to the Female Athlete Triad as it: (a) acknowledges a state of low energy availability can be experienced by both males and female athletes, as well as athletes prior to puberty and; (b) highlights that this phenomenon extends beyond the three components of menstrual function, energy availability, and bone health” (Mountjoy et al, 2014).

Health consequences of RED-S may affect the cardiovascular, gastrointestinal, immunological, endocrine, haematological and psychological systems, and can also impact menstrual function, bone formation, and general growth and development (Mountjoy et al, 2018). Performance consequences of RED-S can include a decline in training responses, coordination, concentration, glycogen stores, muscle strength, and endurance performance, as well as impaired sporting judgement, irritability, depression and increased injury risk (Mountjoy et al, 2018).

Gaudiani (2018) suggests that instead of commonly and detrimentally restricting food intake in an effort to enhance exercise performance, exercising individuals should instead strive for adequate hydration, sleep, recovery, energy intake and emotional health. It is suggested that decreasing or modifying exercise, both with and/or without increased caloric and nutritional consumption, may favour improved energy availability (Gordon et al, 2017). It is expected that with increased EA, the health and performance of RED-S may begin to diminish.

Potential contraindications and modifications to exercise in individuals with RED-S are listed throughout this document and pertain to the cardiovascular system, electrolytes, sex hormones, body composition, psychological and behavioural states and other outcomes of ED. Mountjoy and colleagues (2018) suggest that modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favour improved energy and nutritional availability and the improvement of LEA-related consequences.

Amenorrhea

Amenorrhea is defined as the absence of menstrual cycles for more than 90 days (Nattiv et al., 2007). One subtype of amenorrhea, functional hypothalamic amenorrhea (FHA), can occur as a result of metabolic stress, such as low EA, or psychological stress imposed on the body (Berga & Loucks, 2006) (Box 9). It has been suggested that women with FHA may commonly

Amenorrhea:

- Menstrual cycle interval persistently >45 days and/or amenorrhea is present for ≥ 90 days (*regardless* of weight change or athlete / non-athlete).

Caused by:

- **Metabolic stress** (LEA of ≤ 30 kcal/kg FFM/day (≤ 125 kJ/kg FFM/day) (*subclinical presentations can occur both above and below this threshold)
- **Psychological stress**

Box 9: Functional hypothalamic amenorrhea: definition and causes

display, “more dysfunctional attitudes, have greater difficulty in coping with daily stresses, and tend to have more interpersonal dependence than do eumenorrhic women” (Gordon et al, 2018, pp. 1427). Irrespective of the stressor causing the FHA, an increased release of stress hormone, cortisol, and a decreased release of sex hormones, including gonadotropin-releasing hormone (GnRH), luteinising hormone (LH), oestrogen, and follicle stimulating hormone (FSH), amongst others can occur (Berga & Loucks, 2006; Silveira, MacColl, & Bouloux, 2002). The altered release of both stress and sex hormones disrupts menstrual homeostasis, ultimately inducing menstrual dysfunction (Berga & Loucks, 2006).

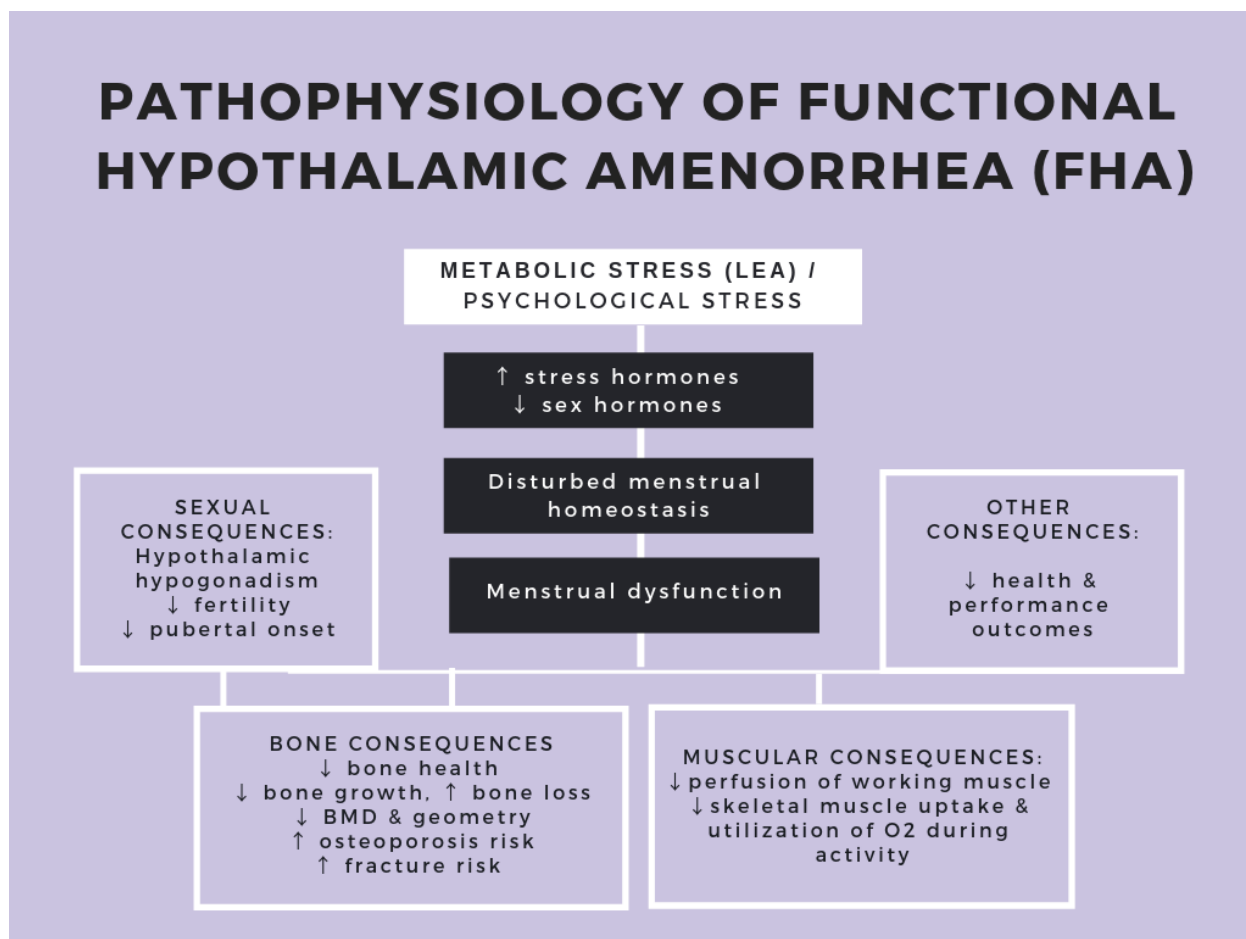
FHA must only be diagnosed when organic or anatomic issues causing the amenorrhea have been excluded and when the menstrual cycle interval persistently exceeds 45 days and/or when amenorrhea is present for 90 days or more (Gordon et al, 2017). The complexity of hormonal changes in a LEA state provide an ongoing area of research (Mountjoy et al, 2018).

