REVIEW ARTICLE

The Benefits of Strength Training on Musculoskeletal System Health: Practical Applications for Interdisciplinary Care

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Abstract

Global health organizations have provided recommendations regarding exercise for the general population. Strength training has been included in several position statements due to its multi-systemic benefits. In this narrative review, we examine the available literature, first explaining how specific mechanical loading is converted into positive cellular responses. Secondly, benefits related to specific musculoskeletal tissues are discussed, with practical applications and training programmes clearly outlined for both common musculoskeletal disorders and primary prevention strategies.

1 Introduction

The importance of strength with regard to athletic performance has been highlighted within recent reviews [1, 2]. The benefits of increasing muscular strength include a positive influence on rate of force development (RFD) and power [1, 3, 4], improved jumping [1], sprinting [5] and change of direction (COD) performance [6], greater magnitudes of potentiation [1], and enhanced running economy [7]. Strong evidence supports the notion that maximal strength serves as one of the key foundations for the expression of high power outputs and that improving and maintaining high levels of strength are of utmost importance to best capitalise on these associations [8–13].

What appears to be discussed less so is the impact of strength training on musculoskeletal health. This is surprising given that within previous literature it has been highlighted that strength training can reduce acute sports injuries by one-third, and overuse injuries by almost half [14]. Furthermore, strength training programmes appear superior to stretching,

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Key Points

• Strength training confers unique benefits to the musculoskeletal system in common disorders and in healthy people.

• The application of mechanical loading must be specific to obtain the desired positive adaptation.

• Healthcare professionals should promote strength training among the general population due to its multi-systemic and specific musculoskeletal benefits.

proprioception training, and multiple exposure programmes for sports injury risk reduction [14]. Malone et al. [15] found that players with a higher relative lower body strength (3 repetition maximum [RM] trap-bar deadlift normalised to bodyweight) had a reduced risk of injury compared to weaker players. In addition, stronger athletes had a better tolerance to both higher absolute workloads and spikes in load than weaker athletes. Despite its apparent effectiveness for the reduction of injury risk, there is still far less coverage regarding the positive effect of strength training on injury risk or occurrence within the scientific literature, which may be due to its poor integration within musculoskeletal rehabilitation [16] and primary prevention strategies for sports injuries [17, 18]. This is further limited by a poor understanding and knowledge of physical activity guidelines among healthcare professionals [19–21], which provides challenges for its integration into sports medicine practice. Indeed, it is not uncommon for healthcare professionals to recommend "strengthening programmes" using 10 or more repetitions per set without a clear indication of the intensity adopted [22, 23]. Although most resistance training modes have demonstrated improvements in strength in inactive/untrained individuals during the first weeks [24], it must be pointed out that "strengthening programmes" and "strength training" are not the same; hence, they cannot be used interchangeably.

Strength training is not an exclusive cornerstone of sports performance or injuries. The World Health Organization (WHO) has provided global recommendations for the general population relevant to the prevention of non-communicable diseases. They recommended at least 150 min of moderate-intensity aerobic physical activity (3–5.9 metabolic equivalent tasks, METs) [25], with muscle strengthening activities involving major muscle groups on two or more days a week [230–233]. The biological principles underlying these global recommendations rely on the unique multi-systemic and multidimensional benefits of exercise [26] (see Fig. 1), its inexpensive adoption, and natural human responsiveness [27]. To mention the most salient point, recent evidence showed that vigorous physical activity has potential anti-tumorigenic properties [28]. In fact, it is associated with larger reductions in all-cause mortality [25] and cancer mortality [29, 30]. Specifically, resistance training alone was associated with 21% lower all-cause mortality [31]. Furthermore, patients with breast, colorectal, and prostate cancer involved in superior levels of exercise following cancer diagnosis, were associated with a 28–44% reduced risk of cancer-specific mortality, a 21–35% lower risk of cancer recurrence, and a 25–48% decreased risk of all-cause mortality [32, 33].

In this narrative review, we focus on the available literature related to strength training and musculoskeletal health, with the aim of providing practical recommendations in line with best practice for healthcare professionals involved in orthopaedic and sports medicine. Clear prescription details will be outlined to foster the best possible biological adaptations and thus, facilitate the use of strength training within all populations. In doing so, we will first outline the key principles underpinning mechano-transduction to illustrate how the body converts mechanical loading into cellular responses, before finally providing evidence-based recommendations for the safe interdisciplinary application of strength training across different populations.

2 Strength, Mechano-Transduction, and the Neuroendocrine System

Strength training has been shown to demonstrate a superior, dose-dependent and safe risk reduction strategy for acute and overuse sports injuries [34]. Information regarding the underpinning qualities of muscular strength development and the interaction of both cellular and metabolic processes in response to specific mechanical loading will first be discussed. Strength training's wide application to improve musculoskeletal tissues, and its role in the regulation and prevention of systemic disorders will then be examined.

2.1 Underpinning Factors

The development of muscular strength can be broadly divided into morphological and neural factors [10]. The maximal force generated by a single muscle fibre is directly proportional to its cross-sectional area (CSA) (number of sarcomeres in parallel) [35, 36], and by the muscle fibres' composition [2, 9, 10, 37]; specifically, type II fibres (IIa/IIx) have a greater capacity to generate power per unit of CSA, than the relatively smaller type I fibres. Architectural features such as longer fascicle length and the pennation angle also affect the force generating capacity of the muscle. Longer fascicle length allows more force production through an optimal length-tension relationship [10]. The number of sarcomeres in series influences a muscle's

Fig. 1 Multi-systemic benefits of strength training

contractility and the rate at which it can shorten. As pennation angle increases, more sarcomeres can be arranged in parallel, thus improving the muscle force generating capacity [10]. Greater pennation angles are more common in hypertrophied than in normal muscles. In regards to neural factors, the size principle dictates that motor unit (MU) recruitment is related to MU type, and that MUs are recruited in a sequenced manner based on their size (smallest to largest) [38]. Thus, the availability of high-threshold MUs is advantageous for higher force production. Furthermore, a higher rate of neural impulses (firing frequency) and the concurrent activation of multiple motor units (motor unit synchronization) enhance the magnitude of force generated during a contraction. These, together with an effective neurological system and intermuscular coordination (i.e., appropriate magnitude and timing of activation of agonist, synergist, and antagonist muscles) permit maximal force production [2, 9, 10, 37, 39, 40]. The development of these specific features underpinning improved force capacity is determined by the mechanical stimuli applied to the musculoskeletal system. Indeed, the musculoskeletal system not only enables locomotion and the transmission of forces for functional movements, but

also provides protection to vital organs. Furthermore, the musculoskeletal system stores and secretes key substances (e.g., amino acids, glucose, myokines, ions, etc.) that regulate whole body metabolism [41, 42].

