Safe Exercise At Every Stage: Athlete SEES-A

A guideline for managing exercise and return to sport in athletes with eating disorders

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Acronyms

ACSM: American College of Sports Medicine
AGREE-II: Appraisal of Guidelines for Research and Evaluation
AHA: American Heart Association
AN: Anorexia nervosa
AN-P: Anorexia nervosa - Purging subtype
AN-R: Anorexia nervosa - Restrictive subtype
ANS: Autonomic nervous system
BED: Binge eating disorder
BF: Body fat
BMD: Bone mineral density
BMI: Body mass index
BN: Bulimia nervosa
BPM: Beats per minute
BUN: Blood urea nitrogen
CBT: Cognitive behavioural therapy
CBT-A: Cognitive behavioural therapy for Anorexia nervosa
CBT-B: Cognitive behavioural therapy for Bulimia nervosa
CBT-E: Enhanced cognitive behavioural therapy
CET: Compulsive Exercise Test
CRT: Cognitive remediation therapy
CVD: Cardiovascular disease
DBT: Dialectical behaviour therapy
DSM-IV: Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition)
DSM-5: Diagnostic and Statistical Manual of Mental Disorders (Fifth Edition)
EA: Energy availability
ECG: Electrocardiogram
ED(s): Eating disorder(s)
EDEA: Exercise Dependence and Elite Athlete Scale
EDNOS: Eating disorder not otherwise specified
FBT: Family based therapy
FBT-AN: Family based therapy for Anorexia nervosa
FBT-BN: Family based therapy for Bulimia nervosa
FFM: Fat-free mass
FHA: Functional hypothalamic amenorrhea
FSH: Follicle stimulating hormone
GH: Growth hormone
GnRH: Gonadotropin-releasing hormone
HR: Heart rate
HRQOL: Health related quality of life
IBW: Ideal body weight
IGF-1: Insulin-like growth factor 1
ISCD: International Society for Clinical Densitometry
Kcal/kg: Kilocalories per kilogram
LEA: Low energy availability
LEAP: Loughborough Eating disorders Activity Therapy
LH: Luteinizing hormone
IBW: Ideal body weight
MET: Metabolic equivalent
mL: Millilitre
mg/dL: Milligrams per decilitre
mmHg: Millimetres of mercury
mmol/L: Millimoles per litre
mEq/L: Milliequivalents of solute per litre
MANTRA: Maudsley Model of Anorexia Nervosa Treatment for Adults
NEDA: National Eating Disorders Association
NHRMC: National Health and Research Medical Council
OH: Orthostatic hypotension
OSFED: Other specified feeding and eating disorders
PT: Postural tachycardia
RANZCP: Royal Australian and New Zealand College of Psychiatrists
RCT: Randomized controlled trial
RED-S: Relative Energy Deficiency in Sport
RMR: Resting metabolic rate
RT: Resistance training
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 (but not limited to) anorexia nervosa (AN), bulimia nervosa (BN), other specified feeding or eating disorder (OSFED) (previously recognised as eating disorder not otherwise specified; EDNOS), 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 all psychiatric diagnoses (Reel, 2013). Treatment of these chronic and severe disorders typically incorporates psychotherapy, pharmacological treatment, and nutrition counselling (Geller, Goodrich, Chan, Cockell, & Srikameswaran, 2012; Grilo & Mitchell, 2011).

Eating disorders in athlete populations

Athletes are particularly at risk of developing an ED, with reported incidence of both disordered eating behaviours and diagnosed EDs rising over the past decade (Conviser, Tierney & Nickols, 2018). The prevalence of EDs in athletes is significantly higher than in the general population, with studies reporting that female athletes experience EDs at rates between 6 – 45% and male athletes up to 19% (Glazer, 2008; Martinsen & Sundgot-Borgen, 2013; Sundgot-Borgen & Torstveit, 2004). However, due to the common underreporting of ED symptoms in athlete populations, the true prevalence is likely to be substantially higher (Sundgot-Borgen & Torstveit, 2004). Additionally, athletes identifying as non-binary or gender queer have not yet been represented in the literature, representing a vital consideration for future research.

Recognition of the heightened incidence of EDs in athlete populations has resulted in new terms being published to describe this phenomenon, such as weight cycling, the female athlete triad, relative energy deficiency in sport (RED-S), and anorexia athletica (Sudi et al., 2004; Sundgot-Borgen & Garthe, 2011). Despite sport participation typically being associated with positive self-perception, competitive sporting environments can also infer additional risk factors for the development and maintenance of ED symptomatology (Smolak, Muren, & Ruble, 2000; Thompson & Sherman, 2011). It is therefore imperative that athletes, as well as their sporting team personnel (including staff and teammates), specialist sporting ED clinicians, sport-specific organisations, as well as national and international sporting governing bodies address the unique sport-related needs and goals of the athletes during their ED treatment. These considerations are vital to successfully navigate complex systems and maximise the likelihood of recovery (Arthur-Cameselle & Quatromoni, 2014).

Dysfunctional exercise (DE) is a term often appearing in ED literature in non-athlete populations. DE is an overarching term used to encompass other terms for unhealthy exercise engagement, including compulsive exercise, exercise dependence, exercise addiction or excessive exercise (Carter, Blackmore, Sutandar-Pinnock, & Woodside, 2004). Continued engagement in DE has been associated with an increased rate of relapse, longer illness chronicity, and poorer

general ED prognosis (Carter et al., 2004; Strober, Freeman, & Morrell, 1997). While athletes may also engage in DE, the relationship between athletes with EDs and exercise may be more complicated than in non-athlete populations. Athletes with EDs can engage in exercise for reasons similar to non-athletes with EDs (i.e. weight and shape concerns, positive mood reinforcement, negative affect avoidance; Thompson & Sherman, 2011). Yet, some also continue their existing training regimen to meet competition demands in the presence of an ED. Irrespective of the purpose or motivation athletes to engage in exercise, engaging in unmodified physical activity levels with an ED puts individuals (including athletes) at heightened risk for increased ED psychopathology and relapse, in addition to a host of medical complications concerns and negative performance outcomes (Dalle Grave et al., 2008; Thomas et al., 2016).

In 2018, the Safe Exercise at Every Stage team developed an initial set of evidence-based guidelines to support clinicians in the assessment and treatment of exercise in adult populations with an eating disorder (http://www.safeexerciseateverystage.com). However, there is an urgent need for appropriate recognition and response by those working with athlete populations, as well by the athletes themselves, of the complex relationship between EDs and competitive sports. This recognition is vital to ensure ED symptomatology is adequately addressed, while simultaneously supporting sporting success, and minimising both short and long-term adverse outcomes.

The rationale for athlete-specific guidelines

Treatment for athletes with EDs may be distinctly different from non-athletes in several ways. For example, many non-athletes with an ED may be motivated to attend treatment to alleviate distress, improve relationships or functioning. However, many athletes with EDs are predominantly concerned with returning to competitive sport (Arthur-Cameselle & Quatromoni, 2014). Additionally, many athletes report initiating restrictive eating, excessive exercise, and/or other unhealthy weight control practices primarily for performance benefits, as opposed to the primary drive for thinness that is more common in non-athlete populations (Prelack, Dwyer, Ziegler, & Kehayias, 2012). Such differences contribute to athletes reporting feeling "out of place," in standard treatment, potentially resulting in decreased motivation for treatment (Plateau et al., 2017). Subsequently, significant space exists for more detailed instruction and greater information on how to best adapt treatment for ED to athlete populations (De Souza et al., 2014).

ED treatment for both athletes and non-athletes continues to be plagued by a lack of detailed instruction on how to best assess and manage safe exercise engagement. This is further complicated for the treatment of athletes, who, in addition to standard interventions, may also require additional and specialised support to transition safely back to training and competition (Mountjoy et al., 2014). As such, many health professionals have therefore 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). While this recommendation may seem reasonable considering the range of medical complications present in ED populations (Davies, 2015; Hechler et al., 2005; Thien et al., 2000; Zunker et al., 2011), prescribing complete exercise abstinence has also 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).

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 all exercise (Beumont, Arthur, Russell, & Touyz, 1994; Ng, Ng, & Wong, 2013; Quesnel et al., 2017). These concerns have continued to gain momentum given that research has supported that nutritionally adequate, supervised, and clinically prescribed exercise can be 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; Mathisen et al., 2020; Moola et al., 2013; Vancampfort et al., 2014). Specifically, for athlete populations, considered, individualized, and evidence-based opportunities to continue training or participate in competition allows athletes to maintain their sporting identity, a key source of self-esteem and social support, during ED treatment and recovery (Lally, 2007). Even prior to returning to competition, promoting the athlete identity and incorporation of exercise into ED treatment has been associated with increased motivation for treatment, and improved performance markers (Thompson & Sherman, 2011). Consequently, the Safe Exercise at Every Stage - Athlete (SEES-A) guideline has been developed to better facilitate the management of safe exercise in ED treatment, as well as return to training and competition for athletes with an ED. The SEES-A guideline builds upon the core concepts of the original Safe Exercise at Every Stage (SEES) guideline, with additional adaptations for the unique needs of athlete populations. The SEES-A guideline will remain a stepped and concise, simple and graded process to support clinicians in determining the level of exercise and education appropriate for each athlete, 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 and expert stakeholders worldwide (including clinicians, researchers and individuals with lived experience). It is our hope to disseminate the guideline as an international standard (for further details see http://www.safeexerciseateverystage.com).

Statement of Intent

The Safe Exercise at Every Stage-Athlete (SEES-A) guideline aims to provide evidence-based information to support clinicians in the management of safe exercise and return to sport (training and competition) during ED treatment for athletes. This guideline serves as a general tool for use under a specialist 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-A 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 EDs. As such, the SEES-A guideline is intended to be administered by a trained medical or exercise professional (see glossary, pp.75), preferably with expertise in working with athletes, in conjunction with a interdisciplinary team (see glossary, pp.75). The SEES-A guideline was adapted from the original SEES guideline, which 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 original SEES and SEES-A guidelines have been developed based on a consensus by our steering committee (comprising an international panel of experts in the combined ED and athlete field; 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-A guideline as new information and stakeholder feedback emerges.

Executive Summary

Disordered eating behaviours and diagnosable EDs are more common in athletes than in non-athlete populations (Glazer, 2008; Sundgot-Borgen & Torstveit, 2004). Athletes engaging in unmodified exercise with a concurrent ED or disordered eating behaviours, are at risk of injury, fatigue, and dehydration as well as worsened mental and physical health outcomes (Conviser et al., 2018; Mathisen & Sundgot-Borgen, 2013; Mountjoy et al., 2018). Although returning to sport is often a key motivator for athletes to initiate ED treatment (Arthur-Cameselle et al., 2018), a hasty return to training may increase the likelihood of relapse (De Souza et al., 2014). This return to sport process is a milestone that requires a step-up/step-down approach and recommendations for exercise engagement tailored to the individual athlete's current physical and psychological well-being (Cook & Leininger, 2017). The SEES-A guideline provides a framework for thoroughly assessing each individual's current physical and psychological health status. These results are then used to augment clinical decision-making to select safe levels of exercise and sports participation. We anticipate that incorporating the SEES-A guideline into evidence-based treatment for athletes with ED will promote an ethical standard level of care and improve consistency in the management of dysfunctional exercise.

Key Principles

The primary authors identified five key principles as underpinning their intentions in developing the original SEES guideline. These principles have been tailored to apply to athlete populations, have guided the development of this guideline, and should be employed when applying its recommendations. The key principles include: non-abstinence, safety, holistic, body awareness and connection, and collaboration.

1. Non-abstinence

This principle was central to our motivation to develop the original, and then athlete-specific, SEES guidelines. 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 contribute to negative affect withdrawal symptoms (Geller, Cockell, & Goldner, 2000; Morris, Steinberg, Sykes, & Salmon, 1990; Noetel et al., 2016). 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). For athletes specifically, Stranberg and colleagues' (2020) report that 15 athletes completing a skills group that incorporated exercise and meal prescription, described increased tolerance of rest, improved understanding of health consequences of DE, as well as recognition of the importance of recovery for athletic performance and success. As such, this guideline was developed in order to facilitate clinicians in promoting non-abstinence during ED treatment.

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). In athletes, the relationship between exercise and EDs becomes increasingly complex due to the central role it plays in their lives. However, studies conducted in athlete populations have also demonstrated that dysfunctional exercise is associated with more chronic ED symptomatology (Plateau et al., 2014). Thus, it is crucial that exercise prescription and return to sport in ED treatment promotes the safety of athletes. Safe exercise prescription is currently limited, due, in part, to gaps in the translation of research to health professionals (Quesnel et al., 2017). This limited knowledge has likely contributed to uncertainty and confusion for professionals on best practices for the safety of clients engaging in any exercise during ED. The SEES and SEES-A guidelines have subsequently been developed to provide a summary of current evidence, with the aim of addressing concerns related to safety and improving upon clinical knowledge of exercise engagement and eventual return to sport during ED treatment. Furthermore, these guidelines have been developed to promote physical and psychological safety as paramount for any exercise intervention during ED treatment. We hope that by providing a safety-focused guideline, SEES and SEES-A may help alleviate concerns of health professionals regarding the prescription and management of exercise during ED treatment.

3. Holistic

The human relationship with exercise is multifaceted, comprising physical, emotional, social, cognitive, and sociocultural components (Calogero & Pedrotty, 2007). Athlete engagement in exercise, sport, and competition, consequently, influence and are influenced by each of these individual components (Calogero & Pedrotty, 2003). Clinical management of exercise in ED treatment commonly considers physical risks of returning to exercise, however, can an athlete's psychological relationship with exercise. While promoting a safe and healthful exercise has been paramount in developing the SEES and SEES-A guidelines, we also further define this relationship with exercise to incorporate these socio-emotional aspects of exercise and sport engagement. Addressing these dimensions through a holistic lens is integral to supporting

individuals to develop a healthy relationship with exercise and their sport. Our prioritisation of a holistic approach underpins the combination of medical, cognitive, emotional, and behavioural benchmarks in the SEES and SEES-A guidelines. 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. Body awareness and connection

A healthy body must include a healthful connection with one's body and awareness of physical cues (Calogero and Pedrotty, 2007). This concept may be foreign to some athletes and sporting team personnel, as many components of dysfunctional exercise (i.e. rigid inflexibility, punitive attitudes, and guilt), are praised and promoted in competitive sporting environments (Meyer, Taranis, Goodwin, & Haycraft, 2011; Thompson & Sherman, 2012). Further, some athletes – particularly those in aesthetic sports – may view themselves through an objective lens (i.e. how their body is viewed, rather than how it feels), thus diminishing interoceptive awareness. Although athletes are often accustomed and required to push their body through pain and mental barriers to achieve peak performance, this approach may intensify ED psychopathology and risk exacerbating medical complications of the ED (Conviser et al., 2018). Clinicians can educate athletes about risks associated with a hasty return to exercise, as well as benefits of a graded return to sport emphasising physical, mental health, and performance outcomes (Conviser et al., 2018). Building a healthful connection with one's body also includes learning to observe 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).

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). This can be particularly important in the case of athletes where The SEES-A guideline has, consequently, been designed to promote collaboration between the athlete, their loved ones, sporting personnel, 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 motivation and ambivalence, and fostering 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. This approach aims to facilitate open and honest conversations with athletes about their training, sporting environment, and goals, as well as identifying their needs and concerns about return to sport.

Overview of the SEES-A guideline

Objective

Return to sport is often the key priority for athletes with an eating disorder (ED), however, the recovery process must carefully consider the psychological, physical, and performance risks of engaging in physical activity during ED treatment. As athletes with ED symptomatology often exhibit an array of medical health complications, it can be challenging for clinicians, coaches, and other team personnel in this field to determine safe prescriptions for exercise engagement. The SEES-A guideline was developed to address the management of exercise (see glossary pp.72) and training (see glossary pp.75) in adult athlete ED populations. Recommendations in this guideline are intended to provide guidance for clinicians to better facilitate clinical decision-making related to exercise prescription and return to sport for individuals with ED symptomatology. The use of the SEES-A guideline is intended to reduce the likelihood of over- and under-prescribing exercise relative to the athlete's individual physical and psychological health status. Consequently, this will support athletes with ED symptomatology to engage in safe levels of exercise, training and education throughout their treatment and return to sport, contributing to improved psychological and physical outcomes.