Amongst its various consequences, such as the delay and stunting of important growth during puberty (Gordon et al, 2017), infertility, decreased perfusion of working muscle, and decreased skeletal muscle oxygen uptake and utilization during activity, FHA can also significantly endanger skeletal health (Gordon et al, 2017; Nativ et al, 2007). It has been identified that LEA in FHA can cause a decrease in the presence of oestrogen, which usually maintains bone health by promoting calcium absorption, reducing calcium excretion, decreasing the rate of bone break-down and overall, protecting the uncoupling of bone (De Souza, 2019; McArdle et al., 2010). Low levels of oestrogen can indicate a state of hypothalamic hypogonadism, a clinical syndrome which occurs as a result of abnormal pituitary gonadotropin levels, regardless of body weight (Silveria et al., 2002). The presence of both menses and normal endogenous oestrogen have been found to be crucial to supporting a healthy bone mineral density (BMD; Karlsson, Weigall, Duan, & Seeman, 2000).

The essential role of oestrogen in protecting bone health is thought to be so strong, that the state of hypoestrogenism in the presence of LEA may negate the usual positive effects of weight-bearing exercise at the spine in athletes with menstrual dysfunction (Kandemir et al, 2018). Indeed, Ackerman et al (2015) found that oligomenorrhic athletes were not only unable

to experience the usual benefits of weight-bearing exercise on bone mineral density, but also reported a significantly higher occurrence of stress fractures when compared with both eumenorrheic athletes and non-athletes. Amenorrhea can increase the relative risk of stress fractures in both female athletes by two- to four-fold, when compared with athletes with regular menses (Nattiv et al., 2007).

Furthermore, a history of menstrual disturbances (including infrequent, light or absent menses) between menarche and young adulthood can also increase the likelihood of developing decreased BMD and geometry, even with regular exercise engagement (Mallinson et al, 2016). The peak formation of bone in healthy females occurs during a short window in adolescence; however, if this critical period is interrupted by menstrual dysfunction, this lifetime bone supply cannot be properly developed, potentially causing irreversible osteoporosis and a serious risk of repeated fractures (Mallinson et al, 2016). It is important to note that BMD will continue to decline as the number of missed menstrual cycles accumulates as cumulative menstrual dysfunction is a significant predictor of poor bone mass (Drinkwater, Bruemner, & Chesnut, 1990; Mallinson et al, 2016). Importantly, non-athlete exercisers also experience detrimental consequences of LEA and menstrual dysfunction, suggesting they are not exempt from the issues arising from a hypothalamic hypogonadal state (Torsveit & Sundgot-Borgen, 2005). BMD and geometry may be compromised most when accompanied by a low BMI, late menarche or when amenorrhea persists for several years or more (Mallinson et al, 2016).



Box 10. Pathophysiology of functional hypothalamic amenorrhea

One study, by Reed et al (2015), demonstrated that an EA threshold below 30 kcal/kg FFM/day (125 kJ/kg FFM/day) clearly discriminates women who are menstruating compared to amenorrhoeic; however, that this threshold does not discriminate subclinical presentations (such as oligomenorrhea or other functional menstrual abnormalities). Indeed, subclinical presentations can occur both above and below 30 kcal/kg FFM/day (125 kJ/kg FFM/day) (Reed et al, 2015). Despite this, Lieberman and colleagues (2018) found that EA this level increases the risk of developing menstrual dysfunction increased by over 50%. Further, this study found that for each unit decrease in EA, menstrual dysfunction increases by 9%, suggesting a linear relationship between EA and menstrual dysfunction is likely to occur (Lieberman et al, 2018).

It is important to note that FHA can still occur even if no changes in body weight are present and that growing adolescents require a higher EA than older women to continue normal menstrual function (Gordon et al, 2017). It has been suggested that normal menstrual function

may return with six to twelve months of weight stabilisation in combination with psychological support; however, it is also documented that the longer the amenorrhea has been present, the longer the time it may take to reverse (Gordon et al, 2018). For some, regular menses may never resume (Gordon et al, 2018).

It has been recommended that the most effective method of preventing adverse hormonal effects of physical activity is to increase EA to above 30kcal/kg of lean body mass per day, particularly in athletes (Melin et al., 2015). Indeed, if an individual is amenorrhoeic and unable to engage in a nutrition plan, this may contraindicate exercise due to bone and fertility implications (McArdle et al., 2010; Warren & Perlroth, 2001). Further, high impact exercise, such as jumping, as well as running without normalised sex hormones or resumption of menses may negatively impact long term bone health and would likely, thus, be contraindicated (Schied et al., 2011; Zanker et al., 1998). Finally, failing to modify exercise in the presence of FHA may worsen existing hormonal and energy disruptions, which could further contribute to menstrual dysfunction and its' consequences (Gordon et al, 2017; Nattiv et al, 2007).