Given their mechanical role, musculoskeletal tissues are capable of responding and adapting to mechanical forces via a process called mechano-transduction [43]. The body converts mechanical loading into cellular responses, which in turn, promotes structural changes in tissue mass, structure, and quality [44]. For example, an appropriate increase in mechanical loading of skeletal muscle results in an augmented skeletal muscle mass (i.e., increased CSA). The same rules apply for bone and tendon properties, which are in large part, dependent on skeletal muscle-derived mechanical loading [41]. Both acute and chronic mechanical stressors may temporarily compromise the body's "allostasis". This refers to the process by which the body responds to stressors and maintains homeostasis [45, 46], with the neuroendocrine system responsible for regulating the maintenance of an optimal catabolic/anabolic state. Dysregulation induced by allostatic overload has been associated with the breakdown of musculoskeletal tissues, inflammation [47, 48], and delayed tissue healing [49]. The neuroendocrine

system plays an important role not only in acute exercise performance, but also in tissue growth and remodelling. Relevant to mechano-transduction, the endocrine system secretes hormones into the circulatory system that are generally categorised as catabolic, leading to the breakdown of muscle proteins (e.g., cortisol), or anabolic (e.g., testosterone), leading to the synthesis of muscle proteins [50]. Muscle protein synthesis, recovery, and adaptation are the results of the dynamic interaction between these anabolic and catabolic hormones [51]. Although several factors such as exercise selection, intensity and volume, nutritional intake and training experience appear to influence the acute testosterone response [50–52], it has been shown that compound exercises, such as weightlifting exercises, squats, and deadlifts, are capable of producing larger elevations of testosterone than isolation exercises [52–54]. Furthermore, programmes characterized by moderate load, high total volume load and short rest periods (i.e., hypertrophy schemes) may produce substantial elevations in total testosterone, thus reinforcing the importance of specific exercise prescription to reach the targeted physiological adaptation [51, 52]. Similarly, increases in acute cortisol levels tend to be influenced by high volume programs, and not by typical strength training protocols [51, 55], thus altering the testosterone/cortisol ratio [56, 57].

Understanding the coupling of the mechanical stimuli into molecular responses appears vital for regenerative medicine applied to musculoskeletal disorders and for primary prevention strategies in a wide range of health issues and medical specialties. Mechanical forces may be manipulated in such a way that maximises the positive body responses within a predictable physiological timeframe. The next section includes relevant information for interdisciplinary care.

3 Multi-Systemic Benefits

Physical inactivity increases the risk of type 2 diabetes, cardiovascular diseases (CVD), colon cancer, postmenopausal breast cancer, dementia, and depression [58–60]. Furthermore, physical inactivity is associated with abdominal adiposity, which may carry the detrimental effects of visceral fat and persistent systemic low-grade inflammation [61, 62]. It is suggested that the skeletal muscles counteract the harmful effects of inactivity via release of specific myokines, such as myostatin, leukemia inhibitory factor (LIF), interleukin (IL)-6, IL-7, brain-derived neurotropic factor (BDNF), insulin-like growth factor 1 (IGF-1), fibroblast growth factor 2 (FGF-2), follistatin-related protein 1 (FSTL-1) and irisin [63]. Therefore, contracting skeletal muscles may be capable of releasing protective factors into the circulatory system during exercise. This may then mediate metabolic and physiological responses in other organs, such as the adipose tissue, liver, the cardiovascular system, and the brain [63]. Increased energy expenditure via resistance training can lead to a decrease in abdominal fat and specifically visceral fat, improving the catabolism and hydrolysis of very low-density lipoprotein-triglycerides [61]. These changes in body composition decrease inflammatory products, thus reducing the risk of developing multiple associated chronic diseases such as type 2 diabetes and CVD [31]. Furthermore, resistance training improves mitochondrial function in skeletal muscles, oxidative and glycolytic enzyme capacity, and glucose homeostasis, thus leading to decreased blood glucose [64] and improved type 2 diabetes symptoms [31, 61]. Also, resistance training is associated with reduced treatment side effects in cancer patient [33, 65, 66]. The anti-tumorigenic effects of exercise appear to be related to the suppression of cancer cells growth, restriction of inflammatory signalling pathways in myeloid immune cells, and regulation of acute and chronic systemic inflammatory responses [28, 67, 68].

Further benefits of resistance training include a reduction in anxiety (overall mean effect $\Delta = 0.31$) [69] and depressive symptoms, with a moderate effect size of 0.66 (95% CI 0.48–0.83) [70, 71]. Mental health benefits may be underpinned by the social interactions typically experienced during exercise and by the positive expectations toward exercise [72]. However, alterations in the hypothalamic pituitary adrenal (HPA) axis and in the neural circuitry involved in affective, behavioural, and cognitive processes have been documented in anxiety and depression-related disorders [73]. Although still speculative, strength training may affect the HPA axis through modulation of cortisol activity [74] and may have antidepressant effects through circulation of neurotrophins such as BDNF [26] and growth factors such as the IGF-1 [75]. Considering that sleep disturbance is one of the cardinal symptoms of depressive illness, it is not surprising that chronic resistance training in isolation also improves subjective sleep quality and day-time function, with moderate-to-large effect sizes [76].

Furthermore, there is strong evidence that exercise, including strength training, delivered within a biopsychosocial approach, is effective for musculoskeletal pain [77–79]. From a neurobiological perspective, it can strengthen central pain inhibitory pathways and the immune system response to potentially nociceptive stimuli [80–85].

In regard to coronary heart disease, progressive resistance training provides improvement in cardiorespiratory function comparable to aerobic training alone. When combined, they offer more substantiated improvements in both fitness and strength [86]. Resistance and aerobic training seem to increase the number of a specific subset of stem cells, broadly referred as circulating angiogenic cells (CAC). This enhances the vascular endothelium regeneration and angiogenesis, thus improving myocardial perfusion and lowering

the risk of cardiovascular diseases [26, 87]. Also, systolic and diastolic blood pressure may be significantly lowered by dynamic and isometric resistance training [88].

3.1 The Effect of Strength Training on Cartilage Health

The connective tissue that lines the ends of bones in all diarthrodial joints is called articular cartilage. Its role is to support and distribute forces generated during joint loading [89]. The articular surface is covered with hyaline cartilage, which is avascular, firm, yet pliable. It adapts its structure under forces but may recover its original shape on the removal of such forces. Of note, the ability of cartilage to repair is somewhat limited, which is mainly the result of its avascularity [90]. Differences in cartilage morphology between individuals cannot be readily explained by variability in mechanical loading history. It seems that mechanical stimulation does not play a significant role in cartilage regulation, with evidence to suggest that cartilage thickness is strongly determined by genetics [91]. Although it has been demonstrated that immobilisation reduces cartilage thickness (range 5–7%) [92], the adaptive functional ability of human cartilage in relation to exercise does not seem to be linear [91]. Interestingly, Hudelmaier et al. [93] found that thigh muscle CSA (which is a modifiable factor) is a good and independent predictor of cartilage morphology in both young and elderly adults. Similarly, Ericsson et al. [94] showed that lower thigh muscle strength 4 years after partial meniscectomy was associated with more severe radiographic osteoarthritis (OA) in the medial tibiofemoral compartment of the operated and the contralateral knee 11 years later, suggesting that muscle strength can help to preserve joint integrity.