Target users and population

We developed the SEES-A guideline for use by trained exercise and medical professionals working with athletes 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-A 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-A guideline apply to athletes at all stages of their ED and to not determine health status based upon a specific diagnosis (for example, bulimia nervosa or anorexia nervosa). Consequently, this guideline was developed for use with athletes over the age of 18 exhibiting ED symptomatology (e.g. dietary restriction, purging, and compensatory behaviours). The SEES-A guideline may require adaptation by a trained medical team or accredited exercise professional (pp.74-75) for special populations such as: children/adolescents, and individuals with diabetes, osteopenia/osteoporosis, or other existing metabolic, neurological, cardiovascular/respiratory, 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-A guideline is a tool for supporting clinical decision-making regarding exercise interventions for health professionals working with athletes with eating disorder symptomatology.
- The SEES-A guideline is intended for use in all levels of care for individuals experiencing an ED or ED symptomatology.
- The SEES-A guideline applies to individuals at every stage of their eating disorder, regardless of their weight, size, or individual diagnosis.
- The SEES-A guideline was developed for use in adult populations and is not suitable for use with children or adolescents.
- The SEES-A guideline is not intended to replace clinical or medical judgment and recommendations should be made with consideration of the clients' individual needs.
- The SEES-A guideline may need to be adapted or not be appropriate for individuals with comorbid conditions.

Methodology

The SEES-A 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. Overall recommendations and content have been adapted from the original SEES guideline, with research and feedback from experts in the treatment of athletes with EDs. 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-A meets high standards.

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

- Review of existing guidelines.
- 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.

With the following adaptations for SEES-A:

- Focus groups with steering committee members.
- Review of relevant literature.
- Consultation with steering committee experts.
- Review of final guideline by steering committee.
- Approval of SEES-A final document by steering committee.

Method of consultation process

Building upon the consultation process outlined in the original SEES document, we completed further consultation for SEES-A with our steering committee (pp.3). Committee members were able to provide their feedback during an online focus group or via written submission of information. Following completion of the SEES-A guideline, the steering committee were again invited to provide feedback related to the guideline.

Results of initial consultation process

Feedback from steering committee members was as follows:

Differentiating athletes from non-athletes. The steering committee identified that athletes with EDs are clinically different from those with EDs who are "athletic," or who engage in dysfunctional exercise. Committee members highlighted several factors that may help identify an athlete, including: regular competing (prior to ED onset), engagement in purposeful training (with the purpose being to maximise sporting performance), having athletic-focused goals, working with a coach and/or sports personnel, and possess motivation to return to sport and competition. Although individuals may self-identify as an athlete, it was noted that "athlete status" should be an ongoing, collaborative conversation between the athlete and the treatment team, although this is ultimately to be determined by the treatment team.

The role of coaches in ED recovery. Coaches play a key role in determining team culture and environment surrounding the athlete with an ED. They are also vital in removal and return to play decisions pertaining to the athlete, as well as managing the athlete's periodised training schedule. Our steering committee recommended ED treatment teams should engage with coaches early (if permission to converse and disclose information is received from the athlete) and work to build rapport with them as a valued and important contributor to the athlete's recovery. Further recommendations included providing coaches with resources to better assist their understanding of the seriousness of their athlete's ED, help screen and monitor disordered eating behaviours in their athletes, be presented with ways to constructively communicate the effects of EDs with team members, and support their athletes' safe return to play. Finally, it was recognised that not all sport team personnel may be constructive to the athlete's ED recovery.

Weight class sports. One risk factor unique to athlete populations is the need to meet specific weight requirements for sports participation (e.g. combat sports, lightweight rowing, and horse racing). In particular, athletes striving to alter their weight and body composition to meet sport-specific standards may be at risk of engaging in disordered eating and/or exercise behaviours associated with negative health outcomes and impeded performance. The committee supported the notion that (where possible) sport selection should consider an athlete's natural phenotype including their build, body shape, weight, and size (when engaging in healthful eating, exercise and training behaviours), rather than solely on their sporting preference. Attempts to alter an athlete's natural phenotype may contribute to disordered eating and exercise behaviours to make and maintain the "ideal" weight, shape or size required by the sport. Unfortunately, this recommendation may preclude some individuals from competing in their originally chosen sport.

However, our steering committee recommended that engaging athletes in this conversation early, sensitively, and with a sense of optimism about the opportunity to excel in a well-suited sport, may help improve outcomes.

Weigh in protocols. The steering committee agreed that weigh ins for sport can be particularly stressful for athletes with EDs. They discussed that a team physician or athletic trainer may be best suited to conduct weighing procedures, if required, and for this information and process to remain confidential.

Team messaging about EDs. Team culture was identified as playing a helpful or potentially harmful role in ED recovery for athletes. The steering committee proposed that team members and personnel, including the athlete, should be critically aware of the complexity, seriousness, and risks of an ED in athlete populations. Using scientifically accurate, legitimising, and empathetic language throughout team messaging related to ED, RED-S, and LEA was recommended, for example describing the ED as a 'metabolic injury'. Additionally, the committee proposed that team messaging should promote healthful and evidence-based nutrition information, highlight the pivotal role of adequate, varied, and regular nutrition in optimising health and sporting performance, as well as promote healthy athlete weights and body composition (and not a fear of fatness). Positive role models may support an athlete with an ED, however, unhealthy modelling can also contribute to the normalising of ED behaviours. In particular, the committee highlighted the importance of addressing the unintentionally destructive normalisation of amenorrhea, pubertal delays, and stress fractures, recognising that these may represent signs of low energy availability (LEA) or malnutrition or other problems related to an athlete's health.

Differentiating return to exercise, return to training, and return to competition. Our steering committee also identified that an athlete's return to sport may comprise several stages, from the medical clearance of returning to exercise and then training, to the later stages of returning to regular competition. It was highlighted that medical stability and safety to participate in exercise should not differ between athletes and non-athletes; that is, the individual's safety must be strictly prioritised regardless of potential or perceived costs of ceasing the sport at the time (e.g. financial costs, loss of scholarship, or missed competitions). An athlete who is medically and/or psychologically compromised must abstain from engaging in unmodified training, only engaging in appropriately modified exercise only until progression markers are achieved (as outlined in the original SEES document). Only once these criteria are met can return to sport be considered, with the steering committee stating that athletes must demonstrate additional and unique markers to progress to returning to training for their sport and for competition. These markers may utilize information from the RED-S and REDS- CAT return to play guidelines (Mountjoy et al., 2014; 2015) as well as psychological markers highlighted below, and other individual factors specified in a treatment contract (see pp.23) with athletes during treatment.

Psychological readiness to return to sport. The steering committee agreed that absence of contraindications and achievement of specific health markers required to return to tailored exercise will look similar for both athletes and non-athletes with an ED due to similar human homeostatic requirements. However, they recognized that psychological readiness to return to

training, sport, and competition was unique to this group when compared with non-athletes. It was noted that an athlete's brain and body may often recover at different speeds or even in a nonlinear manner. This can complicate the return to training and competition progression, for example, potentially providing medically stable athletes with false sense of security or readiness for their return to sport. Important markers of psychological readiness to return to sport were identified as: ability to tolerate rest, demonstrated use of non-exercise strategies to regulate emotions, psychological and nutritional treatment plan compliance (Currie & Crosland, 2009), and decreased perseveration on return to sport. Finally it was noted that it may not be physically or psychologically healthy for all athletes to return to their sport, or return in the same capacity.

Literature search strategy and data extraction

The following literature review was conducted for the development of the original SEES guideline. The authors (AD, MC) conducted comprehensive systematic searches of the literature during the period of January-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- analysis.

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-A guideline be reviewed for updated information in 2025; 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-A 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-A guideline through our website (https://www.safeexerciseateverystage.com/).

Limitations

It is important to highlight the following limitations to the SEES-A 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 athletes with EDs. Consequently, the SEES-A 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-A guideline will support clinicians in their management of athlete activity and return to sport, as an adjunct 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-A guideline will support trained medical and exercise professionals (pp.63) in these situations to augment their current clinical judgement and knowledge.

- The SEES-A guideline was developed to address the specific needs of athletes and should not be implemented in diverse cultural or other special populations. We recommend that clinicians applying the SEES-A guideline should tailor interventions to each individual's needs.

Application of Guidelines

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

Facilitating the implementation of SEES-A

The following are encouraged strategies for supporting the implementation of the SEES-A guideline when working with athletes with an ED. Please see the original SEES guideline for further recommended strategies for working with adults with ED which included: use of in-vivo tasks, incorporating social supports, using a motivational and collaborative approach, promoting intuitive movement, increasing awareness of the ED function, and psychological strategies for managing dysfunctional exercise.

Determining athlete status. An athlete should be defined as someone who is striving for excellence by training to enhance performance. This includes any person who trains purposefully, with a structured frequency, and devotes themselves to a sport/s for the purpose of competition or sustained recreational purposes (for example serious mountain climbing). We note that athletes do not have to be currently competing or ever planning to compete at an elite level to be classified as an athlete. To account for athletes in different stages of the year (e.g. on/off seasons), we highlight that purposeful training with or without competition is key to the definition, such that it contributes to the development of an identity tied to that sport. During ED treatment, athletes will typically prioritise returning to sport and must be doing so in order to compete and train regularly. Self-identification as an "athlete," may be the first factor in deciding if a client should be considered an athlete in ED treatment. Other factors influencing this decision are: the context of an athlete's sport, for example, are they on a team sport or individual sport; what level of sport do they play; and the role of sport in their future finances and careers. Higher-level athletes, and those who require more tailored athletic ED treatment, must have a coach, and team personnel that bring structure to the sport as well as facilitate sporting goals. Other considerations such as professionalism or being considered part of a professional or semi-professional leagues, amount of time spent training, participation in elite talent development programs, or reaching peak goals in a sport such as becoming a black belt may be considered in this decision-making process (Swann, Moran, & Piggott, 2015). As a cautionary note, we would like to outline that EDs can become chronic and prolonged, in which case athlete "status" can be revoked if return to sport is not feasible due to the ED, associated physical or psychological complications, or relapse of ED symptomatology.

Interdisciplinary approach. The importance of an interdisciplinary 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, nutritional, and psychological interventions (Hay et al., 2014). 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). For athletes this team commonly expands to include members from the coaching team (e.g. athletic trainer, strength and conditioning coach, physical therapist, team physician, and coach Conviser et al., 2018), with the treatment team having ideally being experienced in working with athlete populations. Typically one member from the coaching team will be designated as a representative, usually an athletic trainer, team physician, or physical therapist (Bonci et al., 2008; Joy, Kaussman & Nattiv, 2016; Thomspon & Sherman, 2011). The representative role involves weigh-ins (if required for sport participation), maintaining the privacy of the athlete's treatment information (including weight), and collaborating with other treatment team members to progress or regress the athlete's level of sport participation (Bonci et al., 2008). As such, they will often work directly with medical and mental health providers from the treatment team to address health and performance considerations (Conviser et al., 2018). Conviser and colleagues (2018) outline an example of roles for members in the treatment and coaching team below.

Figure 1 Interdisciplinary, collaborative & credentialed professional treatment providers adapted from Conviser, Tierney, and Nickols (2018)

The key role of coaches. Coaches play a key role in an athlete's development and environment, representing a valuable asset in supporting ED recovery. Pragmatically, coaches spend a large portion of time with the athlete, inform removal and return to play decisions, and adjust the training schedule (Thompson & Sherman, 2011). Research shows that the behaviour, attitude, and management style of coaches can impact the development and maintenance of EDs (Nowicka, Eli, Ng, Apitzsch, & Sundgot-Borgen, 2013), with support from coaches significantly influencing an athlete's success in ED recovery (Sherman et al., 2005). Environments that emphasise surveillance and sacrifice are more likely to reinforce disordered eating practices (Papathomas, 2018). While many coaches report feeling worried and anxious about their athletes diagnosed with an ED (McArdle, Meade, & Moore, 2016), many also report a lack of confidence and knowledge in how to address and manage an ED (Nowicka et al., 2013; Plateau et al., 2014; Sherman, Thompson, Dehass &, Wilfert, 2005). Yet, most coaches are willing to work with the treatment team, understanding the seriousness and consequences of EDs for their coaches' health and performance (Sherman, DeHass, Thompson, & Wilfert, 2005; Thompson & Sherman, 2011). As such, it may be helpful for the treatment team to provide psychoeducation to the coach and coaching team as necessary to facilitate all treatment team members (inclusive of the sporting team) being aligned in working with the athlete and returning to sport. If a coach is not available or appropriate for involvement with the treatment team, we recommend that the athlete identify another member of their sports team with whom the treatment team could engage.

Create a written training 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), while for athletes this may also include requirements for ongoing training and/or competition participation (Bonci et al., 2008). The key components of behavioural contracting highlighted are: collaboration, relevant goal setting, clear monitoring of exercise/training within the parameters of the guidelines as well as treatment compliance, and setting appropriate consequences. Finally, it is noted that behavioural contracts should be flexible and collaborative with other treating team members as well as the coaching team (Yager, 1992).

The athletic identity. Addressing an athletes' relationship to and definition of the "athletic identity," is a unique aspect in the treatment of ED for athletes. The athletic identity can take up a large portion of an athlete's self construct and represent a vital source of self-worth for athletes with or without an ED (Arthur-Cameselle et al., 2018; Lally, 2007). Further, maintaining one's athletic identity throughout ED treatment may be associated with increased motivation for treatment (Arthur-Cameselle et al., 2018). And yet, those with high endorsement of the athletic identity may also be at greater risk for engaging in dysfunctional exercise (Goodwin et al., 2016; Turton, Goodwin & Meyer, 2017). Additionally, some athletes with EDs may be required to leave their sport due to irreparable consequences of the ED (Thompson & Sherman, 2011). For both athletes remaining in the sport but particularly those retiring, clinicians should support clients in developing a sense of self-worth and interests outside of their sport (and ED). Traditional ED therapies such as CBT-e (Fairburn, 2008) and MANTRA (Schmidt, Wade, & Treasure, 2014) include interventions addressing self-worth and identity that may prove helpful in addressing the athletic identity.

Identifying with a sport or being an athlete is often central to an athlete's self-concept, particularly in high-level sports (Kerr & Dacyshyn, 2008). For many athletes, the concept of retiring due to their ED and related consequences can be incredibly challenging to hear and process (Baum, 2006; Kerr & Dacyshyn, 2008). Athletes retiring from sport describe their experience as characterized by feelings of disorientation, void and frustration (Kerr & Dacyshyn, 2008), they may also be at greater risk of low self-confidence, depression, substance abuse, and risk of ED relapse, in the context of also losing vital support networks (Arthur-Cameselle et al., 2018; Lally, 2007). As such, these conversations must be conducted sensitively to the athlete's personal level of investment in the sport and to the athletic identity (Lally, 2007). We recommend that a trained mental health professional carefully monitor signs of hopelessness and suicidal ideation as well as work with athletes on developing coping skills to manage this transition.

Sporting weigh in and body composition assessment procedures. Many athletes may be uncomfortable with weigh in requirements for sport, these can be particularly anxiety provoking and emotional for athletes with an ED (Arthur-Cameselle, Sossin, & Quatromoni, 2017; Thompson & Sherman, 2011). Weigh ins and body composition assessments should only be conducted if necessary, particularly in lower levels of competitive sport and if chronological age is >18 years), and with consideration of associated risks. As such, we highlight several recommendations to increase sensitivity and consideration during these procedures including:

1. Identifying one individual (commonly team physician, athletic trainer, or registered dietitian) that the athlete trusts and approves of to conduct weigh in procedures, with this information remaining confidential (Bonci et al., 2008; Thompson & Sherman, 2011).
2. Conducting the weigh in or body composition assessment privately, separate from other team players or team staff (Bonci et al., 2008), as well as ensuring that weight data is confidential to sports medicine personnel and treatment team members only.
3. Discourage self-weighing. Preoccupation with weight can be a symptom of the ED itself, thus increasing the risk of engaging in disordered eating or exercise behaviour.
4. Identify the optimal frequency of weighing or body composition assessment, taking into account the balance of benefits or necessity, alongside risk factors.

Barriers to implementing SEES-A

We recognise several potential barriers to the implementation of the SEES-A guideline as well as those related to managing safe return to sport 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-A 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-A 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 professionals 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-A guideline aims to alleviate some of these historical tensions and help facilitate the process of formalising safe exercise prescription into ED treatment for athletes.

Ambivalence 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. Additionally, modified exercise for athletes may come at the cost of scholarships, financial loss, and missed opportunities further disincentivizing adaptations to training. Consequently, athletes may feel ambivalent about reducing their exercise engagement. Both the development of a collaborative client-practitioner relationship (Geller et al., 2003), as well as the psychological interventions highlighted here and in the original SEES document (Calegoro & Pedrotty, 2004; Schlegel et al., 2015; Taranis et al., 2011), may be helpful in addressing ambivalence.