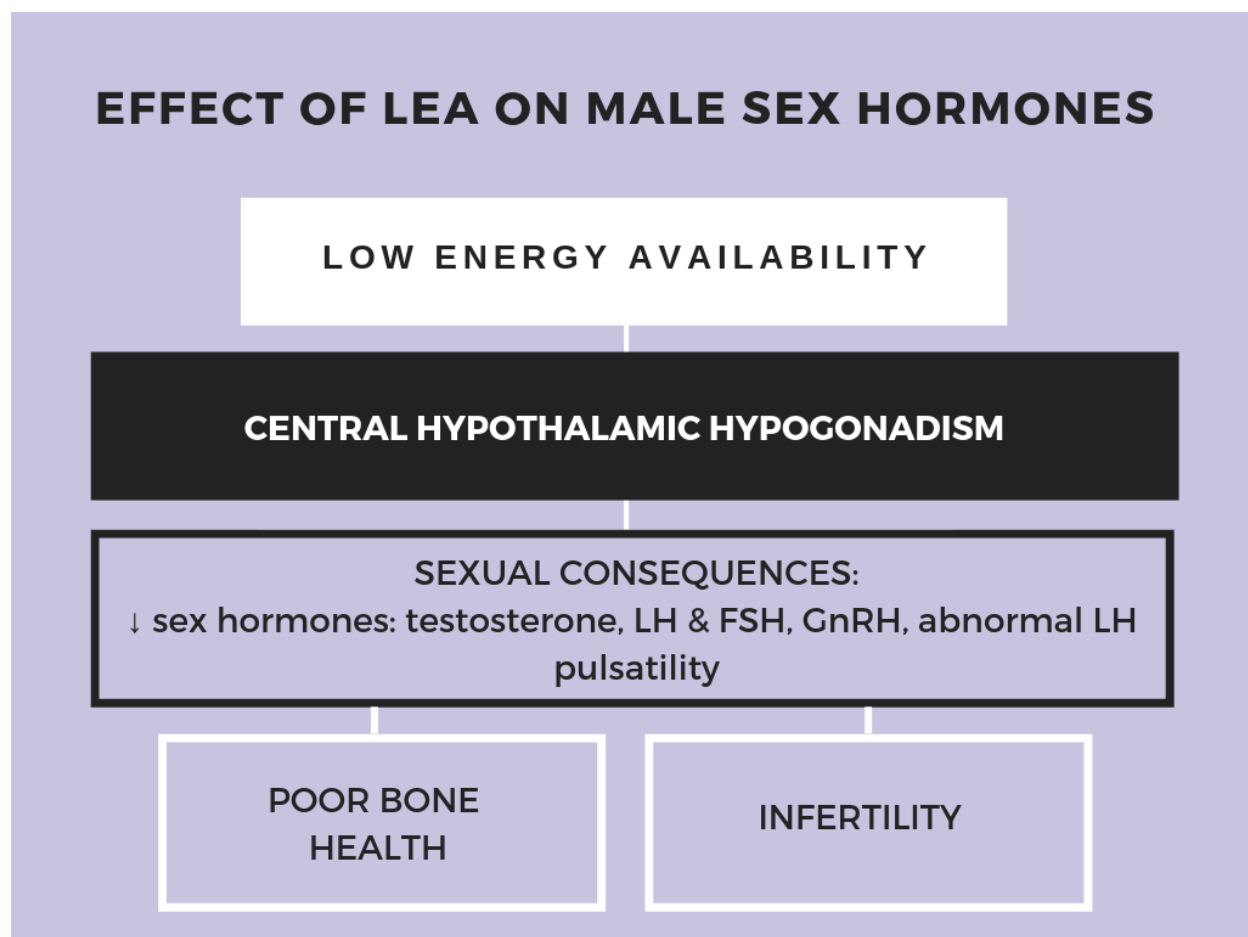
Female sex hormones

Female sex hormones, such as GnRH, LH oestrogen and FSH, amongst others can be associated with menstrual dysfunction, poor bone health, the stunting of important growth during puberty, infertility, and suboptimal muscular performance (Berga & Loucks, 2006; Gordon et al, 2017; Kandemir et al, 2018; Nattiv et al, 2007; Silveira et al., 2006). Although it has been found that improving EA and a trend of weight gain in underweight individuals can positively alter metabolic hormone profiles in a matter of days to weeks, exercise in the presence of abnormal hormone profiles must still be prescribed cautiously (Moutjoy et al., 2014). It has been suggested that the most effective method of preventing adverse hormonal effects of physical activity is to increase energy availability to above 30kcal/kg of lean body mass per day, particularly in athletes (Melin et al., 2015).

Male sex hormones

Sex hormones in males, especially in male athletes, can become negatively affected as a result of starvation-induced central hypothalamic hypogonadism (Mehler & Brown, 2015). Low levels of sex hormones, including testosterone, LH, FSH, GnRH, as well as abnormal LH

pulsatility, can cause impaired reproductive function, decreased immunity and impaired bone health (Mehler & Brown, 2015; Mountjoy et al., 2014) (Box 11).



Box 11. Effect of low energy availability on male sex hormones

4. Body Composition

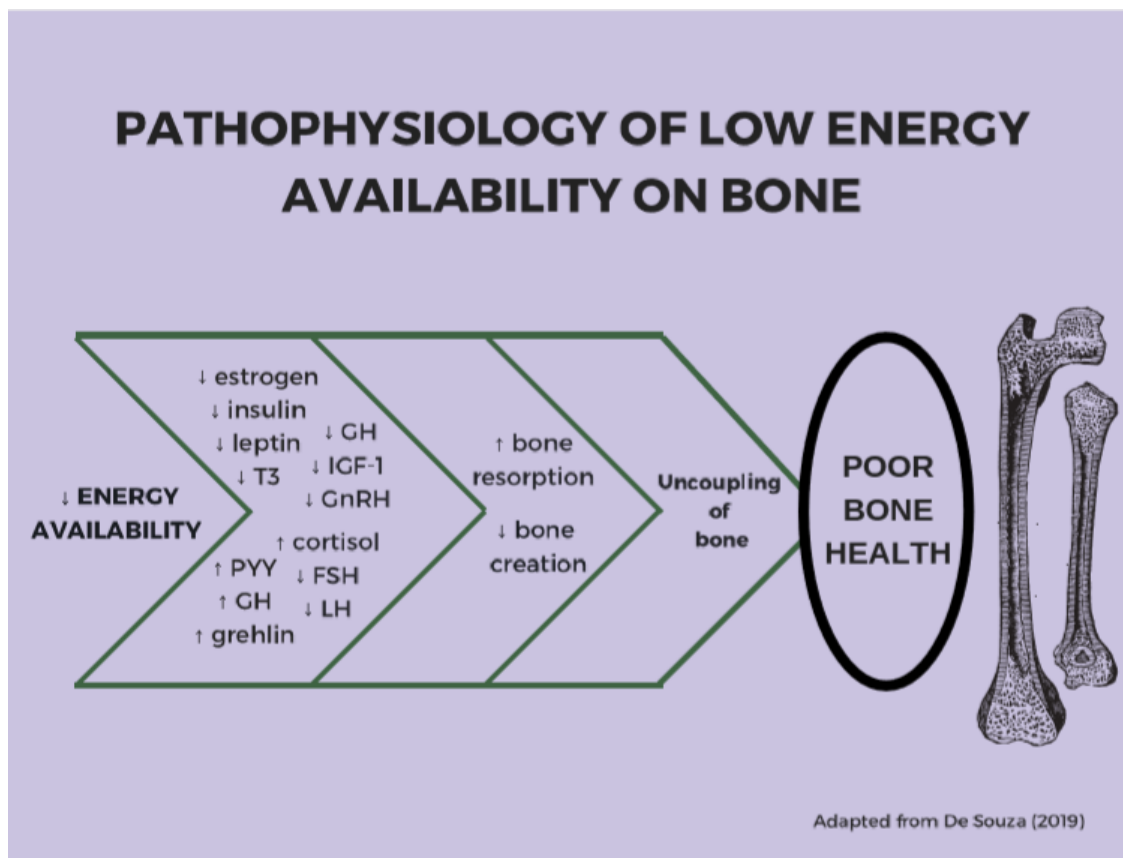
Low Bone Mineral Density (BMD)

A low BMD is defined by a Z-score less than -1.0 (ACSM, 1997; Leib, Lewiecki, Binkley, & Hamdy, 2004). A low BMD can be caused by a larger amount of bone breakdown than creation due to the negative effects of LEA (Nattiv et al., 2007). Importantly, a low BMD can occur in both males and females, with males with AN found to display greater bone loss at the lumbar spine than in women (Mehler, Sabel, Watson and Andersen, 2008). Further adding to this concerning data is that the duration of AN in males was shorter than in females, suggesting bone health may decline faster in men than in women (Mehler et al., 2008). Contrastingly, some

women with LEA and amenorrhea do not display any bone concerns when tested, as pathogenesis has not yet translated to bone (De Souza, 2019). However, once this bone damage has occurred it can still remain for additional months to years following adequate energy availability and recovered menses and in some cases may never fully recover (De Souza, 2019; Warren et al, 2002).