For years, changes in the articular surface have been erroneously deemed the only cause of symptoms of patients suffering of OA. Compelling evidence shows the coexistence of multiple comorbidities such as obesity, cardiovascular diseases, diabetes, and metabolic syndrome in OA patients [21, 95]. Metabolic disturbances, chronic low-grade inflammation, and vascular endothelial dysfunction appear to be important factors in OA development and progression [21, 96]. Consistent with these findings, a negative correlation between knee cartilage volume and the concentration of circulating inflammatory cytokines, such as IL-6 and TNF, as well as C-reactive protein (CRP) has been demonstrated [95]. Therefore, contemporary evidence frames the definition of OA within a biopsychosocial model, in which multidimensional aspects modulate inflammatory processes and tissue sensitivity [97, 98]. Among these potential factors, recent reviews stated that knee extensor muscle weakness is a risk factor for knee OA [98, 99]. Segal et al. [100] found that thigh muscle strength did not predict incident radiographic, but did predict incident symptomatic, knee OA. In contrast, Thorstensson et al. [101] showed that reduced functional performance in the lower extremity predicted development of radiographic knee OA 5 years later among people aged 35–55 with persistent knee pain and normal radiographs at baseline. Pietrosimone et al. [102] found that higher levels of quadriceps strength correlated with higher physical activity in knee OA patients ($r = 0.44$; $r^2 = 0.18$).

Clinical guidelines for knee OA recommend strength training as one of the key elements of OA management [98, 103]. Indeed, the systematic review and meta-analysis conducted by Juhl et al. [104] showed that more pain and disability reduction occurred with quadriceps specific exercise than general lower limb exercise (standardized mean difference [SMD] 0.85 versus 0.39, and 0.87 versus 0.36, for pain and disability, respectively). Strength training should be an integral component of OA management together with education, weight loss, increase of lean mass, and improvement of aerobic capacity [103]. Beyond the aforementioned benefits on pain and disability levels, Bricca et al. [105] showed that loading the knee joint (via strength training) was safe and provided no detrimental effects for articular cartilage in people at increased risk of, or with, knee OA. Although the dosage is still unclear [106], potential beneficial mechanisms may be related to stiffening of the pericellular and inter-territorial matrix in response to dynamic loading [107], increased cartilage volume and glycosaminoglycan [105], and the protective role of muscle strength against cartilage loss [108].

3.2 The Effect of Strength Training on Bone Health

Bone tissue regulates metabolic demands on the skeleton largely through calciotropic hormones (vitamin D3, parathyroid hormone, and calcitonin) [109]. Secondly, it maintains the structure needed to withstand daily loading. These structural functions are determined by genetic factors as well as adaptation mechanisms to the loading environment, which are mediated by osteoprogenitor cells, including stromal cells, osteoblasts, and osteocytes [110, 111]. Osteocytes are believed to be the critical mechanical sensor cells. Their stimulation cannot be derived directly from matrix deformation, as the required magnitude of strains is so high that it would cause bone fracture [112, 113]. Therefore, it appears that mechanical loading induces the dynamic flow of the pericellular interstitial fluid in the lacunar-canalicular system. This seems to contribute significantly to osteocyte mechano-transduction and bone remodelling process [114].

Improved bone tissue mass provides higher structural strength and better protection against fractures [91]. Hence, failure to maintain a positive bone adaptation needed to withstand daily loading might be used to define osteoporosis

[110]. Indeed, according to Wolff's Law, a sufficient stimulus needs to be applied to the bone tissue to promote a specific magnitude of positive adaptation [115]. Contrary to societal misconceptions, bone responds positively to mechanical loads that induce high-magnitude strains at high rates or frequencies [116–118]. Indeed, despite being common advice from healthcare professionals, data showed that regular walking has no significant effect on preservation of bone mineral density (BMD) at the spine in postmenopausal women [119]. In contrast, Watson et al. [120] demonstrated the superior benefits of high-intensity resistance and impact training (HiRIT) compared to a low-intensity exercise program (10–15 repetitions at $< 60\%$ 1RM) in postmenopausal women with osteopenia and osteoporosis. Specifically, after a first month of safe transition and familiarisation, a supervised HiRIT program was completed over an 8-month period, twice-weekly, for 30-min. Resistance exercises included compound movements such as a deadlift, overhead press, and back squat, performed in 5 sets of 5 repetitions at an intensity of 80–85% 1RM. Impact loading was applied via jumping chin-ups with drop landings. HiRIT was significantly ($p \leq 0.001$) superior compared to the control group for lumbar spine BMD ($+2.9\% \pm 3.0\%$ for exercise group versus $-1.2\% \pm 2.3\%$ for control; 95% CI 2.1% to 3.6% versus -1.9% to -0.4%) and femoral neck BMD ($+0.1\% \pm 2.7\%$ versus $-1.8\% \pm 2.6\%$; 95% CI -0.7% to 0.8% versus -2.5 to -1.0%) and physical function (lumbar and back extensor strength, timed up-and-go test, 5 times sit to stand test, functional reach test, and vertical jump). Furthermore, it did not increase the risk of vertebral fracture, and was associated with a clinically relevant improvement in thoracic kyphosis [121]. Similar results have been reported in a meta-analysis including 1769 postmenopausal women [122]. Combined resistance and impact training (i.e., jumping, skipping, hopping) are estimated to promote clinically significant gains (almost 1.8 and 2.4%) in hip and spine BMD in postmenopausal women [122]. Considering that in the first few years after menopause women lose up to 5% of bone mass annually, smaller changes may be considered a valuable result to counteract the decline in bone mass during the aging process [123]. This further highlights the effectiveness of progressive resistance training combined with high-impact or weight-bearing exercises in increasing BMD at the femoral neck and lumbar spine. The cumulative body of evidence shows that the greatest skeletal benefits to the spine and hip are provided by progressive resistance training [124, 125] and can be achieved with high magnitude of loading (around 80–85% 1 RM), performed at least twice a week, targeting large muscles crossing the hip and spine through multijoint movements (e.g. squats and deadlifts) [126, 127]. Such intervention may show positive changes after 4 or 6 months, although greater magnitudes are expected when the intervention is continued for more than 1 year. Progressive resistance training, combined with weight-bearing impact training, can be implemented among different populations, with men and premenopausal women showing consistently positive adaptations [123, 128–130].