Sporting environment and culture. Several unique aspects of the sporting environment are ripe for the development and maintenance of EDs. Firstly, many qualities of a "good athlete" overlap with characteristics that may promote ED symptomatology, particularly in AN (Thompson and Sherman, 2011). These factors include mental toughness/asceticism, mental commitment to training, pursuit of excellence, selflessness, overcompliance to instruction, and performance in spite of pain. The line between ED pathology and being a "good athlete" can easily become blurred and make it difficult for treatment teams to help change the culture in sport, as well as help the athlete reframe their mental processes around these characteristics (Thompson & Sherman, 2011). These same attitudes can prevent athletes from asking for support with their eating for fear of having to give up their sport or let down sporting team members.

Each of these characteristics can become further amplified when we consider other risks present in higher level sports, such as extremes of training, pressure to perform, public nature of sport, media, and finances (Voelker, Petrie, Reel, Gould, & 2018). Pressures inherent to competitive sporting environments also make it challenging to parse out typical training expectations, mandated by coaches and upheld by disciplined athletes, from dysfunctional exercise in the service of the ED (Voelker et al., 2018). The combination of these factors may result in a hasty return to sport and competition, further complicating treatment.

Lastly, unhealthy messaging that promotes thinness/leanness for enhanced performance and presumes that good performance indicates good health, further complicates the recovery

environment for the athletes (Thompson & Sherman, 1999b). Such cultures contribute to the normalisation of indicators of medical risk such as amenorrhea, fracture risks, and pubertal delays, and may overlook these as signs of low energy availability and/or an ED. Particular sports, such as lean/aesthetic and weight class sports, may require additional psychoeducation and support to prevent these forms of messaging.

Stigma in Sport. Individuals with ED are often ambivalent to treatment and change, in particular those with AN (Fairburn, 2000; Fairburn, Cooper, Doll, Norman, & O'Connor, 2003). However, early identification, intervention, and change with treatment are crucial influencers of a positive prognosis (Baum, 2006; Fairburn & Harrison, 2003). Young athletes report perceived stigma in the sporting community most important barrier to help seeking (Gulliver et al., 2012). Further, normalisation of disordered eating and exercise behaviours can contribute to delays in recognising these behaviours as dangerous. These factors contribute to ED detection in athlete populations, particularly in male athlete populations, being highly challenging (Baum, 2006). Stigma can stem from outside (e.g. relationship, context, environmental) sources as well as an individual's self-stigma (McArdle, Meade, & Moore, 2016). Sporting environments such as elite settings, normalizing, disordered eating practises can evoking secrecy particularly "good athlete" (see page.) who are mentally tough, can work to stigmatize the ED further (Papthomas, 2018). The sporting environment may amplify feelings of stigma and, subsequently, reluctance to seek help in athletes due to the greater pressures, and losses they may encounter as a result of revealing their ED. Further, athletes who are often perceived as tough and strong, may perceive help-seeking or attending therapy as threats to their athletic self-perception (Thompson & Sherman, 2011). Perceptions about confidentiality of disclosure, impact of treatment on sport, as well as the likelihood of recovery are key environmental factors increasing stigma in athlete populations (McArdle et al., 2016). Additionally, stereotyped beliefs (e.g. related to ideal female body types or weight focus performance) by coaches were associated with delays to identification of ED symptomatology, thus, leading to delays in intervention and treatment (Plateau, McDermot, Arcelus, & Meyer, 2014). Overall, there are several environmental factors that may impact the perception of ED in athletes, however, having a safe environment to seek help is important to the treatment of EDs (McArdle et al., 2016).

Hasty return to sport. Although returning to sport and competition is often a key motivation for athletes recovering from their ED, re-entering training or competition too early has been associated with increased risk of relapse (De Souza et al., 2014). This risk may be reduced through developing a clear treatment contract (see pp. 21) for athletes to gain a clear understanding of treatment progression. Despite the need for a paced return to competition, that may be regressed if symptoms reoccur, this progression can be frustrating for athletes and trigger pathological thoughts as outlined below (Conviser et al., 2018). Further, Conviser and colleagues (2018) recommend that mental health providers support the athlete to foster emotional coping skills, distress tolerance, self-awareness and self-esteem. Finally, encouraging help-seeking behaviours and identifying trusted individuals in the athlete's support network may promote reaching out to social supports (Conviser et al., 2018).

The Safe Exercise at Every Stage-Athlete Guideline

Instructions for use

The Safe Exercise at Every Stage - Athlete (SEES-A) guideline was developed to better facilitate clinical decision-making related to safe return to exercise, training, and sport for athletes with ED symptomatology. This step-up/-down model involves three 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 prescription (prior to return to training in Component 3) related to the level of risk identified in Component 1.

3. Return to competition: Once Component 1 markers are cleared, clinicians will assess psychological, performance and environmental factors to inform the level of the athlete's training and competition. Clinicians may also find it useful to refer to RED-S CAT during this stage.

This guideline does not replace clinical judgment, but rather augments the ethical and clinical decision-making process. Clinicians must regularly review clients' medical and psychological progress as recommended in the guideline (i.e. weekly in Level A, decreasing in relation to risk). Additionally, psychological intervention should occur concurrently to exercise and nutrition interventions to best support clients.

Importance of Safe Exercise at Every Stage – Athlete

Graded exercise can be safely undertaken during ED treatment to achieve positive outcomes such as improved eating disorder symptomatology, general psychological well-being, musculoskeletal health and cardiac functioning as well as increased meal plan and treatment adherence. 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-A guideline

This guideline was developed to support clinicians in making safe, evidence-based decisions when recommending exercise and return to sport for athletes with EDs. Exercise and training sessions must be supervised initially with increasing autonomy permitted as treatment and recovery progress. Please note that it is not a requirement, nor always possible, for treatment or coaching team member to supervise these sessions. In these circumstances, we recommend that a trusted friend or partner with knowledge of the athlete's individualised exercise plan and limitations be present. Regular medical reviews are required to decide whether the current exercise is maintained, progressed, or regressed, depending on client symptomatology and physiological results.

Part A – Safe Engagement in Exercise

1. Assessment: Use psychological and physiological results (as per page 28) to determine your client's level of risk for engaging in exercise. Always begin the assessment using the markers from the highest-risk category, Level A. If no Level A risk factors are present, progress to 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-D), match this with the corresponding exercise recommendation on page 39. Please note that regardless of which risk level an individual is assessed at initially, clinicians are still encouraged to continue education interventions from prior levels. 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) in their risk level throughout treatment and recovery. Consequently, regularly reviewing (e.g. weekly, monthly) the athlete's risk level is vital. Stepping up requires not only clearing all risk markers up to and including their current level, but also adhering to treatment recommendations, achieving adequate nutritional intake, and exhibiting improvements in health status, where necessary. 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 in the case of treatment/meal plan non-compliance, return to exercise compulsions, or a worsening of ED behaviours.

Intensity, Duration and Type of Exercise

The SEES-A guideline provides recommendations regarding the intensity, duration and type of exercise, however, deliberately does not specify the frequency of exercise sessions per week. Clinicians and athletes are to determine this collaboratively to prioritise safety, minimise harm, and optimise treatment outcomes. The frequency of training in Part B below will also require consideration of periodization (i.e. strategic implementation of training phases often used in athletics; Bompa & Buzzichelli, 2018), with training phases based upon increasing or decreasing the intensity and volume of exercise within a program.

Exercise is a positively indicated treatment component but is not compulsory and boundaries are important to prevent engaging in 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 better characterise an individual's total daily energy expenditure in relation to energy intake.

Part B – Return to Sport

1. Assessment: Use psychological, environmental and performance markers (pp. 30) as well as sport specific recommendations (pp.32-36) to determine your client's level of risk when returning to sport. Always begin the assessment using the markers from the highest-risk category. If no risk factors from Stage 1 are identified, assess the measures in Stage 2, and so on.

2. Recommendations: Once the level of participation risk has been identified (e.g. Stage 1-4), match with the corresponding participation recommendation on the top of the recommendations table (pp.31). Please note that even once an individual positively progresses past Stage 1, clinicians are still recommended to continue interventions from this level as they include important education regardless of health status. This continues to apply at Stage 4, whereby interventions from Stage 1 through 3 should continue to be implemented.

3. Step up/step down: Individuals may step up and down on the SEES-A guideline throughout treatment as often we find that recovery is not linear. The assessment (pp.30) and recommendation (pp.31) tables are meant to represent a continuum and if criteria are not met, the individual can step down from the SEES-A guideline (pp.30-31) back to the SEES tables (pp.28-29). 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.

Limitations

This guideline does not replace clinical judgement by the treatment team. It has been developed for the use of trained medical, exercise, and sporting professionals with expert knowledge in the physiology of eating disorders when working with an adult athlete population (aged 18 years 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 74) 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): 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, Purposeful Dehydration, and Hypovolemia,* pp.70).

Increasing progression with nutritional plan

Level A Review weekly	Level B Review fortnightly	Level C Review monthly	Level D Review as required
Cardiovascular profile: Resting HR <44bpm or >120bpm Postural tachycardia >20bpm Orthostatic hypotension >20mmHg systole (independent of symptoms) Systolic BP <90mmHg Prolonged QT/c interval >450msec Arrhythmias Valve ventricular disproportion **Biochemical profile:** Hypokalemia <3.0mmol/L Hypophosphatemia <0.8mmol/L Hypomagnesemia <0.7mmol/L Hypercarbia>32mmol/L Hyponatremia <130mmol/L Hypoglycaemia <4mmol/L Hypoalbuminaemia <3.6g/ml hypovolemia **Psychological profile:** EDAS score > 2 for three or more subscales **Other:** Temperature <35°C	*Individual has cleared all prior risk markers and is also adhering to:* **Individuals with AN:** Positive weight gain trajectory in line with treatment goals **Weight-restored individuals:** Weight stabilisation/mobilisation in line with treatment goals **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 **Extra note:** Individuals with iron deficiency anaemia should consider a reduction in weight-bearing/ jogging/running/jumping on hard surfaces	*Individual has cleared all prior risk markers and is also adhering to:* Weight stabilisation or gain if still required Level A markers related to ED are completely normalised as per medical recommendation Managing ED behaviours (e.g. self-induced vomiting, restriction/ bingeing, fear of becoming fat, & laxative use) Normalised sex hormones without exogenous replacement (return to menses & normalized oestrogen for females; testosterone for males) **Psychological profile:** Improvement in EDAS scores ***SEES-A: Once above is met progress to SEES-A Stage 1 non-contact/low-impact sport or Level D for contact/high-impact sport***	*Individual has cleared all prior risk markers and is also adhering to:* Weight progression >90% of IBW (considering individual weight history & family characteristics) ***SEES-A: Once above is met, progress to SEES-A Stage 1 for contact/ high-impact sport***

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 Nutritional rehabilitation and counselling Ambulation assessment & injury prevention in daily living tasks (e.g. correct bending technique) Breathing & body awareness tasks Introduction to body awareness Assessment of exercise habits prior to treatment & long-term exercise goals Physiological education	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

Stage 1	Stage 2	Stage 3	Stage 4
Athlete has cleared all prior risk markers (including SEES Level C if non-contact/low-impact sport or D if contact/high-impact sport) and demonstrates competency in:	*Athlete has cleared all prior risk markers (including Stage 1 and SEES Level D) and demonstrates competency in:*	*Athlete has cleared all prior risk markers (including Stage 2) and demonstrates competency in:*	*Athlete has cleared all prior risk markers (including Stage 3) and demonstrates competency in:*
Physical Adequate nutrition and hydration for training load No presence of overtraining syndrome No ECG abnormalities Resolution of stress fractures, no new fractures At least 6 menses over last 12 months for females Absence of recurrent upper respiratory tract infections during Level C or D	**Physical** Passes team fitness/performance test without experiencing adverse **physical** outcomes prior to, during, or after testing	**Physical:** Engaging in competition without symptom regression Maintenance or improvements in musculoskeletal, cardiorespiratory/vascular, neurological and metabolic fitness testing as per team expectation	**Physical** Maintenance of Stage 3.
Psychological Includes regular rest days from any exercise Maintenance or improvement in EDEA score Adherence to individual exercise plan from Level C & D Abstinence from fasting and purging Demonstrated maintenance of minimum 95% IBW Engaged in valued actions outside of sport (e.g. education, non-sporting hobbies, social activities)	**Psychological** Passes fitness/performance test without experiencing adverse **psychological** outcomes prior to, during, or after testing		**Psychological** Maintenance of Stage 3.
Training-related Ability to tolerate and adapt to unexpected change in exercise or training Engaged in a variety of exercise/training types Process-oriented professionalism/decision making (e.g. following injury protocols; accept unplanned rest day or meal plan changes if fatigued) Demonstrated ability to alter planned exercise		**Training-related** Compliance with pre- and post-competition changes to meal plan, hydration, and training	

Increasing progression with nutritional plan

Stage 1	Stage 2	Stage 3	Stage 4
Return to sport intervention: Individual training/practice (tailored to Level C restrictions i.e. 30 minutes, functional body weight, etc.)	**Return to sport intervention:** Return to team training/practice (if applicable)	**Return to sport intervention:** Intermittent* competition	**Return to sport intervention:** Normal competition and periodization of training
Supervision: Sporting or treatment team personnel during sport related activity	**Supervision:** Sporting and/or treatment team personnel during sport related activity	**Supervision:** As per Stage 2	**Supervision:** As per Stage 2
Education interventions: • Identify and address dysfunctional beliefs associated with exercise, training, nutrition, rest, and competition • Identify and address barriers in return to sport (e.g. competition pressures, team environment) • Practice awareness of physical cues before, during, and after training (i.e. in real-time/during training with the strength and conditioning coach or when training alone) • Evaluate short- and long-term sport, wellbeing, relationship, career, and other life goals • Develop sense of self and identity outside of sport • Nutrition/behaviour/training log	**Education intervention:** • Continue relevant/outstanding interventions • Identify and address any increases in performance or appearance comparisons • Complete exposure tasks related to competition aspects (e.g. competition attire, public weigh ins) **Preparations for returning an athlete to competition:** • Identify sport-specific demands (e.g. eligibility, competition weight, training requirements) • Consider sports' governing body requirements (e.g. whether adaptations to any sporting protocols may be acceptable)	**Education intervention:** • Relapse prevention • Continue outstanding interventions *Frequency will be collaboratively determined by the athlete, treatment team and sporting team (considering sport-and season-specific factors)	**Education intervention:** • As per Stage 3

Symptom regression, decreased fitness/performance markers, treatment/meal/hydration plan noncompliance, return to exercise compulsion, or repeated deviation from training plan

Sporting Considerations

Performance

Optimising performance is a key motivator for athletes, as evidenced by their years of dedication to training and willingness to sacrifice other aspects of their life such as relationships and schooling for their sport. Understandably then, athletes are driven to enhance their sporting performance, even if their methods are associated with negative psychological and/or physical outcomes. Increasing lean musculature and decreasing overall weight is a common approach for athletes trying to improve their performance, however, this may precipitate disordered eating and/or exercise behaviours (Sundgot-Borgen & Torstveit, 2010). Further, self-directed attempts to change body composition can backfire, detrimentally impacting fat-free mass, metabolic function, strength, and performance (Sundgot-Borgen & Garthe, 2011; Trexler et al., 2017). For example, inadequate protein can lead to muscle weakness, wasting and injury; while, a lack of carbohydrates can result in early glycogen depletion, fatigue and utilizing protein (instead of glycogen) for energy, as well as negative consequences to the HPA axis and associated hormones contributing to severe health consequences (Nickols, 2020). Further, dehydration can lead to fatigue, cramping, nausea, reduced exercise capacity, and depletions in glycogen stores (El Ghoch et al., 2013; McArdle et al., 2010). These behaviours may also contribute to the development of Relative Energy Deficiency in Sports (RED-S) syndrome. RED-S was first identified by the International Olympic Committee (2014) to highlight the negative health and performance consequences of continued sporting engagement in the presence of inadequate nutrition and/or overtraining. Driven by LEA (pp.40), RED-S is associated with negative mental health outcomes (such as poor concentration, irritability, depression, and impaired judgement that may predispose to disordered eating), physical health outcomes (decreased glycogen stores, impaired gastrointestinal function, low bone density, and increased injury risk), as well as performance outcomes (poor coordination and decreased endurance, muscle strength, and training response; Mountjoy et al., 2014). In fact, athletes with EDs with RED-S were twice as likely to sustain a musculoskeletal injury during the competitive season (Thein-Nissenbaum, Rauh, Carr et al., 2011).