Consequences of poor bone health can include the complete inability to engage in exercise and other daily activities, pain and disability, permanent rounding of the upper back due to micro fractures (kyphosis), the requirement of a walker aid, and the use of pain medications which can cause dependency and constipation, amongst other negative sequelae (Gaudiani, 2018).

A low BMD can occur when a chronic energy deficiency or LEA triggers physiological adaptations to conserve energy (De Souza, 2019) (Box 12). Adaptations may include a reduction in oestrogen, which is involved in the disruption or elimination of reproductive function to conserve energy, as well as the suppression of a range of metabolic hormones (De Souza, 2019). Metabolic hormones, such as fullness hormone leptin, thyroid hormone triiodothyronine (T3), glucose-regulator insulin, and anabolic growth hormone (GH) and insulin-like growth factor 1 (IGF-1), are decreased in a LEA state (De Souza, 2019). Other metabolic hormones, such as hunger hormone ghrelin, fullness hormone peptide tyrosine tyrosine (PYY), stress hormone cortisol, and growth hormone (GH) are increased in a LEA state (De Souza, 2019). Further, a LEA state can decrease the release of sex hormones GnRH, LH and FSH, in combination with other hormones mentioned above, to disrupt menstrual homeostasis and ultimately induce menstrual dysfunction, which can endanger skeletal health (Berga & Loucks, 2006; Gordon et al, 2017). These hormonal changes resulting from LEA causes the uncoupling of bone turnover, whereby resultant low levels of oestrogen allows increased bone resorption and the changes in metabolic hormones cause decreased bone formation (De Souza, 2019). The ultimate result of LEA and menstrual dysfunction is dangerously low bone strength and a resultant high risk of fractures (De Souza, 2019). BMD and bone mineral geometry may be compromised most when accompanied by a low BMI, late menarche, or when amenorrhea persists for several years or more (Mallinson et al, 2016).



Box 12. Pathophysiology of low energy availability on bone

Energy deficiency prior to puberty is particularly damaging to bone as vital bone growth can be stunted and important sexual development, contributing to bone growth, can be delayed (Schneider et al, 2000). Further, menstrual disturbances between menarche and young adulthood increases the likelihood of developing a low BMD and geometry, even with regular exercise engagement, which would normally encourage bone growth (Mallinson et al, 2016). It has been suggested that a state of hypoestrogenism negates the usual positive effects of weight-bearing exercise at the spine in oligomenorrhic athletes (Kandemir et al, 2018). The peak lay down of bone, contributing to BMD and bone content, in healthy females has been shown to occur for a period of three years (between 11-14 years of age) and quickly falls after 16 years and/or two years post-menarche (Theintz et al, 1992). In males, this lay down occurs for a duration of four years (between the ages of 13-17 years) and continues at a slower rate from 17-20 years (Theintz et al, 1992). If bone accrual is interrupted in adolescence due to menstrual dysfunction or inadequate sex hormones, it is therefore difficult to recover the lost bone in early adulthood and

beyond, potentially initiating a lifetime consequence of osteoporosis and fractures (Mallinson et al, 2016). It is important to note that BMD will continue to decline as the number of missed menstrual cycles accumulates, as cumulative menstrual dysfunction is a significant predictor of poor bone mass (Drinkwater, Bruemner, & Chesnut, 1990; Mallinson et al, 2016).

Amenorrheic female athletes have been observed to possess significantly lower hip and lumbar spine bone mineral density when compared with eumenorrheic athletes (Ackerman et al, 2012). Evidently, young female athletes with oligomenorrhea are at a higher risk of experiencing stress fractures than young female athletes with normal menstrual function and non-athletes with normal menstrual function (Ackerman et al, 2015). Ackerman and colleagues (2015) demonstrated the oligomenorrheic athletes were simultaneously unable to experience the usual benefits of weight-bearing exercise on bone and increased their susceptibility to stress fractures (Ackerman et al, 2015). Indeed, athletes with amenorrhea have lower BMD and bone geometry than athletes with normal menstruation, particularly at the lumbar spine (Mallinson et al, 2016). Importantly, non-athlete exercisers also experience detrimental consequences of LEA and menstrual dysfunction (Torsveit & Sundgot-Borgen (2005).

Howgate and colleagues (2013) recommended that high impact activity may be contraindicated until a BMI of 16 is reached, due to the damaging effects of low BMI on bone growth. Furthermore, when symptoms such as LEA, amenorrhea, and low BMD are present in an exercising individual, they may be at an increased risk of bone stress injury, which poses the potential for a further delay in the ability to engage in exercise (Barack et al., 2014; Mountjoy et al., 2014). One study revealed athletes with oligomenorrhea, without a current ED, were significantly more likely to report a stress fracture (approximately one in four) when compared with individuals with AN not presently exercising (one in twenty) as well as eumenorrheic controls (1.2%); although individuals with AN were found to report higher rates of overall fracture (Kandemir et al, 2018).

Miller and colleagues (2006) found that the greatest increases in lumbar spine and hip bone density can occur with a simultaneous improvement in weight of >85% of ideal body weight (IBW) and/or by 10% of body weight (BW), as well as the resumption of menses (having had at least one menses in the past three months). Bone density remained consistent in women who gained weight only (~70% of these women did not resume menses with the weight gain); increased slightly in those who recovered menses only (only a small weight gain of <10% BW

occurred); and increased the most in those who recovered both menses and weight (Miller et al, 2006). The study also found that bone loss occurs rapidly (up to 3% per year) in young women with AN, but that with both weight gain and resumption of menses, hip BMD can recover at a rate of 2% per year and that with a resumption of menses alone spine BMD can recover by 3% per year (Miller et al, 2006). At present the recovery of bone geometry is not known (De Souza, 2019). Barack and colleagues (2014) recommend a combination of strategies to reverse or minimise low BMD including nutritional intervention, meeting both minimum weight requirements and/or weight gain goals, psychological counselling, and engaging in tailored exercise programs to achieve outcomes including, but not limited to, improved muscle strength (Barack et al., 2014).