The transition from childhood to adolescence is critical for bone mineral accrual. During this phase, growth hormone (GH) and IGF-I are major contributors to bone growth [131]. Participation in sports that emphasize weight-bearing, high-impact and multiplanar-impact (e.g., soccer and racquet games) exercises promote peak bone mass and geometry [132]. Exposure to mechanical loading has substantial benefits not only in youth. It also appears to translate to greater bone strength over a lifetime [133], with consequent reduced risk of fracture, as well as potential delay in osteoporosis development [134]. Consistently, research has shown that youth athletes exposed to high or unusual impact weight-bearing sports with rapid rates of loading have superior bone mass at loaded skeletal sites compared to non-athletes or athletes in non-weight-bearing or lower impact sports [127]. For example, Courteix et al. [135] found that elite pre-pubertal female gymnasts displayed significantly ($p \leq 0.05$) higher BMD at mid-radius (+15.5%), distal radius (+33%), L2-4 vertebrae (+11%), femoral neck (+15%) and Ward's triangle (+15%) than swimmers and active peers. This further reinforces how bone mineral accrual responds positively to physical activity and specific sites of impact loading. Collectively, the available data strongly suggest to include exercise that is weight-bearing and characterized by impact loading in youth to promote and maintain bone health over one's lifetime [131].

Stress fractures in the lower limb account for 80%–90% of all stress fractures, representing between 0.7% and 20% of all sports medicine injuries [136]. The proposed mechanism underpinning stress fractures appears to be related to an imbalance between the rate of stress-induced micro-fractures and the rate at which bone repairs [136]. Although it is important to recognise their multifactorial pathophysiology, Schnackenburg et al. [137] showed a correlation between impaired bone quality, particularly in the posterior region of the distal tibia, and decreased muscle strength with lower limb stress fractures in female athletes. Clark et al. [138] revealed that lower grip strength correlated with higher risk of upper limb fractures (odds ratio 2.10, 95% CI 1.23 to 3.31) in active young people aged 12–16 years. They also showed that muscle strength was positively associated with BMD, BMC, or bone area. Popp et al. [139] analysed competitive distance runners with and without a history of stress fracture. Lower cortical bone strength, cortical area and smaller muscle CSA were present in runners with a history of stress fracture. Hoffman et al. [140] found that military recruits who were one standard deviation below the population mean in both absolute and relative strength had a five times greater risk for stress fracture than stronger recruits.

This is probably related to increased BMD associated with greater strength levels.

3.3 The Effect of Strength Training on Tendon Health

The tendon is a connective tissue that transmits the force exerted by the corresponding muscle to the skeleton [141]. Its key role is to store, recoil, and release energy while maintaining optimal efficiency in power production [142]. Hence, tendon stiffness (i.e., the slope of the force–elongation relationship or the resistance to deformation in response to an applied force) plays a critical role in athletic performance, stretch shortening cycle (SSC) activities, and movement economy [141]. Changes in tendon stiffness are a consequence of periods of increased mechanical loading. Alterations of the tendon material (i.e., increase of Young's modulus) and morphological properties (i.e., increase in CSA) are the two underpinning mechanisms [143]. Excessive mechanical loading is commonly considered an important factor in the development of tendinopathy, which is an umbrella term that indicates a nonrupture injury in the tendon or paratendon that is exacerbated by mechanical loading [144]. Clinical features are activity-related pain, focal tendon tenderness, and reduced load capacity and performance [145, 146]. A disconnection between tendon structure and symptoms in tendinopathy exists [147, 148], thus confirming multifactorial aspects contributing to its occurrence and persistence [149]. Nonetheless, loading protocols have been shown to be effective in the management of this condition [150, 151]. Evidence-based recommendations for an effective stimulus for tendon adaptation in healthy adults suggest high-intensity loading (85–90% of maximal voluntary isometric contraction [MVIC]) applied in five sets of four repetitions, with a contraction and relaxation duration of 3 s each, and an interset rest of 2-min [141]. This has been shown to increase maximal strength, tendon stiffness, Young's modulus, and tendon CSA [141, 143, 152, 153]. Eccentric actions are the most commonly used loading schemes in the management of tendinopathies, despite their non-superiority to other loading programmes [154–157]. The load employed is usually less than the concentric 1RM, which is in contrast with the documented benefits of supramaximal eccentric training stimuli [158, 159]. Similarly, in the absence of clear supporting evidence, isometric exercise has recently become the latest debated trend in tendon rehabilitation in the initial phase [160–162]. Overall, key factors such as time under tension and load/intensity are missing in most tendinopathy studies [150, 154, 163], thus making unclear which physical adaptation is targeted and limiting the synthesis regarding optimal doses into evidence based recommendations [22]. In fact, the magnitude and duration of the force application on the tendon appear more relevant than the type of contraction [141]. This highlights the need for adequately designed studies to improve knowledge within this field [23].

Achilles tendinopathy (AT) is one of the most common tendinopathies with an incidence rate of 2.35 per 1,000 within the general adult population and a prevalence of 36% among recreational runners [164]. Reduced plantarflexor strength has been recognised to be a significant risk factor for AT [165, 166]. Cross-sectional studies confirm large deficits in plantarflexor torque between AT symptomatic subjects and healthy controls [167, 168]. Although it may appear intuitive that strength training could be adopted as a primary prevention strategy for reducing the risk of tendinopathies, current literature to support this notion is lacking. A recent systematic review found limited evidence for the efficacy of preventative interventions for tendinopathies [169]. Among the studies examined, strength training was employed with much lighter loads and subsequently higher repetition ranges [170] and thus did not meet evidence based recommendations for an effective stimulus for the tendon [141, 143]. Therefore, further prospective studies are needed in this area.

Loading programmes have been shown to positively enhance structural adaptations among patients presenting with tendinopathy [150, 164]. However, Heinemeier et al. [171] found that renewal of adult core tendon tissue is extremely limited especially following adolescence. Kubo et al. [172] revealed that length and CSA of the patellar tendon correlated with increases in body size during growth, whereas Young's modulus was lower in the pre-pubertal phase compared to junior high school students and adults. Waugh et al. [173] demonstrated that dimensional and maturational aspects of Achilles tendon stiffness were underpinned not only by age, but also by body mass and peak force production, thus reinforcing the correlation between tendon stiffness and muscular force capacity in childhood and adolescence. In this regard, it should be noted that safe improvements in muscular strength are possible in youth of all ages and stages of maturation with resistance training [174]. Concomitant with a reduction in the number of sport-related injuries [175], this reinforces the importance of engagement in youth athletic development programmes in the pre-pubertal years with continuation throughout the later stages of maturation and into adulthood [176, 177].