Achieving peak sporting performance requires a complex interplay of physical and mental health components (El Ghoch, Soave, Calugi, & Riccardo, 2013). This requires athletes to build a positive relationship with their sport and training (Fewell et al., 2018), learn skills in effective goal setting, challenge unhelpful cognitions, and develop constructive emotional coping techniques (Conviser et al., 2018) in adjunct to achieving sport-specific fitness markers and developing a healthy relationship with food and body image. While an attitude of mental toughness and relentless pursuit of perfection is often glorified in athletes (Thompson and Sherman, 2012), a recent study demonstrated that self-compassion, not self-criticism, was positively associated with sporting performance (Killham, Mosewich, Mack, Gunnell & Ferguson, 2018). As attitudes regarding perfectionism and excessive self-criticism may extend past the individual athlete to permeate team culture, it is important to support sport personnel in promoting mental resilience within their athletes (Madigan, Stoeber,& Passfield, 2017).

Physical fitness is evidently a large component of performance, however, an ED can impact an athlete's cardiovascular fitness, endurance, and muscle strength even after initial weight restoration. Fitness testing may support athletes recovering from an ED and their treatment team to set individualised markers of returning to health and sport readiness (McCallum et al., 2006; Quesnel et al., 2018). This testing may include basic assessments measuring the sum of morphological fitness such as bone strength, muscular fitness, flexibility, motor, cardiovascular and metabolic fitness (El Ghoch et al., 2013). These results may be used to determine baseline markers for those returning to training after an ED diagnosis, as well as to measure progress in various areas of physical health and performance (Fewell, Nickols, Schlitzer Tierney, & Levinson, 2018). Fewell and colleagues (2018) provided an overview of a protocol for safe fitness testing and how assessments relate to ED-specific markers. In this study, Fewell and colleagues (2018) found that evaluating markers of functional stress during treatment was associated with athlete's improved confidence in their nutritional plan, acceptance of recommended weight goals, and adherence to fitness recommendations (Fewell et al., 2018).

Sport Type

Demands of different sporting categories are inherently related to ED risk, recovery, and prognosis. Additionally, sporting type may be associated with differing LEA risk, based upon the physical demands of the sport, e.g. endurance athletes may be at higher LEA risk as both mental and physical tolerance activity develops (Di Lodovico, Poulnais, & Gorwood, 2019). However, multiple studies have struggled to find associations between sport type and dysfunctional exercise (De La Vega et al., 2016; Maselli et al., 2019; Szabo et al., 2016) indicating that individual, cultural, and environmental factors inherent to each sport may protect against or promote ED.

Lean/body focused. The following section will review the unique risks associated with lean sports, referring to sports where "a thin or lean body or a low weight are believed to provide an advantage in sport performance" (Thompson & Sherman, 2011, pp.32). Lean sports include the sub-categories of aesthetic sports, weight-dependent/class sports, and endurance sports. Research has shown higher rates of EDs in lean sport athletes compared to non-lean sport athletes, for example one study reported 38.2% of male lean sport athletes in their sample engaged in dysfunctional exercise, whereas only 13.8% of non-lean male athletes did so (Dimitrova & Vanlyan, 2019). Together these three sporting types carried the highest risk for the use of pathological weight management behaviours (Sundgot-Borgen, 1993; Sundgot Borgen et al., 2011)

Weight-dependent/class sports. Weight class sports rely on athletes achieving a certain weight range prior to competition as a requirement for participation (e.g. judo or wrestling, boxing, lightweight rowing; Sundgot-Borgen & Garthe, 2011). While weight loss during the competitive season is common, with athletes in non-weight class sports losing an average 3-6% of total body weight; athletes in weight class sports lose on average 13% of body weight during their season (Sundgot-borgen & Garthe, 2011). Unfortunately weight manipulation through dehydration and/or dieting and unsafe weight loss methods (e.g. diet pills, laxatives, enemas) are common in these sports (Pallares et al., 2016; Samadi et al., 2019; Sundgot-Borgen et al., 2013), despite evidence that engaging in these behaviours can impede health and performance even in the absence of an ED (Drinkwater et al., 2005; Nattiv et al., 2007, Slater et al., 2005a).

Some studies have estimated as many as 94% of athletes in weight-dependent sports engage in extreme weight control methods prior to competition (Sundgot-Borgen & Garthe, 2011), putting athletes competing in these sports at high risk of developing an ED (Rosendahl et al., 2009). Particularly, athletes in lighter tiers of weight categories are at greater risk for engaging in unhealthy weight management behaviours than those in higher weight categories. These behaviours can lead to a higher incidence of the female athlete triad, dehydration and life-threatening electrolyte imbalances, low bone mineral density, and osteoporosis (Smather, Bemben & Bemben, 2009: Sundgot-Borgen et al., 2011) as well as RED-S syndrome (Mountjoy et al., 2014). Further, research has indicated that 30-35% of females, and 17-18% in males weight class sport athletes meet diagnostic criteria for an ED, compared to 5% and 16% in ball/game sports or 4% and 17% for technical sports in males and females respectively (Sundgot-Borgen & Torstveit, 2010). Consequently, athletes with an ED intending to return to competition in these sports must be medically safe to return to sport but also mentally and physically prepared to return to the weight-related pressures and practices inherent in these sports.

Aesthetic sports. Another form of lean sports, aesthetic sports are judged based upon an athlete's physicality and appearance, as well as their technicality and artistry (Prelack et al., 2012; Thompson & Sherman, 2011). These include gymnastics, competitive dance/cheer, diving, figure skating, synchronized swimming, and bodybuilding. These sports often promote a lean physique, however, pressures to achieve this build can be in conflict with ensuring adequate nutrition to maintain sporting performance (Prelack et al., 2012), placing these athletes at heightened risk for both LEA and EDs. Additionally, aesthetic sports often require uniforms that may exacerbate weight and appearance concerns in these athletes, as well as promote unhealthy body comparisons between athletes (Rel, Soo Hoo, & Estes, 2005; Thompson & Sherman, 2007; 2011). Overall, athletes in aesthetic sports are more likely to report greater drive for thinness and ED psychopathology than non-aesthetic athletes (Schwarz, Gairrett, Aruguete, and Gold, 2005). Both social pressures to be lean as well as the desire to increase leanness for performance have been identified as predictors of disordered eating in aesthetic athletes (Krentz & Warschburger, 2011; 2013), highlighting the need for education regarding the impact of inadequate nutrition on performance as well as supportive team messaging.

Endurance sports. The category of endurance sports refers to aerobic activities requiring substantial and sustained energy (Maselli et al., 2019; Morici et al., 2016) including triathlon, cycling, distance running, and swimming. Endurance sports are associated with an elevated risk of EDs (Sundgot-Borgen & Torstveit, 2004; Trostveit et al., 2008) and are commonly abused by non-athletes engaging in dysfunctional exercisers (Cook & Luke, 2017). Given the length and energy demands of endurance sports, studies report higher incidence of female athlete triad symptoms (Mudd, Fornetti, & Pivarnik, 2007), exercise dependence (Cook & Luke, 2017), as well as RED-S syndrome in endurance athletes (Torstveit et al., 2019). High intensity running in particular has been related to delays in gastric emptying, potentially due to the dyspnea encountered by athletes with EDs (Leiper, Nicholas, Ali, William, & Maughan, 2005). Additional risk factors include endurance athletes exhibiting a high athletic identity and male athletes with comorbid narcissism, both of whom are more likely to engage in dysfunctional exercise (Nogueira, Tovar-Galvez, & Gonzalez-Hernandez, 2019; Turton, Goodwin & Meyer, 2017). Consequently, it may be particularly important for treatment teams to screen endurance athletes

for dysfunctional exercise alongside EDs.(using the EDEA pp.69, or similar measures adapted for athletes such as the Compulsive Exercise Test-Athlete).

Returning to lean sports. Despite the inherent risks associated with lean/body-focused sports, athletes who experience EDs may wish to return to their sport. This will require discussions between the athlete, treatment team, and sporting team about how to use safe, healthy behaviours to achieve sport-related physique goals, as well as whether these goals are realistic given the athlete's history. Additionally, it is key to identify and address concerns about returning to sport including strategies for coping with body image (particularly when training with mirrors and/or wearing revealing sporting attire), dealing with social comparison and weight-talk with peers, ensuring nutritional adequacy, and preventing compulsive exercise (Conviser et al., 2018).

The athlete, treatment team, and sporting team may also need to discuss changes to the athlete's weight category. These changes must consider the athlete's natural phenotype including their build, body shape, weight and size (when engaging in healthful eating and training behaviours) and take steps to work with the athlete to improve athleticism within those parameters (Oppliger et al., 2003). Athletes should be engaged in this conversation early and sensitively. Important aspects of this conversation may include helping athletes to appreciate the strengths of their natural phenotype for certain sports and providing education on medical and psychological risks associated with unhealthy body change behaviours, as well as performance benefits of maintaining a healthy and sustainable body size.

Ball/game sports. Ball/game sports include volleyball, hockey, softball, football, and soccer. There has been less research examining ED symptomatology in these sports; however, recent studies have found athletes in ball sports also report body dissatisfaction and disordered eating (Torstveit & Sundgot-Borgen, 2005; Wright, Ford, & Bothra, 2014) as well as a heightened drive for muscularity (Stewart, Benson, Michanikou, Tsiota & Narli, 2003; Raudenbush & Meyer, 2003), and LEA (Wright et al., 2014). Furthermore, the general sporting environment and competitive sport engagement, irrespective of sporting type, is still associated with several factors that increase risk for EDs and potentially impairing recovery such as promotion of selflessness and performance in spite of pain or for the team, overcompliance to instruction, pursuit of excellence, and the normalisation of both injury and diet culture (Thompson & Sherman, 2011).

Team sports vs individual sports. Stages of return to sport may differ between athletes competing in individual sports versus those in team sports. Overall, there does not appear to be a difference in prevalence of EDs between athletes from individual and team sports (De La Vega et al., 2016; Maselli et al., 2019). Team sports may present environment-specific risks for athletes due to greater prevalence of obsessive passion (a risk factor for dysfunctional exercise in athletes) as well as greater pressure to return to sport "for the team," requirements to participate in team weigh ins, and role modelling of disordered eating and/or dysfunctional exercise behaviours by teammates (Arthur-Cameselle et al., 2018; De La Vega et al., 2016; Galli, Petrie, & Chatterton, 2017; Scott, Haycraft, & Plateau, 2019). Yet, team sports may also possess characteristics protective against ED development such as social support, positive modelling from teammates, and accountability in treatment and recovery from peers (Arthur-Cameselle et al., 2018; Arthur-Cameselle & Curcio, 2018; De La Vega et al., 2016; Scott et al., 2019; Smolak et al.,).

Due to the potential benefits of the team environment, Thompson and Sherman (2011) recommend that athletes with EDs should remain part of the team, when willing and able. This involvement may include attending functions, team meetings, and practices/training (even if unable to participate in training). Participation in team activities may support athletes with EDs to maintain their athletic identity, which is related to positive self-worth (Thompson & Sherman, 2011), and can mitigate social isolation and declines in psychosocial well-being. Additionally, this attendance may facilitate the sporting team in monitoring and supporting the athlete's return to sport goals and treatment motivation. We do note that some athletes may struggle with continued team participation whilst sidelined for ED treatment and that some team environments may not be conducive to ED recovery; in these cases, athletes may postpone participation in team events until a later stage of recovery.

Level of Competition

An athlete's level of competition is an important consideration when determining readiness to return to competition and training. Elite levels of competition are associated with greater pressure, expectations, and more competitive environments. Although some studies have indicated greater use of unhealthy weight management strategies and malnutrition in elite athletes when compared to recreational or amateur athletes (Meng et al., 2020; Pallares et al., 2016), others have indicated mixed results regarding the influence of sporting level on the severity and presence of ED psychopathology (Thompson & Sherman, 2011). Consideration for returning an athlete to sport will include the demands of the specific sport, the sport governing body, and whether any required adaptations to sporting protocols may be acceptable.

Injuries

Athletes with an ED are more likely to experience injuries than non-athletes with EDs, with up to 40% of athletes reporting a stress fracture (Snyder, Koester, & Dunn, 2006). And while experiencing an ED or LEA increases injury risk, sustaining an injury may also precipitate disordered eating or ED onset (Arthur-Cameselle, Sossin, & Quatromoni, 2017). Risk of injury can be exacerbated by training, experiencing RED-S syndrome or female athlete triad, low bone mineral density, low muscle size, and fatigue with stress fracture risk, in particular, also related to engagement in DE and low lean tissue mass in legs (Duckham et al., 2012).

Health and Performance Complications: Unmodified Exercise Training with an Eating Disorder

The following section reviews *health and performance complications* related to eating disorders that may contraindicate or detrimentally impact unmodified exercise engagement and training. In addition, Table 1 (pp. 71) lists signs and symptoms that may contraindicate or impact exercise engagement. These complications are consistent with those experienced by non-athletes (as per the original SEES document), however, additional concerns specific to the athlete population have now been added. For further information concerning the importance of macronutrients (i.e. carbohydrate, protein and fat), in addition to supplements in for athletes, see the joint position statement on nutrition and athletic performance by the *American College of Sports Medicine, Academy of Nutrition and Dietetics* and *Dietitians of Canada* (Thomas, Erdman & Burke, 2016).

1. Low energy availability

Research examining a triad of symptoms including EDs/disordered eating, menstrual dysfunction, and low bone mineral density (female athlete triad) identified that numerous deleterious health outcomes in both male and female athletes stemmed from inadequate energy availability or LEA (Nattiv et al., 1994; Nattiv et al., 2007). LEA occurs when an individual's energy intake is insufficient to meet their energy expenditure, related to their fat free mass (FFM; Loucks, 2014). Studies estimate that LEA is likely 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), may be experienced in both the short and long term (Thomas, Erdman & Burke, 2016), and can also occur both in the presence and absence of an ED. For a thorough review see Melin and colleagues (2014). 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)

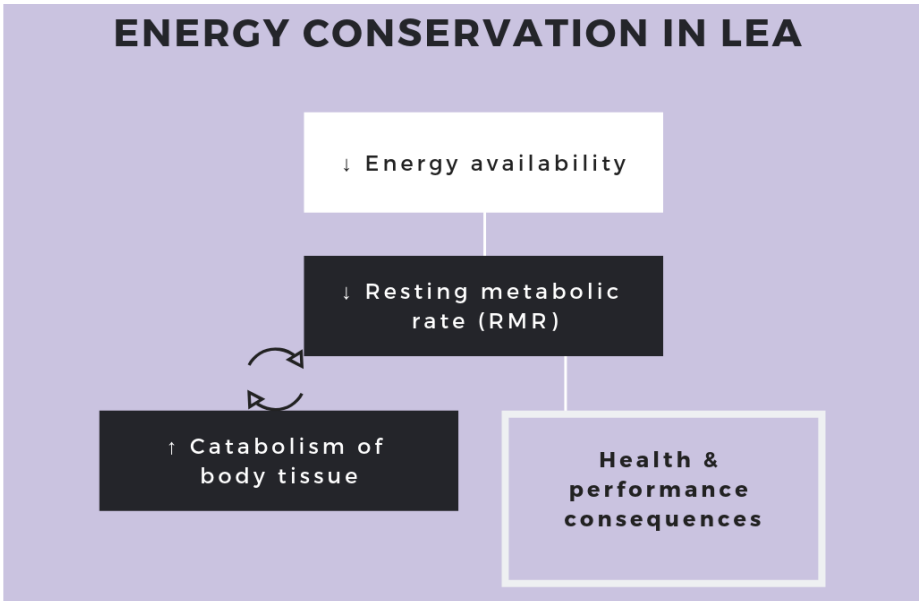
Where:

- Optimal EA (OEA): at least above 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 FFM, such as muscles, bones and organs (Woods et al., 2017). 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 can cause 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. This psychological distress is also a significant contributor to the increased risk for ED in athletes with LEA. 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 state

Unfortunately, despite the consequences of LEA, measuring it can prove challenging for many reasons (Burke, Lundy, Fahrenholtz, & Merlin, 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). Thomas, Erdman & Burke (2016) suggest that providing education to athletes regarding food monitoring and its benefits may support compliance, accuracy and

validity of the documentation of food intake. Yet, the utility and reliability of self-reported food intake information is questionable due to its subjective and potentially biased nature. The Low Energy Availability in Females Questionnaire (Melin et al., 2014) and RED-S Clinical Assessment Tool (Mountjoy et al., 2015) may also facilitate screening athletes at risk for LEA.

The energy requirements of elite athletes differ from sedentary individuals regarding the amount of energy expenditure from exercise. Manore and Thompson (2014) note that while the resting metabolic rate (RMR) of sedentary individuals comprised a large proportion (60-80%) of their total energy expenditure, athletes expended most of their energy due to the thermic effect of activity. Thomas and colleagues (2016) also emphasise that if an athlete is experiencing low energy availability, it does not necessarily mean their total energy balance is also low or that weight loss will definitely occur. However, if energy availability has been lowered to the point of affecting the vital function of the RMR, the body may be forced into 'survival mode', where it can become 'sluggish' in an attempt to maintain an energy balance or safe weight (Thomas, Erdman & Burke, 2016).