It has been recommended, particularly in athletes, that the most effective prevention of hormonal effects of physical activity is to increase EA to >30kcal/kg of lean body mass per day (Melin et al., 2015). Indeed, failing to modify exercise in the presence of FHA may worsen existing hormonal and energy disruptions, which could further contribute to poor bone health outcomes (Gordon et al, 2017; Nattiv et al, 2007). It is suggested that if individual is amenorrhoeic and unable to engage in a nutrition plan, exercise may be contraindicated due to the potential negative bone implications (McArdle et al., 2010). Further, high impact exercise (such as jumping) in the absence of normalised sex hormones, as well as running without the resumption of menses may negatively influence long-term bone health (Schied et al., 2011; Zanker et al., 1998). Exercise modification may therefore be required in these circumstances.

Superior mesenteric artery syndrome

Superior mesenteric artery syndrome (SMA) is caused by the compression of the duodenum due to a diminished fat pad from weight loss that would usually separate the aorta and superior mesenteric artery (Mehler & Brown, 2015). Symptoms of SMA syndrome can include upper quadrant abdominal pain soon after eating, early satiety, nausea, and vomiting (Mehler & Brown, 2015). SMA is most commonly found in individuals with a low BMI and in children and can resolve with weight gain (Arthurs & Mehta, 2012; Shetty, Schmidt-Sommerfeld, Haymon, & Udall, 2000).

BMI and health status:

- BMI does not depict health status
- Some individual's weight is not affected at any stage of their ED
- This highlights the need to evaluate medical and psychological parameters of a person's health for appropriate information.

Box 13: BMI and health status

Body fat and weight factors

Unknown and widely varied responses can occur in individuals with a LEA (Gaudiani, 2018). For example, some people may possess a genetic makeup which protects against physical signs of malnutrition, whilst others may be more likely to develop more obvious symptoms under the same conditions; certainly, people may experience “different genetic sensitivities to environmental exposures” (Gaudiani, 2018, location 485).

Body Mass Index (BMI) is a ratio of weight to height and is determined by dividing one's weight in kilograms by meters squared (McKardle et al., 2010). BMI does not depict health status; indeed, some individual's weight is not affected at any stage of an ED, reflecting the need to evaluate medical and psychological parameters of a person's health (Gaudiani, 2018). Despite this argument, BMI has historically been used as a marker of medical status in people with eating disorders (for example, it has been suggested a BMI of less than 75% of median for age, sex and height may indicate the need for hospitalisation (Geller et al., 2012)).

With regards to exercise suitability and anthropometrics, it has recently been suggested that instead of utilizing a solitary marker of weight (which impacts BMI) to decide appropriate exercise engagement, a progression of weight recovery may be more appropriate (Cook et al., 2016; Noetel, Dawson, Hay, & Touyz, 2017; Quesnel et al., 2017). Further to this, the International Olympic Committee states a prolonged low body fat percentage (measured by DXA or anthropometry using The International Society for the Advancement of Kinanthropometry ISAK or non-ISAK approaches) is a moderate contraindication to return to sport, as is a weight loss of 5-10% in one month (Mountjoy et al, 2014). Research has also proposed that prior to engaging in regular aerobic activity or rigorous training, obtaining 90% of ideal body weight may be important (McCallum et al., 2006).

Despite the above suggestion of removing an exclusive marker of weight from deciding on care, engaging in exercise with a BMI $<13.5\text{kg/m}^2$ may be contraindicated due to the range of health and performance consequences from energy deficiency (Gaudiani, 2018; NEDA, 2018; Mountjoy et al, 2018), for example, increased risk of pericardial effusion (Docx et al., 2010). Further, it has been recommended to avoid high impact activity below a BMI of 16kg/m^2 (Howgate et al, 2013), potentially also due to low energy availability (Gaudiani, 2018; NEDA, 2018; Mountjoy et al, 2018). Further, it has been recommended to avoid high impact activity below a BMI of 16kg/m^2 (Howgate et al, 2013), potentially also due to the effects of low energy availability (Gaudiani, 2018; NEDA, 2018; Mountjoy et al, 2018).

5. Other related outcomes of ED

Temperature: Hypothermia and Hyperthermia

Hypothermia, or a low body temperature $<35^\circ\text{C}$ or $<95^\circ\text{F}$, can occur as a result of malnutrition and hypoglycaemia (Nishita, Ellenwood, & Rockewell, 1985). Importantly, hypothermia can still persist during and beyond weight restoration (Luck & Wakeling, 1982). The state of hypothermia can cause decreased psychological and cardiac functioning, muscle rigidity, unconsciousness and death and consequently may warrant exercise modification (Luck & Wakeling, 1982).

Conversely, hyperthermia, also known as a high body temperature $> 36^\circ\text{C}$ or $>96.8^\circ\text{F}$, can occur as a result of failed thermoregulation when the body produces or absorbs more heat than it can dispel through sweat (McArdle et al., 2010). Hyperthermia can result from serious medical complications including heat stroke, due to excessive metabolic heat from intense exercise, excessive environmental heat or humidity, or some medications, and severe reactions to drugs (McArdle et al., 2010). Similar to hypothermia, exercise may be contraindicated in a hyperthermic state.

Comorbid Illnesses

Engaging in exercise with an ED can result in multiple risks, as outlined throughout this section. However, the severity of this risk may be amplified when an individual presents with both an ED and a comorbid condition. For example, despite the original modifications made to exercise for certain ED-related presentations, exercise may be contraindicated whilst the individual is under the influence of alcohol or other drugs (Rhodes, Temple, & Tuokko, 2011).

Further, if an individual is living with diabetic retinopathy or severe nonproliferative diabetic retinopathy, certain exercise may be contraindicated (Grunberger, Taylor, Dons, & Gorden, 1983). Additionally, exercise may be contraindicated in the presence of peripheral edema (Beumont et al., 1993).

It is inevitable that some special populations will need further support and must be assessed by a medical team and, where accessible, an accredited exercise professional (see glossary pp.63) before recommending an appropriate supervised exercise plan. Please note that this does not preclude these populations from engaging in exercise; however, we encourage that adaptations to the SEES guideline for these populations must be done under the supervision of medical advice specific to their individual requirements. These populations may include (but are not limited to): Athletes, children/adolescents, and individuals with diabetes, osteopenia/osteoporosis, or other existing cardiorespiratory, vascular, metabolic, neurological, psychological or musculoskeletal complications. Finally, whilst purging as a behaviour has not been included as a contraindication to exercise, we encourage practitioners to ensure a thorough and frequent assessment for individuals engaged in vomiting, laxative, or diuretic use and exercise due to the compromising nature of these behaviours.