3.4 The Effect of Strength Training on Muscle Health

Skeletal muscles are characterized by myofibres and connective tissue. The myofibres are responsible for the contractile function of the muscle, whereas the connective tissue supply the structure that binds the individual muscle cells together during muscle contraction [178]. Both mechanical and

Table 1 Summary of benefits for various musculoskeletal tissues and disorders associated with strength/resistance training

Musculoskeletal tissue	Function	Potential beneficial mechanisms	Specific recommendation	Examples of application for common related conditions
Cartilage	Support and distribution of forces generated during joint loading	Stiffening of the pericellular and interterritorial matrix Increase in cartilage volume and glycosaminoglycan Protection against cartilage loss	Specific exercise for targeted area appears relevant Inclusion in multidimensional care management Potential benefits associated with increased CSA	**Knee osteoarthritis** Joint loading exercises Optimal programme characteristics not identified yet Recommended frequency 3 times weekly with a duration of at least 12 supervised sessions
Bone	Regulation of metabolic demands Structural maintenance to withstand loading	Increase in bone mineral density, bone mineral content, and bone area	To target large muscles Safe transition towards high loads ($\geq 80\%$ 1RM) Familiarisation with movement patterns Combination with impact loading exercises	**Osteopenia and osteoporosis** 5 sets of 5 repetitions, maintaining an intensity of 80-85% 1 RM performed at least twice per week
Tendon	Force transmission Storage, recoil and release of energy	Increase in tendon stiffness, Young's modulus and tendon cross-sectional area	To adopt muscle contraction intensities higher than 70% of MVC or RM Type of contraction (isometric, concentric, eccentric) not relevant Longer durations (≥ 12 weeks) more effective	**Reduction of tendon stiffness and Young's modulus** 5 sets of 4 repetitions with high-intensity loading (85–90% MVIC) with a contraction and relaxation duration of 3 s each, and an interset rest of 2 min. To be performed 3 times per week
Muscle	Contraction to produce force and motion	Increase in myofibrillar cross sectional area (CSA) of type I/II fibres, lean muscle mass, fascicle length and pennation angle	Individualised and periodised approach Multi-joint exercise per major muscle group in elderly Type of contraction relevant for muscle fibres architectural adaptations	**Sarcopenia** 2–3 sets of 1–2 multi-joint exercises per major muscle group, with intensities of 70–85% of 1RM, 2–3 times per week

CSA cross-sectional area, *RM* repetition maximum, *MVC* maximal voluntary contraction, *MVIC* maximal voluntary isometric contraction

metabolic stress can trigger muscle adaptation and growth [143]. A protein kinase called the mechanistic/mammalian target of rapamycin (mTOR) appears crucial in the pathway through which mechanical stimuli regulate protein synthesis and muscle mass [41]. Morphological factors such as CSA, muscle fibre composition, pennation angle, and fascicle length, are important in force production. Loss of skeletal muscle mass, reduced motor unit (MU) discharge rate, and impaired function are primarily associated with aging. This is defined as either sarcopenia (age-related loss of skeletal muscle mass and function) or dynapenia (age-associated loss of muscle strength that is not caused by neurologic or muscular diseases) [179, 180]. The reduction of MU discharge rate and type 2 muscle fibres lead consequently to reduced RFD, which is associated with impaired functional capacity during daily tasks (e.g. balance recovery during tripping) [3, 181, 182]. Pijnappels et al. [183] showed that the identification of individuals most at risk of falling could be predicted by their maximal leg press push-off force level. In older adults, lower muscle strength is also associated with an increased risk of dementia [184], loss of independence, and mortality [185–188]. However, the rate of strength decline is dependent on age and physical activity levels. Indeed, individuals participating in strength training can significantly attenuate the loss of muscle mass and strength, and their undesirable consequences [189]. Strong evidence suggests that an appropriately designed resistance training program for older adults should include an individualised and periodized approach working toward 2–3 sets of 1–2 multi-joint exercises per major muscle group, achieving intensities of 70–85% of 1RM, 2–3 times per week [126]. Strength training is a feasible and effective strategy to counteract muscle weakness [190], physical frailty, age-related intramuscular adipose infiltration, decline in physical function, risk for falls, and reduction in CSA [189, 191]. These benefits are underpinned by the ability of strength training to countermeasure age-related changes in muscle and central nervous system function. Specifically, strength training is highly effective in improving MU discharge rate, reducing loss of type 2 fibres, and enhancing RFD and muscle strength, thus explaining the functional benefits in the older population, especially in frail elderly [3, 181].

Overall, strength training increases neural drive, intermuscular coordination, myofibrillar CSA of type I and II fibres, lean muscle mass, and pennation angle [2, 10, 11]. Not surprisingly, primary prevention strategies recommend the employment of strength training for the reduction of
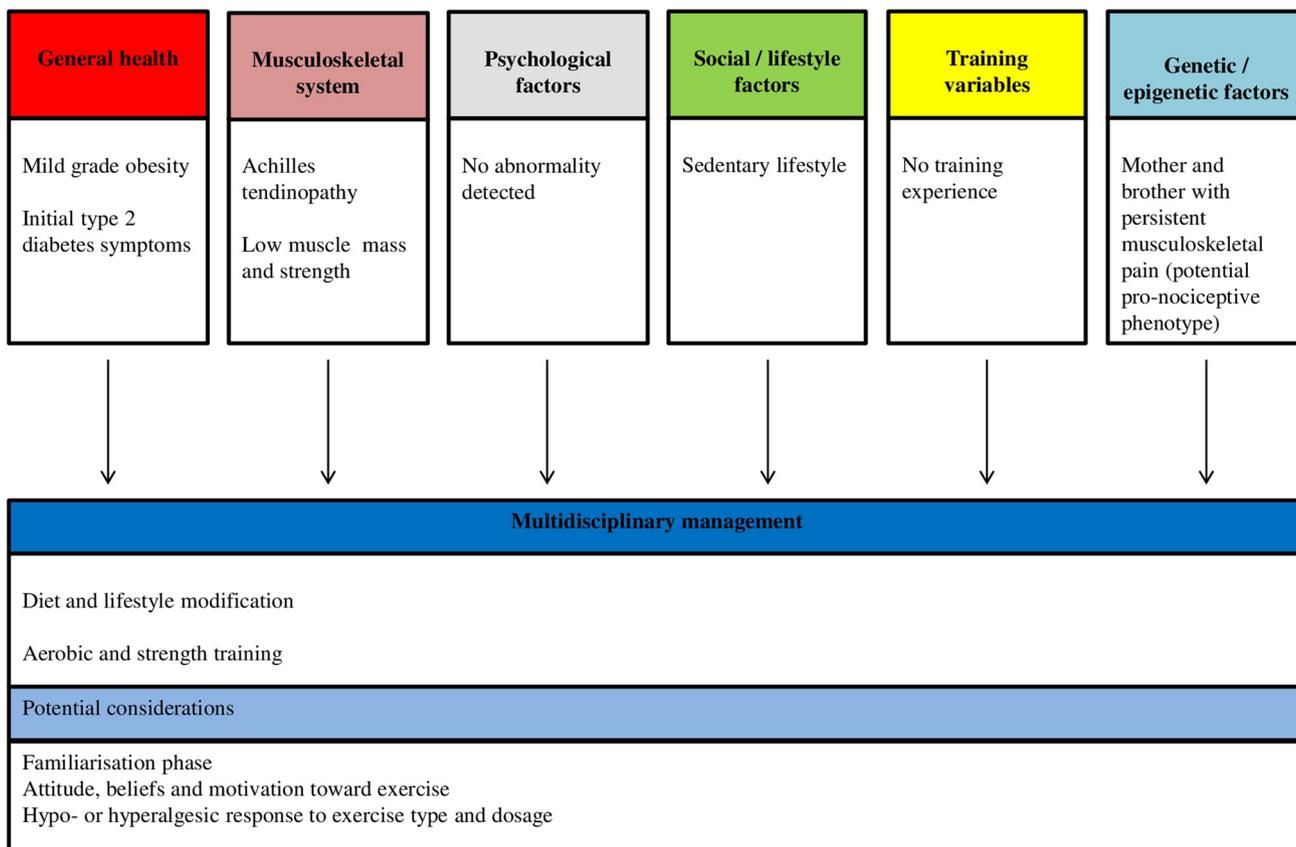
General health	Musculoskeletal system	Psychological factors	Social / lifestyle factors	Training variables	Genetic / epigenetic factors
Mild grade obesity Initial type 2 diabetes symptoms	Achilles tendinopathy Low muscle mass and strength	No abnormality detected	Sedentary lifestyle	No training experience	Mother and brother with persistent musculoskeletal pain (potential pro-nociceptive phenotype)