As most athletes have an on and off-season, with periodisation of training common for many elite sportspeople, energy requirements should not be static and must be flexibly adapted to the athlete's daily requirement given their current training and competition engagement (Thomas et al., 2016). Energy needs may be increased by training load, heat/cold, high altitude exposure, fear, stress, physical injuries, specific drugs or medications (e.g., caffeine, nicotine), increases in fat-free-mass and, potentially the luteal phase of the menstrual cycle (Thomas et al., 2016). These same requirements may also reduce due to decreased training load, ageing, decreased FFM, and, potentially, the follicular phase of the menstrual cycle. (Spriet, 2014). LEA and its associated RED-S syndrome (Mountjoy et al., 2014) contribute to many of the upcoming conditions that may contradict exercise, worsen performance and endanger both physical and mental health.

2. Cardiovascular health

Potential cardiovascular contraindications to exercise

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

- Tachyarrhythmia 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.

Box 4. Potential cardiovascular contraindications to exercise

Cardiovascular Training Adaptations

Cardiovascular complications in athletes, such as sudden cardiac death, are generally uncommon and typically only occurs in states of extreme exhaustion and exertion (Maron et al., 1995). However EDs augment the risk of adverse cardiac events due to the impact of malnutrition in athletes engaging in inappropriate levels of activity (Casiero & Frishman, 2006). Athletes can have electrical, structural and functional cardiac adaptations known as "the athletes heart" resulting from high levels of training and overall fitness, which may skew ECG and pathophysiological testing and monitoring, increasing the likelihood of misinterpretation of findings and unnecessary sport cessation (Maron, Pelliccia, & Spirito, 1995; McArdle et al., 2010; Lai & Chung, 2017). A study of over 1000 athletes demonstrated that about 60% of the sample had normal ECG or only had minor alterations, with 40% having some type of cardiac remodeling. In this sample, the most frequent abnormalities were mitral valve prolapse with mild regurgitation (n=19) and a bicuspid aortic valve with regurgitation (n=10); with less common defects including atrial or ventricular septal defect (n=6), dilated cardiomyopathy (n=4), mild pulmonary artery stenosis (n=2), and myocarditis (n=2) (Pellica et al., 2000). Common ECG changes include sinus bradycardia, sinus arrhythmia, first-degree atrioventricular block and incomplete right bundle branch block; which may not be problematic so long as the athlete is asymptomatic or does not produce cardiac pauses longer than 4 seconds (Lai & Chung, 2017; Zehender, Meinertz, Keul, Just, 1990). Structural and functional changes can include increased left ventricular wall thickness, increased right and left ventricular cavity size, bi-atrial dilatation, increased diastolic filling and increased stroke volume (Lai & Chung, 2017).

HCM, or the thickening of the heart, is the most common cause of sudden cardiac death in athletes. In athletes without EDs, training can lead to increased left ventricular diastolic cavity dimension wall thickness to be 53 to 58 mm thick (Maron et al., 1995) which can cause confusion between HCM and an athletic heart. Additionally, other illnesses such as Dilated Cardiomyopathy, Myocarditis, Right Ventricular Dysplasia can also mimic HCM (Maron et al., 1995). Cardiac wall thickening is proposed to be more prevalent in endurance athletes (Maron et al,. 1995), with those involved in rowing and cycling typically exhibiting greater wall thickness, while, those in power sports such as weight lifting or wrestling not typically having a wall thickness beyond 12 mm (Pelliccia et al., 1991). Training typically evokes a mild increase of equal or less than 12 mm, up to 16 mm (Mauron et al., 1995). Having a left ventral wall above 55 mm occurs in about one third of highly trained male athletes, and wall thickening is generally rare in women (Pelliccia , Maron , Spataro, Proschan, & Spirito, 1991). In athletes, distinguishing between HCM and an athletic heart can be informed by a variety of ECG alterations, including striking increases in voltages, prominent Q waves or deep negative T waves. In an echocardiography, athletes without HCM show normal Doppler diastolic indexes of left ventricular filling independently of whether symptoms or outflow obstructions are present.

Screening for cardiac abnormalities is an important measure of ensuring safety for athletes during the course of ED recovery and return to sport (Currie & Crosland, 2009; Mountjoy et al., 2015). Complex ventricular forms of arrhythmia should prompt cardiac examination as they may indicate increased risk of cardiac death (Williams et al., 2019; Zehender et al., 1990).

Depending on the level of sport, there are different levels of cardiovascular screening. For all elite athletes, regardless of ED presence, a fourteen lead electrocardiogram (ECG) is recommended as part of the sport participation evaluation (Williams et al., 2019). The physiological changes in ECG in athletes should be considered right/left ventricular hypertrophy, exercise-reversible ST elevation, and exercise reversible changes in T waves (T negativity, shape or excessive T waves are all normal sport adaptations). Horizontal ST segment depression is very rare in athletes and should be followed with further cardiologic examination.

Sherman and Thompson (2011) highlight that the American Heart Association proposed a twelve element screening procedure for the cardiovascular health of athletes (Glover, Glover & Maron, 2007). Although these screening questionnaires can be useful to determine general risk, studies have shown that an ECG out-performs these questionnaires in detecting cardiovascular abnormalities and sport engagement risk (Williams et al., 2019). As a result of the elevated risk of cardiovascular events and death related to EDs, ECG screening for cardiovascular risk is recommended prior to athletes with an ED returning to any team or sporting activities including training.

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).

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; 2016) and is a subtype of arrhythmia (American Heart Association, 2016). Bradycardia associated with high exercise levels is common for athletes, who often record a resting heart rate between 40-60 bpm, with beats per minute recorded as low as 30 (Boyett et al., 2013). Bradycardic arrhythmias in athletes include sinus bradycardia, junctional bradycardia, first-degree atrioventricular block, and Mobitz type I atrioventricular block (Huggins & Woods, 2013). Data suggest these are a result of increased vagus nerve activity. Despite this, bradycardia may also be influenced by the effects of LEA (Spaulding-Barclay, Stern & Mehler, 2016) and advanced malnutrition (Sachs, Harnke,

Mehler & Krantz, 2016), and 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, heart failure, fainting, chest pain and blood pressure issues (American Heart Association, 2016).

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) is considerably more conservative, recommending that any noticeable change in heart rhythm by palpitation or auscultation may contraindicate exercise.

Bradycardia: Fitness or ED?

- Both athletes and individuals with ED commonly exhibit a ↓ HR at rest.
- However, upon minimal exertion (such as sittng 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

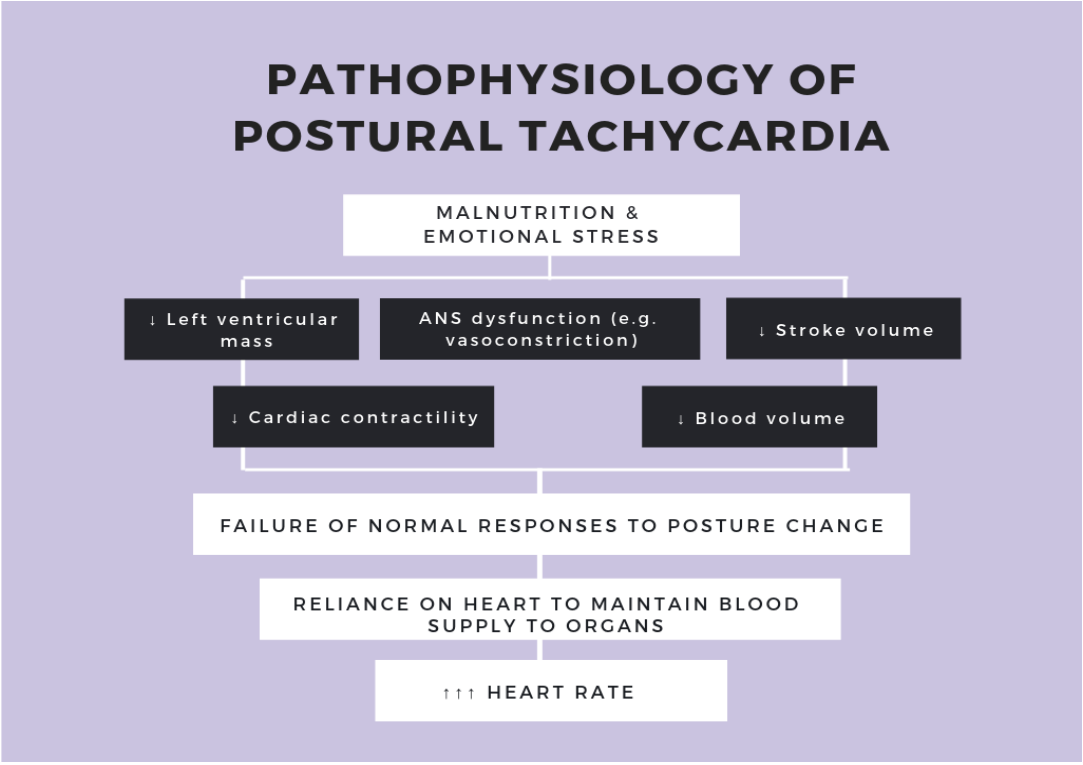
Tachycardia

Tachycardia is defined as a high resting heart rate (>100bpm) and is a second subtype of arrhythmia (American Heart Association, 2016). In athletes, tachyarrhythmias, specifically atrial fibrillation, are more common than in sedentary counterparts, particularly in older athletes (Baggish & Wood, 2011). Atrial fibrillation is speculated to be due to left atrial remodeling and inflammation, increasing sympathetic activity during exercise (Baggish & Wood, 2011). 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 or heart attack (American Heart Association, 2016). With consideration of these consequences, it is essential 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 that the oxygen supply to the brain remains constant (Grubb et al., 2006), PT, particularly in the presence of dysfunctional exercise, 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 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). PT presents as a response to abnormal physiological or psychological influences whereby even a small postural change can cause the heart to overwork result (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 from inadequate cardiovascular and neural responses to standing, allowing 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, light-headedness, 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, insufficient food and fluid intake, and/or purging behaviours (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). Even mild dehydration (represented by a 1-2% loss 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 is recommended to modify exercise engagement until hypotension has resolved (McCallum et al., 2006).

Neurocardiogenic Syncope

Syncope, or the loss of consciousness due to loss of postural tone, is more common in athletes than in the general population. Although syncope may be a result of malnutrition, this is not always the case. In one study examining athletes across 5 years, 6% had experienced syncope, although only 1.6% of participants experienced syncope as a result of neurocardiogenic reasons, and most were attributed to a sudden reduction in venous return (Baggish & Woods, 2011 108). If an individual has a structural or valvular heart disease, appropriate sport restriction may be required. However, neurocardiogenic syncope may be managed by mandating an active cool-down period after exertion and by being mindful of hydration and salt supplementation (Baggish & Woods, 2011).

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).

Valve ventricular disproportion

As a result of an ED, the heart itself can shrink and become weakened as a result of a lack of nutrition. This change has been proposed to result in a disproportion of size between the valves of the heart and the remaining heart, termed valve ventricular disproportion (VD). In VD, there is an increased risk for mitral or tricuspid valve prolapse as well as a potential for decreased exercise tolerance and fitness due to reduced ejection fraction and minute blood volume (Mazurak, ENck, Teufel, & Zipel 2010). Continued exercise in the presence of these symptoms can worsen their states (Thompson & Sherman, 2011).

3. Key micronutrients in athlete populations

Micronutrients (also referred to as vitamins and minerals) play a vital role in maintaining the body's tightly-regulated functions including bone health, immune function, energy production, and metabolic regulation (Gleeson, Nieman, & Pederson, 2004). These may be consumed through regular dietary intake or via supplementation. Athletes with EDs are at risk for micronutrient deficiencies, with iron, vitamin D, calcium and antioxidants being cited as the most common in a joint position statement by the *Academy of Nutrition and Dietetics, Dietitians of Canada,* and the *American College of Sports Medicine* (Thomas, Erdman & Burke, 2016). Additionally, physical training, particularly at the elite athlete level, can exacerbate stress on the metabolic pathways which micronutrients are required to support (Thomas et al. 2016).

Iron

Iron supports multiple important functions including the transport of oxygen to working muscles and other tissues, creation and support of red blood cells, creation of intra-cellular energy, and bolstering the immune system (SDA [Iron], 2020). Iron deficiency may be particularly pertinent and common in athlete populations (Castell, Stear, & Burke, 2015). This can present with the following symptoms: fatigue/tiredness, diminished training capacity, reduced cognition, increased prevalence and duration of illnesses and infections, decresed appetite, higher resting heart rate, paleness, and potentially adverse pregnancy outcomes (NHMRC & MOH, 2014; SDA, 2020). Optimal physical performance depends upon effective utilisation and delivery of oxygen, which, without adequate iron, is less effective (Castell et al., 2015). All adults, including athletes, are encouraged to meet the *Recommended Daily Intake* (RDI) for iron, which may range from 8mg/day for men to 18mg/day for non-pregnant women (NHMRC & MOH, 2014; SDA, 2020).

Although the RDI of iron for elite athletes is not consistent in the literature, it is predicted to be significantly higher than in the general population for a number of reasons (SDA, 2020). For example, Thomas and colleagues support DellaValle's recommendation (2013) that all female athletes are likely to require an additional 70% of the recommended iron intake due to the additional and varied physiologic demands placed on the female athlete. For an in-depth analysis of the identification and management of low iron in athletes see the Joint Position Statement on Nutrition and Athletic Performance by the Academy of Nutrition and Dietetics, Dietitians of Canada, and the American College of Sports Medicine (Thomas, Erdman & Burke, 2016).

Athletes may be at particular risk for depleted iron stores for the following reasons (SDA, 2020):

1. Physical training stimulates the production of new red blood cells and other tissues, of which iron is a key component, contributing to greater demand for iron.
2. Minor ruptures in the digestive tract can cause a 'leaking' of red blood cells and therefore reduce their availability.
3. A phenomenon called 'footstrike haemolysis' occurs, whereby running on a hard surface can cause physical damage to the red blood cells in the feet, increasing iron losses.
4. Iron can be lost in sweat (particularly in those who sweat heavily).

Iron nutritional status may be further compromised in athletes who are engaging in restrictive eating (such as dieting/restriction, vegetarianism and veganism without appropriate non-haem intake strategies, or when red meat is rarely or never consumed), injured, menstruating (particularly if menstruation is heavy), undergoing a rapid period of growth (i.e. teenage years), completing high-altitude training, or experiencing increased via loss in urine or feces (Thomas, Erdman & Burke, 2016). See Thomas and colleagues' (2016) recommendations for managing iron status in athletes. Anaemic athletes should consider reducing physical activity that further depletes iron stores (e.g. weight bearing exercise, running on hard surfaces; Thomas et al., 2016).

Vitamin D

Vitamin D is another key micronutrient for athletes listed by Thomas and colleagues (2016), however, since there are limited food sources of this nutrient, its main source of availability is from the sun or supplementation (SDA, 2020). Vitamin D assists the absorption of calcium (SDA, 2020) and phosphorous (NHMRC & MOH, 2014) from food in the gut, and supports bone health, muscle strength, immune function and skin health (SDA, 2020). Thomas and colleagues (2016) refer to a number of studies supporting Vitamin D's association with reduced injury risk (including stress fractures), inflammation and acute respiratory illness, and improved rehabilitation outcomes, neuromuscular function, and type II muscle fibre size. It is not unusual for athletes, especially those participating in indoor competitions during childhood and adolescence such as classical dance and gymnastics, to train long hours, resulting in inadequate exposure to sunlight. Where exposure is inadequate leading to low Vitamin D, it is recommended that team seek advice from a member of the health team to develop opportunities for regular sun exposure to support and sustain optimal skeletal health. It is important to note that sun exposure should occur safely when the UV Index is below 3, which is generally in the early morning and late afternoon depending on a location's latitude (SDA; 2020). Thomas and colleagues (2016) suggest that aiming for blood levels from 80 nmol/L and up to 100 nmol/L to 125 nmol/L may support training adaptations but note that the process of determining optimal vitamin D requirements is complex.