Blood Urea Nitrogen and Urine Specific Gravity

Urine specific gravity (USG) is a ratio of the mass of a solution compared to the mass of an equal volume of water and normally falls in the range of 1.010 to 1.030 (Shumann & Schweitzer., 1996). Low USG (1.001-1.003) may indicate the presence of diabetes or renal abnormalities as well as overhydration, while high USG can suggest the presence of adrenal, liver, heart or dehydration issues in individuals who are excessively sweating, vomiting or who have diarrhea (Shumann & Schweitzer, 1996).

Exercise does not affect specific gravity directly, however, it does influence a number of components, which will ultimately be reflected in the outcome of a USG test, such as hydration levels, and issues surrounding kidney and adrenal function (Shumann & Schweitzer, 1996). For example, blood urea nitrogen may be abnormal in an individual with an ED as a result of dehydration (Warren & Wiele, 1973). As such, exercise in an already dehydrated state may further increase in an individual's blood urea nitrogen. With this in mind, it has been recommended that strength training could be utilised for individuals with an ED, once a negative

nitrogen balance has been reversed and a trend of consistent weight recovery has been established amongst other reversals of various issues that can present with an ED and that can influence, or be influenced by, exercise engagement (McCallum et al., 2006).

Transaminase

When the liver is damaged as a result of low weight, refeeding, alcohol intake, infection of the liver and some medications enzymes called transaminases are released into the bloodstream (Gaudiani, 2018; Hanachie, Melchior, & Crenn, 2013; Oh & Hustead, 2011). Transaminase levels are often high in underweight persons, however, can become extremely high (above four- to thirty-times normal) with severe low weight (BMI <12) before refeeding (Hanachie, Melchior, & Crenn, 2013). Such high levels of transaminases may indicate vital organ failure (De Caprio et al., 2006). If liver enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are above three times higher than normal, a dangerous symptom of hypoglycaemia is likely to occur, regardless of BMI (Gaudiani, Sabel, Mascolo, & Mehler, 2012).

Causes of high levels of transaminases in the presence of ED may result from the body's break down of its own tissue or a reduction of blood flow through the organs, as a result of myocardial dysfunction (Brown & Mehler, 2015). Although transaminase does not primarily contribute to energy fuel for exercising muscle, there is a significant increase in the rate of amino acid catabolism during exercise (Henriksson, 1991). For example, a process occurs in which amino acid formation from the nonnitrogen-carrying organic compound pyruvate formed in metabolism is permitted, which is important to facilitate muscular work (McKardle et al., 2010). It can be concluded that exercising in the presence of already elevated transaminase levels may cause additional harm and hence exercise should be modified accordingly.

Hypoglycaemia

Hypoglycaemia is defined as a blood sugar level below 4mmol/L (McArdle et al., 2010). Dietary restriction, weight decline and excessive exercise can decrease the available levels of glycogen within the liver (Brown & Mehler, 2013). When stored glycogen levels are lower than normal, the liver's ability to create new glucose is disrupted affecting normal glucose metabolism and leads to ongoing low blood sugar levels (Brown & Mehler, 2013). Further, if

liver enzymes, aspartate aminotransferase (AST) and alanine aminotransferase (ALT), are elevated above three times their normal level, hypoglycaemia is likely to occur regardless of BMI (Gaudiani, Sabel, Mascolo, & Mehler, 2012).

The state of hypoglycemia can trigger a breakdown in both fat mass (FM) and FFM in an effort to provide the brain with fuel (Gaudiani, 2018). Many individuals with prolonged hypoglycaemia do not experience symptoms and have lost the ability to detect a hypoglycaemic 'low' (Gaudiani, 2018). If blood glucose falls below 3.3mmol/L, symptoms such as headaches, mental confusion, and coma may occur (McArdle et al., 2010). Severe hypoglycaemia can cause liver failure and sudden cardiac death; further, premeal hypoglycaemia indicates the potential for serious outcomes such as the risk of death and irreversible brain damage (Gaudiani, 2018; Rich, Caine, Findling, & Shaker, 1990).

If an individual engages in exercise in a hypoglycaemic state, the brain can be one of the most important organs to suffer (McArdle et al., 2010). Exercise can worsen hypoglycaemia and its effects due to the metabolic need for glucose during exercise (McArdle et al., 2010). Thus, exercise may be contraindicated for individuals in a hypoglycaemic state. To counter this situation, it is also suggested that modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favor improved energy and nutritional availability and the improvement of LEA-related consequences (Mountjoy et al, 2018).

6. Key psychological and behavioural factors

Starvation

In a state of LEA, which also includes dieting and 'cleansing,' the body will reduce its absolute and relative resting metabolic rate (RMR) to help conserve energy and to ultimately avoid the breakdown of its own tissue (Gaudiani, 2018; Woods et al, 2017). This reduction in RMR can negatively impact both health and performance outcomes, as outlined in Mountjoy and colleagues (2018). If EA becomes extremely low, also referred to as 'starvation' or 'famine', the body may ultimately be forced to initiate catabolism of its own metabolically-active tissue (NEDA, 2018). This breakdown contributes to an even greater decrease in RMR than originally experienced in a higher state of EA. Muscles, including the heart, are generally utilised as energy while many other negative consequences, including psychological distress can simultaneously occur (Gaudiani, 2018; NEDA, 2018). In a starved state, muscles are generally utilised as energy

first (NEDA, 2018). Not excluded in this break-down process is the heart muscle, which as a result becomes smaller and weakened, leading to a decreased heart rate and blood pressure over time (NEDA, 2018). The utilisation of the heart muscle for energy can subsequently increase the risk of severe cardiac complications (NEDA, 2018).

To help better manage these types of complications, the concept of 'energy availability' has been proposed (Mountjoy et al., 2014). Energy availability has been suggested as a helpful measure for assessing energy adequacy (EA), particularly in athletes (Melin et al., 2015). LEA has been defined as <30kcal/kg of fat free mass for females and between <15 -30kcal/kg fat free mass for males (no consensus has been reached in males; Rocks, Pelly, Slater, & Martin, 2016). LEA can also cause psychological distress, whereby the brain can become hyper alert, anxious, highly vigilant, threat-sensitive, rigid, resistant to change, ruminate, and fearful, which can also effect sleep due to the inability to relax (Gaudiani, 2018). These psychological disturbances can recover with nutritional rehabilitation (Gaudiani, 2018).