Multidisciplinary management

Diet and lifestyle modification

Aerobic and strength training

Potential considerations

Familiarisation phase
Attitude, beliefs and motivation toward exercise
Hypo- or hyperalgesic response to exercise type and dosage

Fig. 2 Profile of a middle-aged man with mid-portion Achilles tendinopathy

acute sports injuries [15, 34]. Among these, muscle injuries are very common in sports [34, 192], constituting 31% of all injuries in elite football [193]. For example, the Nordic hamstring exercise (NHE) (i.e., a form of supramaximal eccentric loading) has been shown to significantly reduce the risk of hamstring injuries [192, 194–196], with long-term benefits associated with increases in fascicle length and improvements in eccentric knee flexor strength [197]. The systematic review and meta-analysis conducted by van Dyk et al. [198] showed that programmes including the NHE reduced hamstring injuries by 51% in athletes across multiple sports. Zouita et al. [199] showed that strength training reduced the risk of injury in elite young soccer players during one season (estimated total injury rate per 1,000 h of exposure were: 0.70 for the experimental group and 2.32 for the control group). Of note, approximately 50% of the total injuries sustained were classified as "muscle strains", thus demonstrating the protective role of strength training on muscle tissues. Although not thoroughly consistent with strength training prescription over the study period, Haroy et al. [200] showed that a single exercise with different levels of targeting the adductors reduced the prevalence and risk of groin injuries in semi-professional Norwegian football

players by 41%. Considering the economic burden of muscle injuries in elite settings (e.g., a single hamstring injury resulting in ~17 days lost from training and competition is estimated to cost about €280,000 in elite soccer clubs) [197] and the importance of muscle tissue health for players' availability and performance, implementation of an accurate strength training schedule during the season appears vital. A summary of the benefits for various musculoskeletal tissues and disorders are depicted in Table 1.

4 Strength Training: Practical Applications

Researchers have challenged the existence of "non-responders" to exercise. Positive adaptations are influenced by multidimensional aspects such as genetic factors, fitness level, training history, nutritional intake, psychological and social states, sleep and recovery, age, weight, and prescribed training workload [27], and therefore, the magnitude of adaptations between individuals may differ. Thus, strength training prescription should begin with an accurate subjective and objective examination. This investigates training and injury history, general health status, coexistent comorbidities,
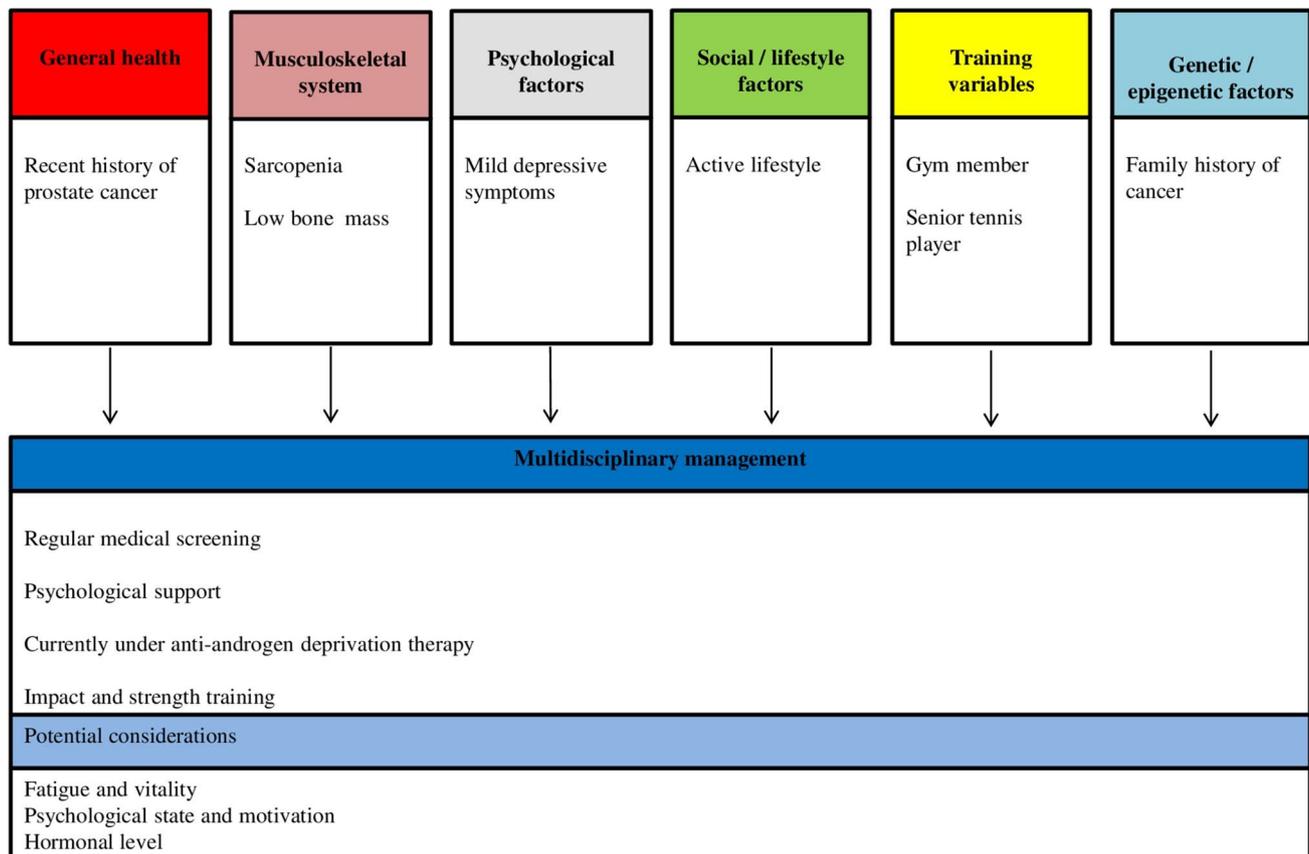