Thomas, Erdman & Burke (2016) cite insufficient empirical data to refine Vitamin D recommendations for athletes. Low Vitamin D can cause structural bone issues via inadequate skeletal mineralisation or demineralisation (NHMRC & MOH [Vitamin D], 2014). These skeletal concerns can present as osteomalacia or osteoporosis in adults (often with accompanying fractures and falls) and rickets in children (SDA [Vitamin D], 2020). Lagowska and colleagues (2014) recommend that athletes should be assessed for an individualised Vitamin D supplementation protocol if they have a history of stress fractures, bone or joint injuries, over-training, muscle pain or weakness or low sun exposure. Similarly, Sports Dietitians Australia (SDA, 2020) recommend seeking medical advice from a medical doctor if deficiency is present or suspected.

Calcium

Calcium plays a number of roles in an athlete's musculoskeletal function (such as bone creation, maintenance and repair, muscle contraction and nerve impulses), vital cardiac function, natural

blood clotting process and optimal dental (teeth) health (NHMRC & MOH, 2014; SDA, 2020; Thomas, Erdman & Burke, 2016). Low calcium may arise due to inadequate intake, poor absorption (e.g. due to low levels of vitamin D, high caffeine or alcohol intake, certain medications, etc.), or excretion from the body associated with other nutrients such as sodium and protein (NHMRC & MOH, 2014; SDA, 2020). While daily calcium intakes are suggested to range from 1,000mg/day for adults to 1,300mg/day for teenagers (and between these levels during pregnancy; NHMRC & MOH, 2014), Thompson and colleagues (2016) recommend a calcium intake of at least 1300-1500mg/day and a Vitamin D intake of 1,500–2,000 IU/day if LEA (see pp.40) or amenorrhoea (see pp.56) are present. Athlete-specific intake recommendations are not currently specified, however are encouraged to be higher than average due to the demand that physical training places on calcium levels within the body (SDA, 2020).

As calcium is required to be readily available in the blood for its various roles, low blood calcium levels trigger hormones to signal the release of calcium from the bones (e.g., via PTH and calcitriol) to bring calcium blood levels back to normal, potentially further worsening bone health (SDA, 2020). Thomas and colleagues (2016) note low calcium intake is common in individuals who restrict energy intake (including people with disordered eating, vegans, and/or who do not consume dairy or other foods high in calcium). Athletes who are restricting dietary intake are particularly prone to low calcium intake, potentially leading to adverse bone health (Mountjoy et al., 2014). The NHMRC and MOH (2014) recommend calcium intake should not exceed 2,500mg/day and Thomas and colleagues (2016) suggest calcium supplementation should only be determined after a thorough assessment of the athlete's dietary intake. We note that replenishing calcium and Vitamin D must co-occur with any required corrections to sex hormones to maximally benefit bone health.

Antioxidants

Key antioxidants include vitamins C and E, selenium, beta-carotene and a variety of other carotenoids and plant-based phytochemicals. These nutrients are commonly found in a colourful variety of fruits, vegetables, vegetable oils and whole grain foods. Consumption of antioxidant-rich whole foods in post-exercise recovery nutrition is vital for athlete recovery and performance (SDA, 2020). Antioxidants play a pivotal role in protecting cell wall breakdown down during exercise, when oxygen travels through the cell wall 10-15 times the normal rate to help create energy for performance, contributing to a phenomenon known as oxidative stress (Peternelj & Coombes, 2011). Chronic exposure to oxidative stress can favourably enhance the body's native antioxidant system functions and reduce future cell wall breakdown (Watson et al., 2005), however, only if the athlete has consumed adequate antioxidant-rich whole foods to support this process (Thomas et al., 2016).

Athletes most at risk of insufficient antioxidant intake are those who restrict energy intake overall as well as those who limit their intake of fat, fruits, vegetables and whole grains (Rosenbloom & Coleman, 2012). Contrary to popular belief that antioxidant supplementation can effectively replace dietary intake, supplements that are taken in place of antioxidant-rich whole foods are discouraged (Thomas and colleagues, 2016). For example, little evidence exists to prove supplementation supports athletic performance (Peternelj & Coombes, 2011), and the

research methodology in existing studies is questionable (Thomas and colleagues, 2016). Further, supplementation may negatively impact training adaptations (Draeger and colleagues, 2014) or even cause a pro-oxidative response if supplemented above the Tolerable Upper Intake Level (Peternelj & Coombes, 2011).

4. Electrolyte abnormalities

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. Electrolytes facts

Electrolytes, or electrically-charged particles or ions, are key regulators of the body's exchange of fluids, nutrients and waste products (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).

Potential electrolyte contraindications to exercise

The following conditions may contraindicate exercise engagement. These conditions may be applied througout 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

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).

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 (<2.5 mmol/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). For athletes, the amount of sweat excreted in sport is negligible and does not impact the healthy athlete. However, in the case of an athlete with low potassium and higher levels of sweat excretion, it may be prudent to reassess potassium levels more frequently, as high sweat loss can contribute to the overall deficiency (Castell et al., 2015). Food sources of this nutrient include a variety of fruits, vegetables, nuts, wholegrains, and dairy products.

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 syndrome, malnutrition, chronic diarrhoea, and the chronic use of diuretics, and chronic alcohol use (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). Adequate phosphate supplementation can have ergogenic benefits for performance for healthy athletes. Indeed, increased phosphate has been related to improved Vo2 max, anaerobic threshold, and race times (Castell et al., 2015; Folland et al., 2008). Food sources of phosphorus include milk, yogurt, cheese, meats, nuts and whole grains. Food sources of phosphorus include milk, yogurt, cheese, meats, nuts and whole grains.

Hypomagnesemia

Hypomagnesemia, or low serum levels of magnesium (<0.7 mmol/L), can present as a result of diarrhoea, 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, as well as 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). Magnesium is an important cofactor of adenosine triphosphate (ATP) generation and

depletion in athletes seems to cause mitochondrial dysfunction. Exercise-induced hypomagnesaemia appears to coincide with alterations in ECG recording, which may represent heightened risk of arrhythmias or exhaustion (e.g. muscle fatigue and body collapse). Athletes may be especially prone to experiencing hypermagnesemia, due to depletion via sweat and urine, with weight class sports using hypohydration methods at greatest risk. Conversely, improving magnesium levels in athletes has been related to improved strength performance (Santos et al., 2012). Food sources of magnesium include whole grains and nuts.

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, hypercarbia can be caused by purging and is defined as a higher-than-normal serum bicarbonate level (30-40 mmol/L; Mehler & Andersen, 2017). Usually, metabolic alkalosis is asymptomatic, however, when bicarbonate increases above 35 mmol/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

Sodium is the main extracellular fluid excreted in sweat and can be lost in significant amounts during exercise. For example, a football player can lose 3-5 gms of salt during a 90 minute training session, with other athletes in other sports recording up to 10 gms lost (Shirreffs et al., 2006). Hyponatremia, or a low concentration of serum sodium, can present as mild to moderate, 120-130 mmol/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 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 extremely high water intake (Gaudiani, 2018; Mehler & Andersen, 2017). Hyponatremia is most common in endurance athletes, particularly those who are low weight and inexperienced with managing hydration during sport (Castell, Stear, & Burke, 2015). Replenishing lost sodium (through external supplementation) is a prerequisite for replacing water losses and euhydration; thus, exercise and sport participation is contraindicated if athletes are unable to do so. Symptoms of hyponatremia can range from less severe (e.g. nausea, confusion, headaches, and vomiting) to more severe (e.g. delirium, impaired consciousness, seizures, and, in some cases, cardiorespiratory arrest; Henry, 2015). Considering these risks, it is reasonable to suggest exercise in individuals with hyponatremia may be contraindicated or require modification.

Hypoalbuminaemia

Albumin is a protein produced in the liver, with adequacy typically assessed via serum albumin testing. Hypoalbuminaemia occurs when there is a low level of albumin (<3.6g/100ml). This may arise from decreased production of albumin in the liver or loss of albumin in the gastrointestinal tract or kidney due to prolonged starvation. Individuals with an ED, particular those with AN, may be at heightened risk for hypoalbuminaemia and related sudden death (Sardar et al., 2015). However, low albumin is neither a sensitive or specific indicator of malnutrition.

5. Sex Hormones and Relative Energy Deficiency in Sport

Relative Energy Deficiency in Sport

The term "Relative Energy Deficiency in Sport" (RED-S) has recently replaced the less inclusive "female athlete triad" to characterise the physiological consequences of low energy availability in athletes (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 expands the term female athlete triad by: (a) acknowledging that 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 to other body systems 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 health, and general growth and development (Mountjoy et al., 2018). Performance consequences of RED-S can include a decline in training reactivity, 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). In male athletes specifically, higher exercise dependence scores, were associated with higher ED symptoms, RED-S biomarkers (i.e. markers of endocrine, gastrointestinal, cardiovascular, haematological, metabolic, and bone health systems), more severe negative energy balance, and higher cortisol levels (Torstveit, Fahrenholtz, Lichtenstein, Stenqvist, Melin, 2019).

For those experiencing LEA, Gordon and colleagues' (2017) suggest that decreasing or modifying exercise, both with and/or without increased caloric and nutritional consumption, may favour improved energy availability (Gordon et al., 2017) and that with increased EA, the health and performance markers associated with RED-S may begin to diminish. Although weight management techniques are often used and promoted to enhance athletic performance, Gaudiani (2018) recommends that more appropriate targets for improving performance should include: adequate hydration, energy and nutrient intake, sleep, recovery, and emotional health. 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 well-being, behavioural patterns, and other outcomes of ED.

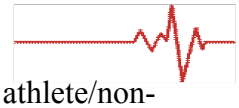
Immunity

As outlined by the RED-S model, immune function is impacted by LEA and can be further exacerbated in individuals engaging in DE (Mountjoy et al., 2018). Spence and colleagues note that impaired immune system functioning related to DE can place athletes at significantly greater risk for upper respiratory tract infections (URI). These may be particularly common during high training season. Further, Nieman (2003) notes that while moderate and healthful activity serves to improve immune system functioning (Nieman, 2003), DE contributes to decreased immune functioning, leading to increased URI risk. As such, screening athletes exhibiting recurring URI may help to identify those with excessive training load or engaging in DE.

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). Research examining differences between women with FHA and eumenorrheic controls, found higher interpersonal dependence and coping difficulties in the FHA group (Gordon et al., 2018). Irrespective of the stressor causing the FHA, an increased release of stress hormone, cortisol, and a dysregulation 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).

Functional Hypothalamic Amenorrhea:



- Absence of menstrual cycle 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**

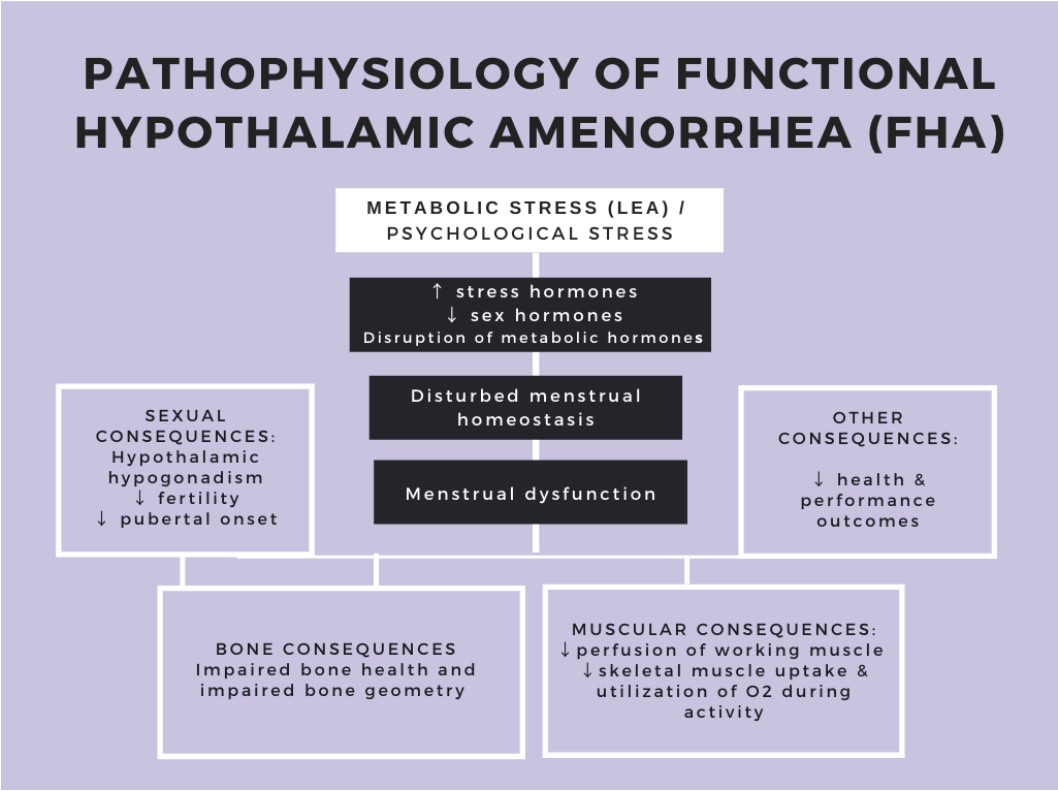
Box 9: Functional hypothalamic amenorrhea: definition and causes

Amongst its various consequences, such as the delay and stunting of growth during puberty (Gordon et al, 2017), infertility, decreased perfusion of working muscle, and decreased skeletal muscle oxygen uptake and utilization during physical 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 metabolism (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, in addition to many other hormones that are disrupted in both LEA and FHA, 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 oligomenorrheic 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 bone geometry, even with regular exercise engagement (Mallinson et al., 2016). The peak rate of bone formation 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). As athletes are generally expending more calories than non-athletes, they are at an increased risk of experiencing LEA (see pp.40) and associated menstrual dysfunction (Melin et al., 2019). That being said, 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 bone 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). Although many physiological consequences of malnutrition are reversible with adequate weight restoration and nourishment, the detrimental impact of LEA on bone health can be the most problematic to reverse and long lasting.



Box 10. Pathophysiology of functional hypothalamic amenorrhea

It is important to note that FHA can still occur even if the athlete is not at an objectively low weight or 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). Gordon and colleagues (2018) suggest that normal menstrual function may return within six to twelve months of weight stabilisation in combination with psychological support; however, they also note that the longer the amenorrhea has been present, the longer time it may take to reverse and for some, regular menses may never resume. The most effective method of preventing adverse hormonal effects of physical activity is to increase EA to at least greater than 30kcal/kg of lean body mass per day, minimize psychological stress (Gordon et al., 2017) 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, excessive high impact exercise, such as repetitive jumping and running, without normalised sex hormones or resumption of menses may negatively impact long-term bone health; these activities would therefore be contraindicated (Schied et al., 2011; Zanker et al., 1998). Finally, failing to modify exercise amount, type and/or intensity 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). The Female Athlete Triad Coalition has proposed a number of factors, which can increase risk of adverse outcomes for athletes participating in sport. These include: less than six menses in the last 12 months, a BMD z-score equal or less than 2 or less then one at high risk of trabecular bone sites, BMI < 17.5kg/m^2, and current ED diagnosis (Joy et al., 2016).

Overtraining Syndrome

There is a range of definitions for overtraining syndrome (OTS); however, for the purpose of this guideline, we will define OTS as the accumulation of stress resulting from physical training in addition to life stressors. OTS can result in psychological (depression, insomnia) and physiological symptoms (fatigue, weight loss, menstrual disturbance) as well as poorer long-term athletic performance (Carafagno & Hendrix, 2014; Thompson & Sherman, 2011). Gleeson and colleagues (2002) note that athletes with OTS engage in excessive training, while attempting to improve their physical condition and performance, which instead contributes to chronic fatigue and worsening competitive performance. Estimated prevalence of OTS ranges are between 5-65% of athletes, with estimates varied due to the diversity of sport types investigated and demographics of the included sample (Carafagno & Hendrix, 2014). It is unclear whether there are gender differences in the prevalence of OTS, however, athletes competing in endurance sports where the body is under significant stress for a long period of time, appear to be at greater risk for developing OTS (Carafagno & Hendrix, 2014). OTS can result in over 200 individual markers, with common symptoms including muscle soreness and weakness, cytokine action, hormonal and haematological changes, mood swings, depression, loss of appetite and diarrhoea (Eichner, 1994; Fry et al., 1991). Gleesson and colleagues (2002) highlight the following markers in detecting OTS:

- Performance
- Mood state questionnaires
- Diary of responses to training (fatigue, muscle soreness) and symptoms of illness
- Sleeping heart rate
- Blood lactate and plasma cortisol response to high intensity or incremental exercise
- Plasma creatine kinase activity
- Cortisol:Testosterone ratio
- Nocturnal urinary noradrenaline and adrenaline secretion
- Routine haematology (blood haemoglobin, serum ferritin, leukocyte counts)
- T-lymphocyte CD4+/CD45RO+ expression
- Experience of coach and athlete

If OTS is detected, team training and competition is contraindicated as continued high volume of training without incorporating an appropriate rest time can result in worsening immune and endocrine system function, depression /mood swings, and overall decline in performance (Gleeson, 2002). It can take weeks to month to recover from OTS (Gleeson, 2002); however, restorative activities in SEES-A Level A may be applicable during this phase. OTS should not be confused with over reaching, which is a normal progression of training and performance defined by short term performance losses during high levels of training (Hawley, Schoene,Harmon & Rubin, 2003; Gleeson, 2002). When an athlete is over reaching, they may experience muscle soreness, decreased coordination, reduced libido, or more frequent URI (Hawley,et al., 2003).