Understandably, inadequate caloric consumption relative to individual energy requirements has been postulated as a contraindication to exercise (Cook et al., 2016; Quesnel et al., 2017). Ghoch and colleagues (2013) have agreed that exercising in a starved state will inevitably pose health and performance risks via the following mechanisms. Firstly, starvation can result in glycogen depletion causing a premature reduction in physical and physiological capacity (Ghoch et al., 2013). Secondly, exercising in a starved state encourages circulatory lactate producing widespread muscular pain (Ghoch et al., 2013). Thirdly, a state of hypohydration may trigger muscular cramps (Ghoch et al., 2013). Finally, if undernourished while engaging in exercise, a loss of lean mass will eventually occur, provoking a reduction in muscle strength and aerobic performance (Ghoch et al., 2013). Consequently, Noetel and colleagues (2017) suggest an inadequate energy availability prior to or following exercise may contraindicate exercise engagement. It has also been suggested that modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favor improved energy and nutritional availability and the improvement of LEA-related consequences (Mountjoy et al, 2018).

Hydration

Moderate exercise engagement over a one-hour period can produce a sweat loss of 0.5-1L (McArdle et al., 2010). Hypohydration can rapidly reach levels that reduce the body's ability to dissipate heat (McArdle et al., 2010). As a result of decreased sweating and skin-blood flow, both of which are vital in cooling the body, heat can accumulate and the cardiovascular system can become strained (McArdle et al., 2010). If an individual's body weight falls just 1-3% due to dehydration, their body can experience a slowed gastric emptying rate, cramping, and nausea (McArdle et al., 2010). If an individual is severely dehydrated and unable to rehydrate themselves, this is a contraindication to exercise as it may contribute to increased risk of heat illness and negatively affect blood volume and exercise capacity (El Ghoch et al., 2013; McArdle et al., 2010). Indeed, the International Olympic Committee note that dehydration induced haemodynamic instability can contraindicate engagement in sport (Mountjoy et al., 2014).

Exercise Dependence Score

Exercise dependence can present when an individual engages in exercise despite being injured or ill, experiences withdrawal effects when unable to exercise (e.g. increased anxiety, low mood) or gives up social, occupational, and family obligations to exercise (Hausenblas & Symons Downs, 2002). Exercise dependence can negatively impact many areas of wellbeing and decrease the overall health-related quality of life (HRQOL) in people with EDs (Cook & Hausenblas, 2011). The negative effects of exercise dependence on HRQOL, combined with the detrimental impact of other ED pathology on HRQOL, may significantly and continually worsen overall HRQOL in people with EDs (Cook et al., 2014). It is important to note that even if an individual's physical health is deemed 'normal', the psychological reliance on exercise may still remain (Quesnel et al., 2017). Considering this, it may be helpful for the individual to engage in lower intensity and less frequent activity to decrease the reliance on exercise (Quesnel et al., 2017). Furthermore, individuals may be encouraged to refrain from engaging in previously dependent exercise formats, particularly if also exhibiting physical or emotional signs of overtraining syndrome (El Ghoch et al., 2013). Healthy exercise engagement, in combination with appropriate movement psychoeducation, has been found to decrease exercise dependence (Schlegel et al., 2015).

Purging and Hypovolemia

Self-induced vomiting and the misuse of laxatives or diuretics are the most common methods of purging used in those with disordered eating and diagnosed EDs (APA, 2013). Using laxatives and vomiting can cause water losses of up to 5000mL, which far exceeds values of normal water loss, and can also cause potentially dangerous fluid and electrolyte imbalances (McArdle et al., 2015). Furthermore, these behaviours are also associated with complications such as issues affecting the throat and mouth (such as dental erosion), heart (including electrolyte abnormalities and cardiac arrest), gastrointestinal system (such as rectal prolapse), and kidneys (such as renal failure) (Forney, Buchman, Schmitt, Keel, & Frank, 2016). Further, these behaviours can contribute to dehydration, metabolic alkalosis, severe kidney disease, and seizures, as well as softening of the bones with the use of certain laxatives (El-Sherif & Turitto, 2011; Finsterer & Stollberger, 2014; Frame, Guiang, Frost, & Reynolds, 1971). Importantly, these negative health sequelae continue to be significant in individuals engaging in subclinical rates of purging (Raj, Keane-Miller, & Golden, 2012). In fact, a history of purging may be a more sensitive indicator of health complications than lifetime BN diagnosis (Raj et al., 2012).

Vomiting can cause water and electrolytes to be lost from the body, which can lead to a state of hypohydration (Mehler & Andersen, 2017). The use of diuretics and laxatives can also create fluid and electrolyte changes, however, in slightly different ways to vomiting (Mehler & Andersen, 2017). Firstly, electrolytes are regularly and negatively altered in purging-related eating disorders, with the most common electrolytes affected including potassium, sodium and bicarbonate (Mehler & Andersen, 2017). In addition to electrolyte loss, a comorbid and effect of purging is significant water loss affecting vital hydration status (McArdle et al., 2015; Mehler & Andersen, 2017). For example, low blood pressure in individuals with ED can occur as a result blood volume depletion due to insufficient food and fluid intake and the presence of vomiting and laxatives (McCallum et al., 2006). As a result, even mild dehydration (1-2% of body weight) can cause an individual to become symptomatic (American Heart Association, 2016).

In addition to the health consequences mentioned above, purging, irrespective of the form, can have a detrimental effect on exercise performance (Mehler & Andersen, 2017). Mehler and Andersen (2017) state that purging can influence the performance via three main mechanisms; 1) the generation of a negative caloric balance; 2) the facilitation of dehydration; and 3) particularly in dehydrated individuals, purging can induce hypokalemia, which is

associated with fatigue and cramps, as well as heart palpitations, fatigue, respiratory difficulty and paralysis (American Heart Association, 2005; Mehler & Andersen, 2017). Severe hypokalemia can cause rhabdomyolysis, significant cardiac arrhythmias and sudden death (Mehler & Andersen, 2017).

Considering the common disturbance of critical electrolytes and water loss from purging, in addition to electrolytes naturally lost in sweat during exercise (Lindsay, 2017; McArdle et al., 2015; Mehler & Andersen, 2017), it has been suggested that electrolyte imbalance and a hypohydrated state may contraindicate exercise engagement (Fletcher et al., 2013; Thomas, Erdman & Burke, 2016). Furthermore, The American College of Sports Medicine (2015) suggests hypovolemia (decreased blood plasma volume) may be a contraindication to exercise (Thomas et al., 2016).

Signs and symptoms that may contraindicate exercise engagement.

The following are a list of signs and symptoms which may contraindicate exercise engagement (ACSM, 2014; ACSM, 2015; ACSM, 2016; Beumont et al., 1993; Rhodes, Temple & Tuokko, 2011; Warren & Pelroth, 2001). Clinicians should assess for the presence of these signs and symptoms and investigate the causes of these symptoms prior to making exercise prescriptions.