Fig. 3 Profile of an older man (73 years old) presenting with sarcopenia and a recent history of prostate cancer

single-joint and multi-joint strength evaluation and movement pattern analysis relevant to the potential proposed exercise programme. Clinical tools such as questionnaires and outcome measures may be implemented in the subjective examination to more accurately detect and discuss the significant aspects that may negatively counteract the expected positive adaptations and can be administered at specific timeframes at the judicious discretion of healthcare professionals. For example, specific questionnaires and outcomes measures can be adopted to monitor sleep [201] and stress levels [202, 203] over the course of an intervention. This transdiagnostic approach attempts to understand commonalities and shared mechanisms among different multidimensional aspects and to identify any adverse responses to

the planned intervention that may be driven by such factors [204]. This enables a stratified model of care (i.e., personalised medicine) to maximise treatment-related benefits, reduce risk of adverse events and increase healthcare efficiency [205] (see examples in Figs. 2, 3, 4).

This process allows a more complete understanding of the person, his/her past and current exposure to loading activities, quality of life, beliefs and attitude towards exercise, relevant impairment in mobility, potential site of loading, adequate skeletal muscle trophism and/or isolated strength deficits that may impair rapid exposure to high-load exercises; thus, requiring a period of familiarisation and anatomical adaptation via adoption of different loading schemes. For example, in untrained individuals sensitive
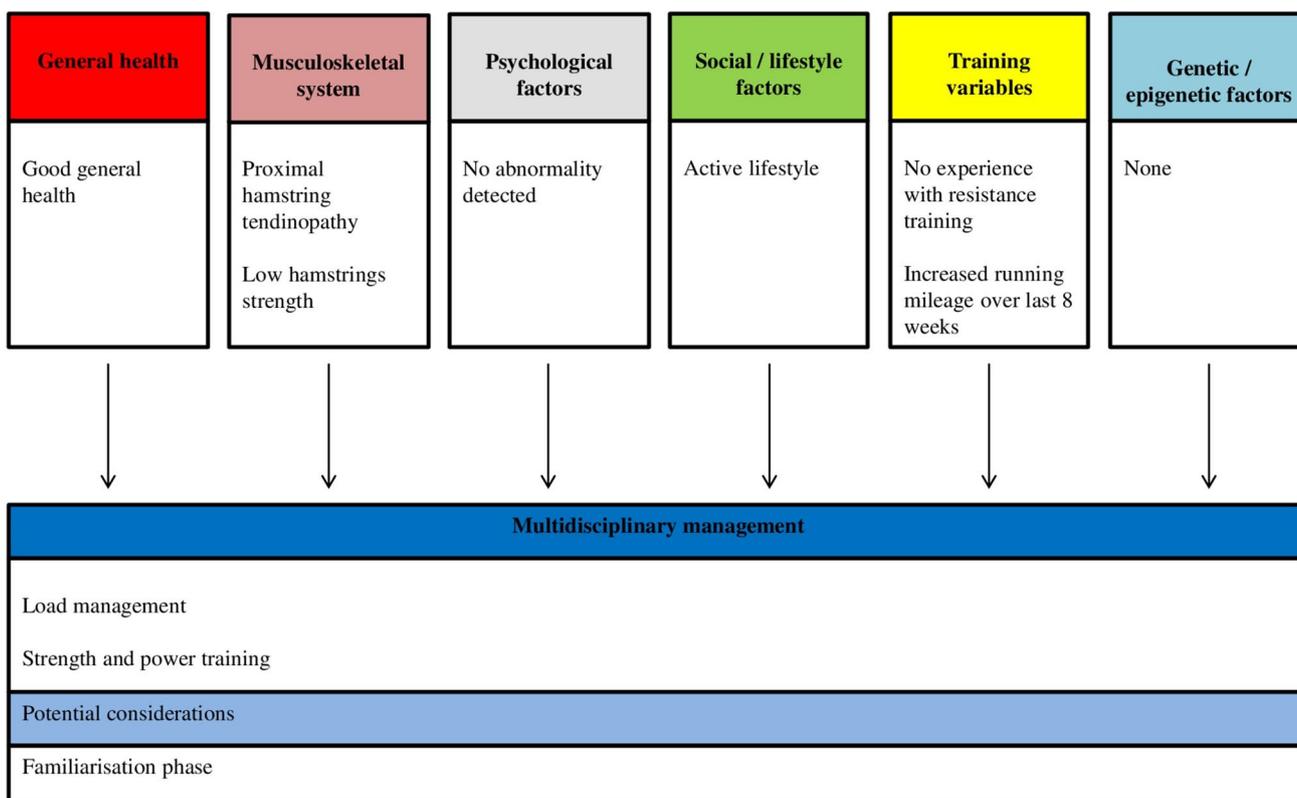

Fig. 4 Profile of a young runner (19 years old) with proximal hamstring tendinopathy preparing for the Marathon

Table 2 Suggested strength training variables when employing the traditional percentage fixed loading program (TL) or auto-regulated training (AR)

PROGRAM	REPETITIONS	SETS	LOAD		REST	FREQUENCY
TL	1–6	3–5	@80–100% 1RM		3–5 min	2–3/week
PROGRAM	RM ZONE	SETS	RPE 0-10	RIR	REST	FREQUENCY
AR	1–6	3–5	8–10	0–2	3–5 min	2–3/week