Overreaching can resolve quickly (i.e. less than one week) if followed by periods of lighter training (i.e. tapering; Hawley et al., 2003). If over reaching continues without appropriate rest periods for the athletes, it can lead to OTS (as seen in Figure 1).

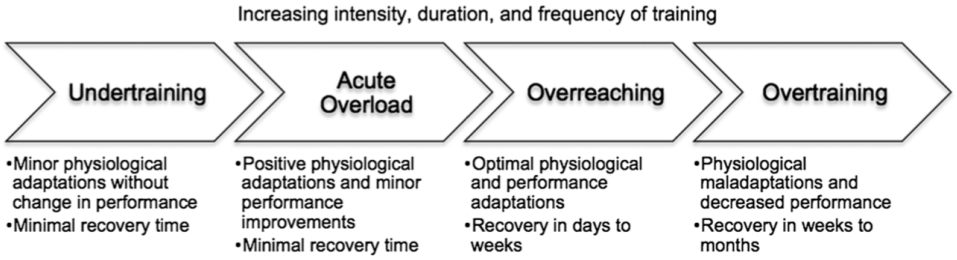
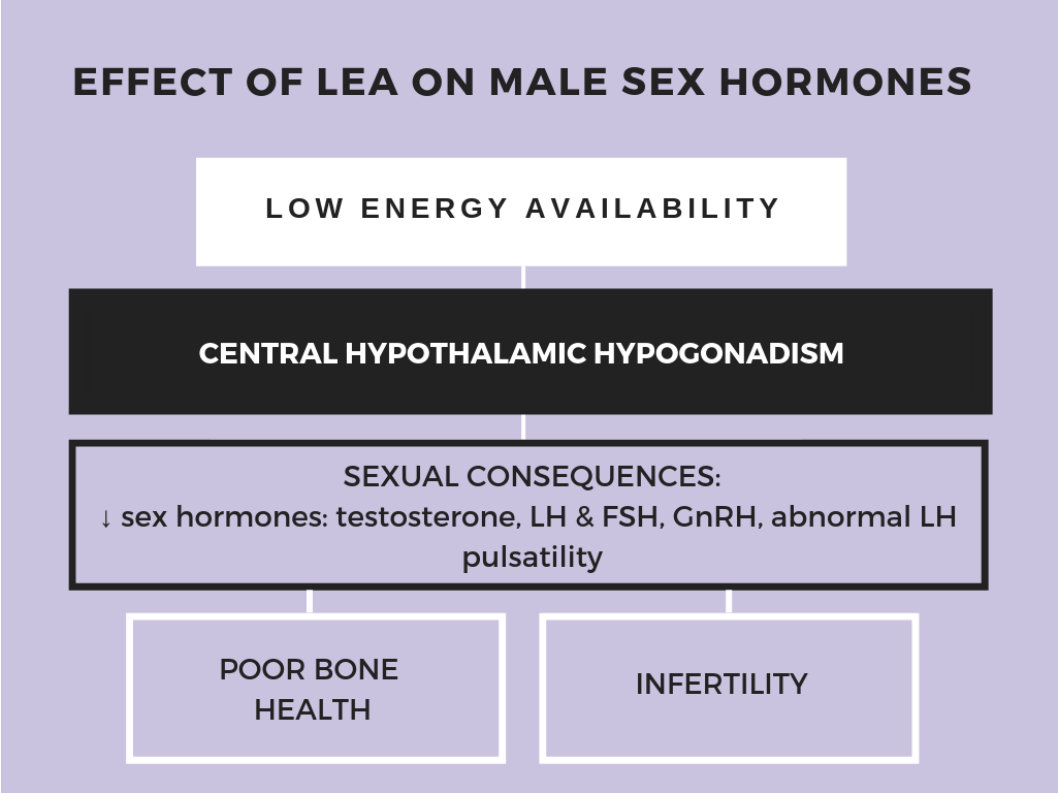


Figure 1. Progression of overtraining by Carfagno and Hendrix (2014)

Sex hormones and gonadotropins

Decreased GnRH drive, disrupted LH pulsatility, low FSH, estriadol, and progesterone, amongst other hormonal disruptions, are associated with amenorrhea and various patterns occurring in menstrual dysfunction. The hormonal suppression seen in LEA leads to poor bone health, the stunting of growth during puberty, infertility, and suboptimal muscular performance particularly in athletes (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 (Mountjoy et al., 2014). It has been suggested that the most effective method of preventing adverse hormonal effects of physical activity is to minimise stress and increase energy availability to at least above 30 kcal/kg of lean body mass per day, particularly in athletes (Melin et al., 2015).

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 LHpulsatility, can cause impaired reproductive function, fertility, and bone health (Mehler & Brown, 2015; Mountjoy et al., 2014) (Box 11). Even prior to the elite level, amateur exercisers with dysfunctional exercise have been found to have lower levels of testosterone with endurance runners being at heightened risk (Geesmann et al., 2017; Hagmar, Berglund, Brismar, & Hirschberg, 2013; Torstveit et al., 2019; Wheeler et al., 1991). Further, the reluctance of male athletes to report these particular issue, in conjunction with normalisation of disordered eating exercise behaviours in athletic circles, may contribute to delays in treatment, poorer outcomes, and protracted return to healthy functioning (Petrie & Rogers, 2001).



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

6. Body Composition

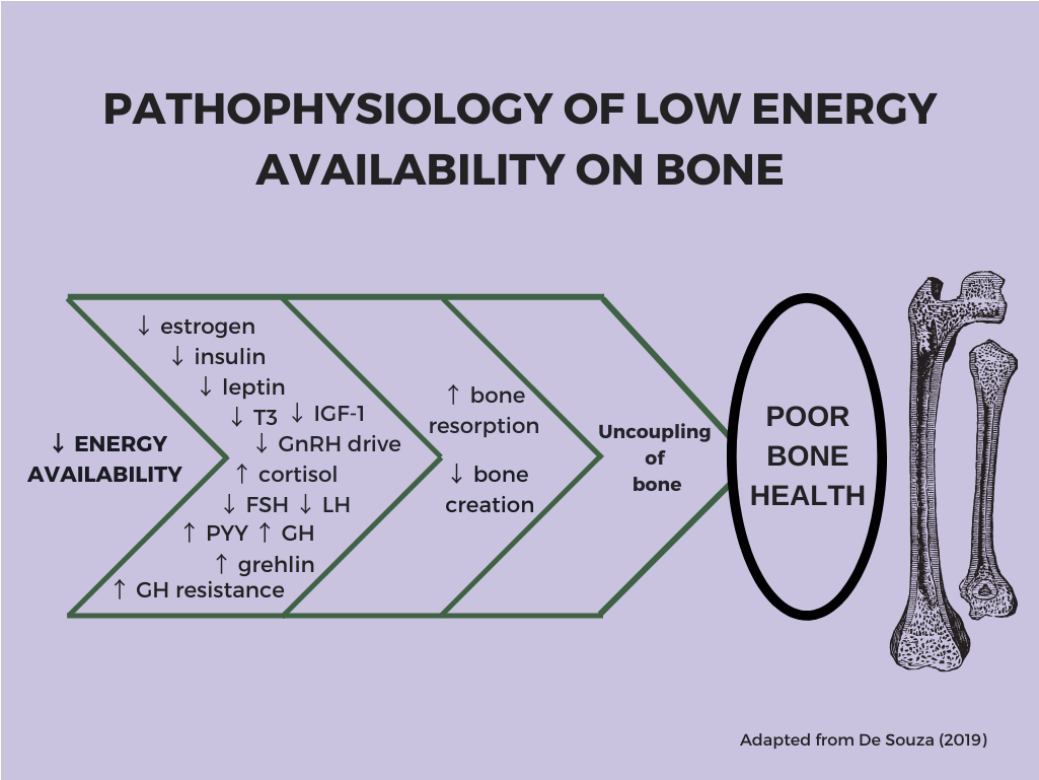
The sporting environment often promotes adapting body composition for the purpose of achieving peak optimal performance. However, self-directed attempts to change body composition can both detrimentally affect fat-free mass, metabolic function, strength, and performance (Sundgot-Borgen & Garthe, 2011; Trexler et al., 2017) as well as put individuals at risk for developing an ED. Further body composition changes that occur in the presence of an ED, can decrease an individual's endurance, cardiovascular fitness, and strength (Santos et al., 2014). Individuals with AN in particular typically lose muscle mass, while gaining fat mass, and have poorer physical performance while underweight, contributing to recommendations for engaging in individualized, health-enhancing physical activity programs to improve fitness (Alberti et al., 2013).

Compared to controls, athletes have higher bone and muscle mass, and lower fat mass (Popovoc et al., 2013). An athlete's body composition will vary largely based upon gender, sport, position, and competition level. For example, athletes competing in lean-focused sports (e.g. cycling, gymnastics, and figure skating) are more likely to have a lower body mass index, lower percent body fat, and greater variation in weight prior to Olympic competition than those not competing in lean-focused sports, with male athletes reporting greater discrepancies than female athletes. Santos and colleagues (2014) developed a standard set of appropriate reference values for body composition and anthropometric measurements in elite athletes in over 10 sports in both male and female athletes which may serve as treatment markers for ED treatment.

Low Bone Mineral Density (BMD)

Low BMD is defined by a z-score less than -1.0 in a weight-bearing athlete (ACSM, 1997; Leib, Lewiecki, Binkley, & Hamdy, 2004). A z-score compares an individuals BMD to someone of their same age, sex, and sometimes race, depending on the database used. A low BMD can be caused by a larger amount of bone breakdown than bone formation 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 observation is the fact that the duration of AN in males in this study was shorter than in females, suggesting that 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 initially tested (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 of living, 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 the “fullness hormone” leptin, thyroid hormone triiodothyronine (T3), glucose-regulator insulin, and insulin-like growth factor 1 (IGF-1), are decreased in a LEA state, while anabolic growth hormone (GH) and GH resistance are increased (De Souza, 2019). Other metabolic hormones, such as hunger hormone ghrelin, fullness hormone peptide 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). The hormonal changes resulting from LEA causes the uncoupling of bone turnover, whereby resultant low levels of oestrogen allow 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

LEA prior to puberty is particularly damaging as vital bone growth can be stunted and important sexual development, required to maximise 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 poor bone geometry, even with regular exercise engagement, which would normally encourage bone growth (Mallinson et al., 2016; Singhal et al., 2018). It has been suggested that a state of hypoestrogenism negates the usual positive effects of weight-bearing exercise at the spine in oligomenorrheic athletes (Kandemir et al, 2018). The peak accrual of bone, contributing to adequate BMD and bone content, in healthy females typically occurs 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 accrual 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, ad cumulative menstrual dysfunction is a significant predictor of poor bone mass (Drinkwater, Bruemner, & Chesnut, 1990; Mallinson et al., 2016).

Amenorrheic female athletes are observed to possess significantly lower hip and lumbar spine bone mineral density when compared with eumenorrheic athletes (Ackerman et al., 2012). Particularly, young female athletes with oligomenorrhea exhibit 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, amenorrhoeic athletes have lower BMD and bone geometry, particularly at the lumbar spine and worse bone architecture, particularly at the non-weight bearing radius (Ackerman et al., 2012). 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, there is an increased risk of bone stress injury, which poses the risk of further delay in one's ability to engage in exercise (Barrack et al., 2014; Mountjoy et al., 2014). One study revealed that 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 with eumenorrheic controls (1.2%); although individuals with AN were found to report higher overall fracture rates (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 an increase in body weight (BW) by 10% along with the resumption of menses (having had at least one menses in the past three months). In this study, 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). This 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% over a one year period and that with a resumption of menses alone spine BMD can recover by 3% over a one year period (Miller et a, 2006). At present, recovery of bone geometry is not known (De Souza, 2019). Barrack 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 an individual is amenorrhoeic and unable to engage in a nutritional rehabilitation, exercise is 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 is therefore be required in these circumstances.

Body fat and weight factors

Body Mass Index (BMI) is a ratio of weight to height and is determined by dividing one's weight in kilograms by meters squared (McArdle 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 that a BMI of less than 75% of median for age, sex and height may indicate the need for hospitalisation (Geller et al., 2012)).

Recent literature recommend that a progression of weight recovery may be more appropriate instead of utilizing a solitary marker of weight (which impacts BMI) to decide appropriate exercise engagement (Cook et al., 2016; Noetel, Dawson, Hay, & Touyz, 2017; Quesnel et al., 2017). Further, 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 physical activity or rigorous training, achieving 90% of ideal body weight may be important (McCallum et al., 2006).

BMI and health status:

- BMI does not depict health status
- Some individual's weight is not affected at any stage of their ED
- It is imperative to comprehensively evaluate medical and psychological parameters of an athlete's health for appropriate information.

Box 13: BMI and health status

A variety of techniques are employed to assess body composition (dual energy x-ray absorptiometry (DXA), hydrodensitometry, air displacement plethysmography, skinfold measurements, and single and multi-frequency bioelectrical impedance analysis), with DXA suggested to provide the most reliable data (Thomas et al., 2016). For increased measuring accuracy, Thomas and colleagues (2016) recommend the following best practices: using a standardised protocol, calibrated equipment, the same piece of equipment used consistently over time, trained technicians with known test-retest reliability, and cross-validated and reliable population–specific prediction equations. Establish an interval of at least 2 to 3 months between serial measurements, so that short-term fluctuations in body weight do not confound interpretations (Bonci et al., 2008). The assessment protocol should be developed in consideration of a "schedule that is appropriate to the performance of the event", practicality and athlete sensitivity (Thomas, Erdman & Burke, 2016). Regardless of body composition assessment method used, all athletes should receive comprehensive information about its limitations, and

should be encouraged to follow the pre-assessment rules as closely as possible to contribute to accuracy and reliability (Thomas, Erdman & Burke, 2016). Communication of body composition and weight information should include education on the limitations of measurement techniques, discussion of normative data and ranges rather than absolute goals, and communication of data in a sensitive manner that avoids endorsing an undue relationship between body composition and performance which can promote an obsession with weight/shape/size (Santos et al., 2014; Sundgot-Borgen & Garthe, 2011).

Many athletes are motivated to alter their body weight and/or composition for their sport, whether to improve their performance or to achieve weight requirements for participation (Samadi et al., 2019; Sundgot-Borgen & Torstveit, 2010). However, without evidence-informed oversight, this may be to the detriment of performance and strength or may result in disordered eating and exercise behaviours or LEA (Sundgot-Borgen & Torstveit, 2010). Thomas, Erdman & Burke (2016) highlight that an athlete's performance cannot accurately be predicted based on body weight or body composition alone. These authors support Sundgot-Borgen and colleagues' (2013) stance that one, static, optimal body composition cannot and must not be recommended for any event or any athlete. Indeed, an athlete's levels of body fat will vary both across seasons and the athlete's entire career (Thomas et al., 2016). Despite this, the pursuit of a particular body composition for various sports still exists (Thomas et al., 2016). While a high level of *absolute* power, size or strength is prioritized for some athletes, other sports such as light-weight rowing, combat sports and weightlifting encourage the highest possible power output, while keeping as light as possible on the water or within a certain weight category to maximise an athlete's "power to weight ratio," or termed *relative* power output (Stellingwerff, Maughan & Burke, 2011). Endurance athletes typically aim for the lowest energy cost of exercise whilst keeping a favorable weight to surface area ratio for optimal heat dissipation; while athletes, such as divers, gymnasts and dancers, may gain a biomechanical advantage if their body is smaller so movements can be achieved in a small space (Prelack et al., 2012; Thomas et al., 2016). Team athletes may need to be lean to be as fast and agile as possible, while other athletes, like figure skaters, are judged on their aesthetics (Thomas et al., 2016). For further discussion of the implications of different sports during ED treatment see the Sporting Considerations section (pp.35)

Sundgot-Borgen and colleagues' (2013) note that pressures to achieve a specific body size can contribute to unrealistic weight or body composition goals in unrealistic time frames. Further, these goals may lead to extreme and/or prolonged dieting, chronic LEA, malnutrition, and attempts to match or beat previously low weight or body fat levels (Sundgot-Borgen et al., 2013; Sundgot-Borgen & Garthe, 2011). Thomas and colleagues (2016) suggest body composition goals should only be set at critical times (e.g. Garthe and colleagues, 2011, suggest this could occur in the first phase of training or far from competition), use techniques which preserve muscle mass and overall health (e.g. ensure the energy deficit is only minimal and the athlete is consuming extra protein and micronutrient-dense foods), and within ranges rather than at or below a specific target. O'Connor & Slater (2011) add that attempts to change weight must be responsible, indicated, and short-term. Best practices would ensure that the athlete is closely monitored by a licensed nutrition professional who has crafted the nutritional plan after assessing the appropriateness for the individual athlete. Thomas, Erdman & Burke (2016) suggest

simultaneously avoiding sudden and/or prolonged weight gain during injury, off-season or in sports that encourage a larger body size. These are vulnerable times for athletes when disordered eating behaviours can escalate, making the relevance of nutritional consultation quite notable. If composition must be altered, it should be periodised (Thomas, Erdman & Burke, 2016), gradual, individualised monitored, and sustainable (Sundgot-Borgen & Garthe, 2011).