Symptoms that may contraindicate exercise engagement:

- **Ongoing, unstable or** moderate to severe chest pain
- **Near-syncope**
- **Dizziness**
- **Pallor (paleness)**
- **Cyanosis (bluish skin colour)**
- **Central nervous system dysfunction**
- **Intoxication from drugs or alcohol**
- **Ataxia**
- Shortness of breath
- Lightheadedness
- Confusion
- Nausea
- Cold/clammy skin
- Wheezing
- Leg cramps
- Claudication
- Fatigue
- Peripheral oedema

Box 14. Symptoms that may contraindicate exercise engagement

Note: Symptoms in bold have been suggested as more likely to contraindicate exercise engagement.

Intensity Measures					
Description of intensity	Rating	Talk Test	Exertion (work)	% of heart rate maximum	MET value*
Maximal	10	Cannot talk, gasping for breath	Difficult to continue, can only maintain for 10-30 sec	86-100%	6+
High	9				
	8	Broken speech, heavy breath	Uncomfortable to continue, can maintain for 5-10 mins	76-85%	
	7				
Moderate	6	Can only finish 1-2 sentences, moderately short of breath	The work is tough, can maintain for 30 mins	61-75%	3 - 5.9
	5				
	4	Taking more effort to talk, slight shortness of breath	Can comfortably maintain for at least 60 mins	51-60%	
Low	3	Normal talking and breathing	Can continue for an extended time	40-50%	1.1 - 2.9
	2				
	1				

Box 15: Intensity Measures

*For a list of MET values with corresponding activity examples, see the Compendium link on our website under the Resources tab.

(<https://sites.google.com/site/compendiumofphysicalactivities/home>)

Glossary of Terms

Anorexia Nervosa (AN): Anorexia Nervosa is a psychiatric disorder characterised by persistent restriction of energy intake leading to body weight that is significantly below expected body weight. These symptomatology comprise restricted food intake with or without behaviours of purging and compensation. These factors are coupled with an intense fear of gaining weight/becoming fat and distorted weight/shape perception or undue influence of weight/shape on self-evaluation. Subtypes of AN include restrictive and binge-eating and/or purging subtype (American Psychiatry Association, 2013).

Binge Eating Disorder (BED): Binge eating disorder is a recent addition to the DSM-5 as an eating and feeding disorder. It is marked by recurrent episodes of eating significantly more in a discrete period of time (typically less than two hours) than most people would under similar circumstances, with these episodes being marked by a sense of a lack of control. During these episodes, individuals may eat alone, too rapidly, until overly full, or overeat when not physically hungry; while afterward, they may feel guilty, disgusted, embarrassed and/or distressed (American Psychiatric Association, 2013). BED is differentiated from BN (below) by an absence of compensatory/purging behaviours.

Body Image: Body image is a multifaceted construct comprising an individual's cognitive, affective and perceptual experience of one's body (Cash & Pruzinsky, 2002). Disordered body image arises when cognitions and emotions associated with an individual's body image perception and satisfaction begin to detrimentally affect their self-worth or body esteem, or result in clinical distress and dysfunctional behaviours (Barlett, Harris, Smith, & Bonds-Raacke, 2005).

Body Mass Index (BMI): A statistical measure of weight according to height. The calculation for BMI is kilograms/metre² (McArdle et al., 2010).

Bulimia Nervosa (BN): Bulimia nervosa is a psychiatric disorder characterised by frequent episodes of binge eating (as with BED above) in conjunction with inappropriate compensation methods, such as self-induced vomiting, excessive exercising, or misuse of laxatives or diuretics to avoid weight gain. Self-evaluation, similar to AN, is also unduly influenced by weight/shape (American Psychiatric Association, 2013).

Clinician: A clinician is defined as a person (such as a doctor or nurse) who works directly with clients rather than in a laboratory or as a researcher (Marriam-Webster, 2016).

Disordered Eating: This term encompasses eating behaviours that are maladaptive, such as restrictive dieting, bingeing and purging, and the combination of these behaviours without diagnostic significance. Individuals may engage in these behaviours less frequently or may experience less impairment/dysfunction related to these behaviours than is required to meet an eating disorder diagnosis (Canadian Mental Health Association, 2015).

Dysfunctional Exercise: Many terms (i.e exercise addiction, exercise abuse, excessive exercise, exercise anorexia, obligatory exercise) are present in the literature outlining variants of exercise symptomatology in EDs. The term dysfunctional exercise serves to encompass all such terms, ranges and labels (Calogero & Pedrotty-Stump, 2010).

Eating Disorder Not Otherwise Specified (EDNOS): A psychiatric diagnosis to encompass disorders of eating that do not meet the criteria for a specific eating disorder (Grilo & Mitchell, 2011).

Exercise: Planned, structured, repetitive and purposeful physical activity (McArdle et al., 2010).

Exercise Prescription: A specific plan of fitness related activities designed by a fitness or rehabilitation specialist (American College of Sport Medicine, 2010).

Exercise Professional/Specialist: A professional who has received a tertiary degree in Exercise Physiology, Kinesiology (in Canada) or Human Kinetics with a focus on exercise physiology and/or promoting physical activity and exercise behaviours.

Health Professional: For the purpose of this guideline, this broad range category will encompass clinicians and researchers who hold a minimum of a Bachelor degree recognising certification in health service provision. This may include but is not limited to, medical practitioners, psychiatrists, nurses, psychologists, exercise physiologists, physiotherapists or occupational therapists.

Inpatient Treatment: Inpatient hospitalisation is provided to individuals who are physiologically and/or psychologically compromised and require intensive medical stabilisation (Geller et al., 2012).

Lived Experience: In phenomenological research, lived experience is a person's perspective of a situation that was acquired through their first-hand account of a situation (Creswell, 2007).

Medical Professional: A medical professional is defined as an individual who's aim is to promote, protect health. While, diagnosing and treating illness with specialized and scientific knowledge

Multidisciplinary Team: This refers to a group of professionals from a range of disciplines who are working collaboratively in support of an individual. While this may include any health professional, clinical guidelines for eating disorders recommend that this team should minimally include professionals administering medical, psychological and dietetic interventions (Hay et al., 2014).

One Repetition Maximum: The maximal amount that can be lifted in one complete repetition with proper technique (Canadian Society of Exercise Physiology, 2006).

Physical Activity: Any bodily movement produced by skeletal muscles that results in energy expenditure (McArdle et al., 2010).

Quality of Life: Encompasses multi-dimensional aspects of one's life including; physical and material well being, social well being, emotional well being and development and activity (Felce & Perry, 1995).

Residential Treatment / Outpatient Treatment: A mode of service delivery that targets medically stable individuals who are in need of a structured and intensive treatment program, outside of a hospital setting (Geller et al., 2012).

Resistance training: This refers to the combination of many consecutive resistance exercise sessions over time. Resistance exercise involves muscular work that causes the muscle to use force against an external resistance (ACSM, 2013).

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