Despite the flaws of the BMI categories and the many factors that influence weight, engaging in exercise with a BMI <13.5kg/m^2 may be contraindicated due to the range of health and performance consequences from energy deficiency (Docx et al., 2010; Gaudiani, 2018; NEDA, 2018; Mountjoy et al., 2018). Further, it has been recommended to avoid high impact activity below a BMI of 16kg/m2 (Howgate et al., 2013), potentially also due to low energy availability (Gaudiani, 2018; NEDA, 2018; Mountjoy et al, 2018).

5. Other related adverse outcomes of ED

Hypoglycaemia

Hypoglycaemia is defined as a blood sugar level below 4 mmol/L (McArdle et al., 2010). Dietary restriction, significant or rapid weight loss, 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, altering normal glucose metabolism and leading to chronic 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 the telltale signs of 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 hypoglycaemic events such as death and irreversible brain damage (Gaudiani, 2018; Rich, Caine, Findling, & Shaker, 1990).

Engaging in exercise while in a hypoglycaemic state can irreparably damage the brain (McArdle et al., 2010). Exercise can worsen hypoglycaemia and its physiologic effects due to the unrelenting metabolic need for glucose during exercise (McArdle et al., 2010). Thus, exercise is highly contraindicated for individuals in a hypoglycaemic state. To counter this situation, modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favour improved energy and nutritient availability and the improvement of a variety LEA-related consequences (Mountjoy et al, 2018).

Gastroesophageal reflux disorder

Gastroesophageal reflux disorder (GERD) is common among athletes who are bingeing and purging while concurrently engaging in aerobic exercise (Collings, Pierce-Pratt, Rodriguez-

Stanley, Bemben, & Miner, 2003). Unfortunately, the severity of the esophageal acid reflux can increase with the intensity of exercise (Collings et al., 2003). Further, chronic acid reflux exposure might facilitate the development of atrial fibrillation in those who dysfunctionally exercise (Swanson, 2008).

Superior mesenteric artery syndrome

Superior mesenteric artery syndrome (SMA) is caused by the compression of the duodenum due to a diminished fat pad (caused by 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, Schimidt-Sommerfeld, Haymon, & Udall, 2000).

Body Temperature Abnormalities: Hypothermia and Hyperthermia

Hypothermia, or a low body temperature <35°C or <95°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; consequently, it warrants exercise modification (Luck & Wakeling, 1982). Conversely, hyperthermia, also known as a high body temperature > 36°C or >96.8°F, can occur as a result of failed thermoregulation when the body produces or absorbs more heat than it can dissipate 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 is 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). Athletes with EDs, in particular, report higher rates of depression than non athletes with EDs (Fewell et al.,2018). 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 when LEA or an ED develops. These athletes 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-A guideline for these populations must be done under the supervision of medical advice specific to the athlete's individual requirements. These populations may include (but are not limited to): Paralympic athletes, Special Olympic athletes, youth athletes, athletes 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 who engage in vomiting, laxative, or diuretic use alongside exercise due to the compromising nature of these compensatory 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. High USG can suggest the presence of adrenal, liver, or heart conditions, or dehydration issues in individuals who are excessively sweating, vomiting or who have diarrhea (Shumann & Schweitzer, 1996).

Exercise does not affect urine specific gravity directly, however, it does influence a number of components that will ultimately be reflected in the outcome of a USG test, such as hydration levels, and issues related to kidney and adrenal function (Shumann & Schweitzer, 1996). For example, blood urea nitrogen may be abnormally high 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 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 syndrome, 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 and/or life-threatening complications are likely to occur, regardless of BMI (Gaudiani, Sabel, Mascolo, & Mehler, 2012).

High levels of transaminases in the setting of ED may result from the body's breakdown of its own tissue or a reduction of blood flow through the organs, as a result of myocardial dysfunction (Brown & Mehler, 2015). Although transaminases do not primarily contribute energy as fuel for exercising muscle, there is a significant increase in the rate of amino acid catabolism that occurs during exercise (Henriksson, 1991). For example, a process occurs in which amino acid formation from the non nitrogen-carrying organic compound pyruvate formed in metabolism is permitted, which is important to facilitate muscular work (McArdle 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.

6. Key psychological and behavioural factors

Starvation

Pathological weight control practices are all too common in athlete populations, even in those without a diagnosable ED. For example, Anderson and colleagues (2012) report that about 30% of swimmers and gymnasts reported some level of eating disturbance, with 14.2-28.2% stating they had dieted or fasted two or more times in the past year, and just over half of this sample exercised for 1 hour a day with the goal of burning calories (Anderson & Petrie, 2012). In a state of LEA, which may be induced by over-exercising, dieting, or '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 athletic performance outcomes, as outlined in Mountjoy and colleagues (2018). If EA becomes extremely low, to the point of starvation or famine, the body will ultimately be forced to initiate catabolism of its own metabolically-active tissue (NEDA, 2018). This breakdown of lean body mass 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, life-threatening cardiac complications (NEDA, 2018). Achieving adequate energy availability was proposed in an effort to better manage and avoid these types of complications (Mountjoy et al., 2014). See section on LEA (pp.40) for further details.

Understandably, inadequate caloric consumption relative to individual energy requirements has been put forth as a contraindication to exercise (Cook et al., 2016; Littlefield and Zuercher, 2003; Quesnel et al., 2017). El Ghoch and colleagues (2013) have agreed that exercising in a starved state will inevitably pose health risks (including death) and sabotage athletic performance 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 increases circulatory lactate, causing widespread

muscle pain (Ghoch et al., 2013). Thirdly, a state of dehydration can trigger muscle cramps (Ghoch et al., 2013). Finally, if undernourished while engaging in exercise, the consequent 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) conclude that inadequate energy availability prior to or following exercise likely contraindicate exercise engagement. It has also been suggested that modifying exercise engagement, both with and/or without increased caloric and nutritional consumption, may favour improved energy and nutritient availability and the improvement of LEA-related consequences (Mountjoy et al., 2018).

Hypohydration

Manipulating water intake and output through the use of fluid restriction, diuretics, and intentional excessive sweating can put athletes at heightened risk for medical complications (Samadi et al., 2019). However, observational studies have reported hypohydration at weigh-in is unfortunately the norm, not the exception, for many athletes competing in weight-dependent sports (Pallares et al., 2016; Samadi et al., 2019). 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 1-2% 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 (and associated electrolyte disturbances) induced haemodynamic instability can contraindicate engagement in sport (Currie & Crosland, 2009; Mountjoy et al, 2014).

Dysfunctional exercise

Dysfunctional exercise (DE) occurs when an individual engages in exercise despite being injured or ill, experiences withdrawal symptoms (e.g. agitation, low mood) when unable to exercise, or gives up social, occupational, and family obligations to exercise (Hausenblas & Symons Downs, 2002). DE 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 DE 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', psychological reliance on exercise may still remain (Quesnel et al., 2017). Considering this, individual with DE may benefit from switching to lower intensity and less frequent physical activity to decrease their depenence on exercise (Quesnel et al., 2017). Furthermore, individuals may be encouraged to refrain from engaging in previously DE modalities, particularly if also exhibiting physical or emotional signs of OTS (El Ghoch et al., 2013). For example, switching from running to yoga may offer an alternative movement therapy that restores the mind-body connection. Healthy

exercise engagement, in combination with appropriate movement psychoeducation, has been found to decrease DE (Schlegel et al., 2015).

Characterizing DE in athletes can become muddied as there are several parallels that can be drawn between DE and characteristics of a disciplined athlete (e.g. rigidity, routine, structured, mental toughness; Thompson and Sherman, 1999). As such, standard measures of DE may overpathologise athletes, requiring a more nuanced examination of exercise behaviours in athlete populations (McNamara & McCabe, 2013). McNamara and McCabe (2013) developed the *Exercise Dependence and Elite Athletes Scale* of dysfunctional exercise for elite athletes. The questionnaire was based on characteristics identified by coaches from over 10 different sports (including endurance, ball, and aesthetic sports) as associated with DE. This tool has good validity with the athletic ED population and may be more tailored than the original compulsive exercise test for this population (McNamara & McCabe, 2012; Plateau et al., 2013). We do note, however, that the shorter compulsive exercise test for athletes (CET-A) has shown good sensitivity to detect athletes with a clinical ED, potentially representing a useful screening tool (Plateau et al., 2013). The EDAS is a 24 item self-reported measure of DE specifically in elite athletes covering 1) *Unhealthy Eating Behaviours* targeting the extent to which exercise is used to control shape and weight; 2) *Conflict and Dissatisfactio*n, reluctance to approach team staff about injuries or illness; 3) *More Training*, reflecting the "more is better" thinking and performance preoccupations; 4) *Withdrawal,* emotional and cognitive consequence associated with not engaging in exercise; 5) *Emotional Difficulties,* continued experiences of anxiety, isolation, depression related with DE, and 6) *Continuance Behaviours*, tendencies to be inflexible and rigid with training. These six subscales are measured across 24 items scored on a 5 point Likert scale from 1 (Never) to 5 (Always). The EDAS has been validated to both detect DE in athletes, and track changes in this relationship (McNamara & McCabe, 2011; 2013). For our purposes, a score above the midpoint (3 or some of the time) in 3 or more subscales will characterise the "at risk" group, indicating that training should remain in SEES-A Stage 1 (McNamara & McCabe, 2011). An improvement of scores indicates a progression through the stages of SEES-A.

Purging, Purposeful Dehydration, and Hypovolemia

Studies report a high prevalence of athletes engaging in compensatory behaviours, with those in aesthetic and weight dependent sports at highest risk (Giel et al., 2016; Samadi et al., 2019). Common methods include passive and active dehydration strategies, exercising for weight loss, self-induced vomiting, and the misuse of laxatives or diuretics (Barley, Chapman, & Abbiss, 2018; Giel et al., 2016; Samadi et al., 2019). Even if an athlete's dietary intake appears sufficient, those engaging in purging or other compensatory behaviours are actively reducing the amount of nutrition available to the body (UK Sport, 2013). Using laxatives and vomiting can cause water losses of up to 5000 mL, which far exceeds values of normal water loss and can cause potentially dangerous fluid and electrolyte imbalances (McArdle et al., 2015). Furthermore, these behaviours are also associated with complications including throat and mouth issues (e.g. dental erosion), heart problems (including electrolyte abnormalities and cardiac arrest), gastrointestinal issues (e.g. rectal prolapse), and kidneys problems (e.g. renal failure; Forney et al., 2016). Further, these behaviours contribute to dehydration, metabolic alkalosis, severe kidney disease, and seizures, as

well as softening of the bones associated 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 with subclinical frequency of purging (Raj et al., 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, contributing to dehydration (Mehler & Andersen, 2017). Diuretics and laxative use can also create fluid and electrolyte disturbances, however, via slightly different mechanisms (Mehler & Andersen, 2017). Firstly, electrolytes are regularly and negatively altered in purging-related ED, with the most common electrolytes affected being potassium, sodium and bicarbonate (Mehler & Andersen, 2017). Additionaly, 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 of blood volume depletion due to insufficient food and fluid intake and the presence of vomiting and laxative use (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).

Purging, irrespective of the method, can detrimentally impact exercise performance (Mehler & Andersen, 2017). Mehler and Andersen (2017) state that this can occurs via three core mechanisms; 1) the generation of a negative caloric balance; 2) the facilitation of dehydration; and 3) particularly in dehydrated individuals, induction of 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 that electrolytes are naturally lost in sweat during exercise (Lindsay, 2017; McArdle et al., 2015; Mehler & Andersen, 2017), hypovolemia (decreased blood plasma volume) or the presence of electrolyte imbalances alongside hypohydrated state contraindicates exercise engagement (Fletcher et al., 2013; Thomas et al., 2016).

Muscle dysmorphia

Muscle dysmorphia was initially described by Pope and colleagues (1993) in male bodybuilders who abuse anabolic steroids, It was more recently added to the DSM-5 as a specifier of body dysmirphic disorder and is characterised by a preoccupation with being insufficiently large and muscular, resulting in individuals engaging in dysfunctional body change behaviours and/or experiencing heightened functional impairment or distress (American Psychiatric Association, 2013; Pope et al., 1997). Risk factors for muscle dysmorphia intersect with EDs and include body dissatisfaction, body distortion, ideal body internalization, low self-esteem, perfectionism and negative affect (Grieve, Truba, & Bowersox, 2009). Athletes engaging in weightlifting or bodybuilders may be at specific risk for developing muscle dysmorphia, however, it can also be present in the general population (Dos Santos Filho, Tirico, Stefano, Touyz, & Claudino, 2016). At a subclinical level, athletes may also attempt to increase FFM through muscularity-oriented disordered eating, which is associated with with traditional eating disorder severity and impairment as well as higher rates of muscle dissatisfaction (Cooper, Griffiths, & Burns, 2020).

***Signs and symptoms* that may contraindicate exercise engagement.**

The following are a list of symptoms that may contraindicate exercise engagement, with those in bold suggested as more likely to 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
- Light-headedness
- Confusion
- Nausea
- Cold/clammy skin
- Wheezing
- Leg cramps
- Claudication
- Fatigue
- Peripheral oedema

Box 14. Symptoms that may contraindicate exercise engagement

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

Box 15: Intensity measures

Glossary of Terms

Aesthetic Sport: Sport judged on both appearance based criteria and performance criteria such as gymnastics, figure skating and diving (Thompson & Sherman, 2011)

Athletic Trainer: An individual whose role is the prevention, examination, diagnosis, treatment, and rehabilitation of emergent, acute, or chronic injuries and medical conditions (National Athletic Trainer's Association, 2019)

Athlete: A person who is training purposefully for the goal of winning competitions or performance in sporting or dance activities. An individual who has a structured training schedule, is personnel supported (i.e. coach, trainers, etc.) and training to improve performance.

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).

Antigravitational sports: Sport that are focused on jumping such as high jump, long jump etc (Thompson & Sherman, 2011).

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/metre^2 (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).

Coaches: An individual who trains a person or persons in a particular sport.

Coachability: Sport participants' willingness to do what their coach asks of them (Thompson & Sherman, 2011).

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).

Interdisciplinary 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).

Lean Sport: Sport in which muscular leanness is emphasised for competitive advantage, which encompasses three categories of sport including endurance sport, weight class sports, and aesthetic sports (Thompson & Sherman, 2011)

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

Non Lean Sports: Sport that does not emphasize the need for lean body composition, such as ball sports and all other sports not encompassed in the lean sporting category.

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

Overtraining syndrome: The accumulation of stress resulting from physical training in addition to life stressors. Overtraining syndrome can result in psychological (depression, insomnia) and physiological symptoms (fatigue, weight loss, menstrual disturbance) as well as poorer long-term athletic performance (Caefago & Hendrix, 2014; Thompson & Sherman, 2011).

Pathological Weight Control Methods: Unhealthy or detrimental behaviours used to control weight and shape. Including those used by individuals with EDs such as vomiting, abuse of laxatives, emetics, fasting, dysfunctional exercise and enemas (Thompson & Sherman, 2011).

Physical Activity: Any bodily movement produced by skeletal muscles that results in energy expenditure (McCardle 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).

Sport Family: Athletes sporting entourage, which include coaches, sport personnel, teammates and others (Thompson & Sherman, 2011).

Sport/Coaching Team: Group of coaches and sport personnel responsible for the overarching care of the athlete

Training: Athletic training involves preparing for sporting competition including: technical training and preparation, tactical training, psychological training, and conditioning training.

Treatment Team: In the context of SEES-A, a treatment team is a group of health care professionals that are responsible for treating EDs in athletes in conjunction with the sport team

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