TL traditional loading, *AR* auto-regulated training, *RM* repetition maximum, *RPE* rate of perceived exertion, *RIR* repetitions in reserve
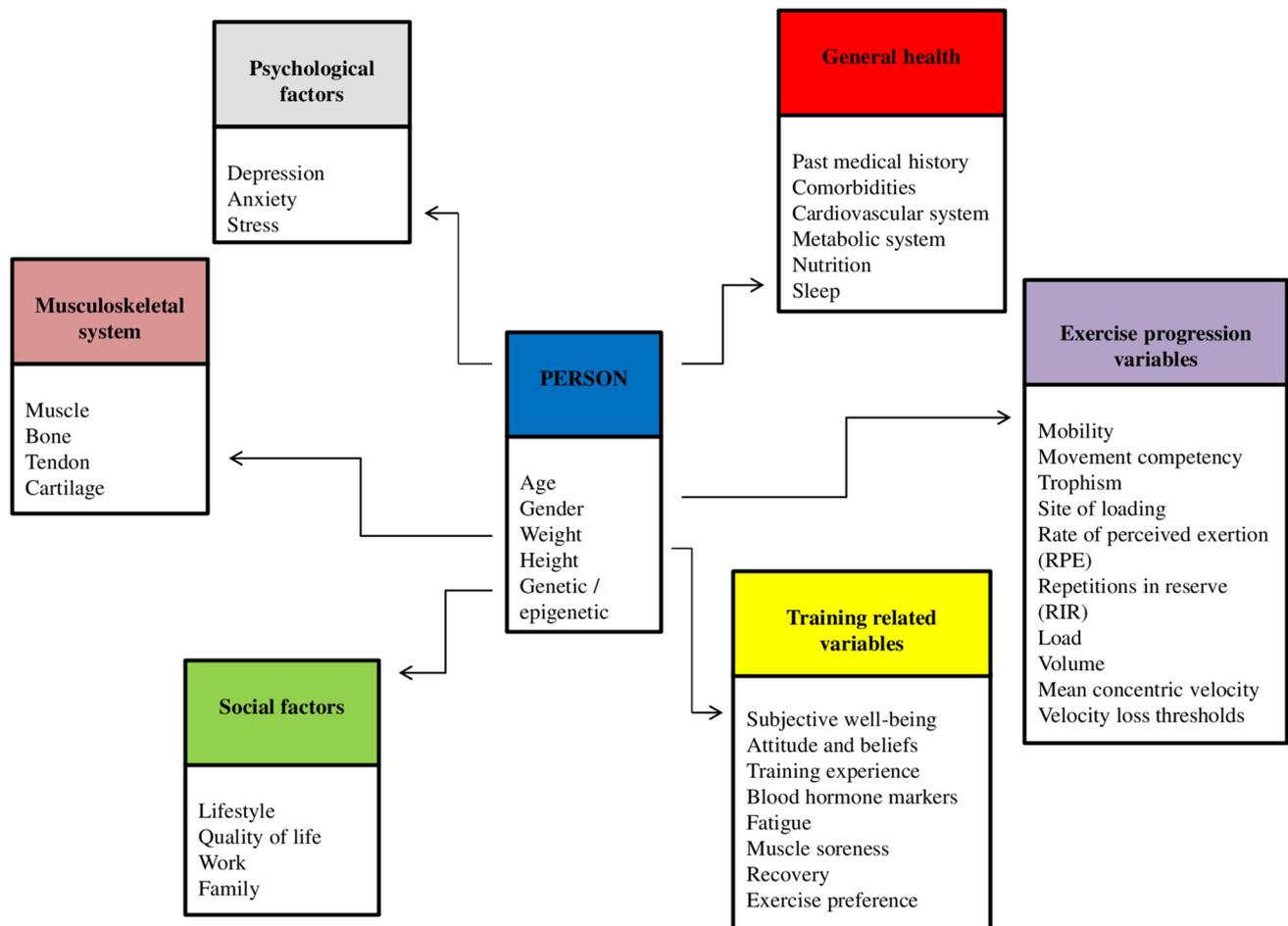
Fig. 5 Graphical representation of common subjective and objective variables that contribute to programming and progression decision making in strength training

to spinal axial loading, who cannot tolerate large external loads, bilateral exercises, such as the back squat can be confidently substituted with unilateral exercises due to similar effectiveness in lower body strength development, despite relative lower external loading [206]. When the goal is to elicit alterations in skeletal muscle hypertrophy in untrained individuals, current literature [24, 207–209] suggests training with a high level of effort, irrespective of load. Whereas momentary failure is important during low load training to capitalise on muscular adaptations, this does not provide any additional benefits when training at high resistance training loads. Hence, lighter loads can be initially lifted until failure to maximise MU recruitment, increase muscle size and increase strength (to a certain extent). With gradual training exposure and increasing resistance training experience, these can be progressed to higher load-lower repetition schemes without momentary failure, thus providing heightened neural impulses to maximise strength gains [208, 210–212].

Global recommendations suggest strength training should be performed two or more days per week [230–233]. Maximal strength can be defined as the upper limit of the neuromuscular system to produce force. Force production against an external resistance is an essential trainable ability [213]. It must be noted that in untrained individuals almost any resistance training exercise programme, load and method may increase strength, which is more likely attributable to neural adaptations in response to the new training stimulus [2, 24, 212, 214, 215]. However, progressive overload stimuli appear essential to promote further strength adaptations in more experienced individuals [24, 214, 234]. For these, current evidence indicates that prescription of maximal strength training should involve a load (or intensity) of 80–100% of the participant's 1RM, utilizing approximately 1–6 repetitions, across 3–5 sets, with rest periods of 3–5 min, and a frequency of 2–3 times per week [234]. This implies that loads are determined by percentages of 1RM, with testing potentially challenging when working

Table 3 Example of a potential strength training session for postmenopausal women with low bone mass (performed at least twice per week for an ideal duration of at least one year). The length of each phase, exercise selection and the progressions are chosen in accordance with the participant's weekly evaluation

Phase 1 -Familiarisation Training aim	Exercise	Fixed loading prescription	Auto-regulated training prescription	Impact loading
To ensure safe transition to high-intensity load; To familiarise with exercises and movement patterns	Goblet Squat ≫ Split Squat; Romanian Deadlift; Box Squat; Overhead press ≫ Press-up ≫ Bench Press; Seated Row ≫ Bent Over Rows	1 sets of 12 repetitions of ~50–60% 1RM; 2 min inter set rest	1 sets of 12 RM with RPE 4–6 and RIR 4–5; 1 min inter set rest	3 repetitions ×4 sets; Snap-downs ≫ jump to box ≫ standing broad jump ≫ depth land ≫ drop jump
Phase 2—Strength endurance emphasis Training aim	Exercise	Fixed loading prescription	Auto-regulated training prescription	Impact loading
To increase muscle mass, strength and musculotendinous stiffness; To facilitate safe transition to strength training emphasis	Split Squat ≫ RFESS ≫ Box Squat ≫ Trap-bar Deadlift; Romanian Deadlift; Overhead Press ≫ Press-up or Bench Press; Seated Row ≫ Bent Over Rows	3 sets of 8–12 repetitions of ~60–75% 1RM; 1–2 min inter set rest	3 sets of 10RM with RPE 6–7 and RIR 2–3; 2 min inter set rest	3 × 20 cm depth land during the first 6 interset rest periods; 2 broad jump during the last 6 interset rest periods
Phase 3—Strength emphasis Training aim	Exercise	Fixed loading prescription	Auto-regulated training prescription	Impact loading
To increase muscle mass, strength, rate of force development and musculotendinous stiffness; To improve motor unit discharge rate; To reduce loss of type II fibres; To increase bone mass, bone mineral content and bone mineral density	Trap-bar Deadlift; Romanian Deadlift; Overhead Press or Bench Press; Bent Over Rows	4 sets of 5 repetitions of > 85% 1RM; 3–5 min inter set rest	4 sets of 5RM with RPE 8–9 and RIR 1–2; 3–5 min inter set rest	4 countermovement jumps during the first 4 interset rest periods; 3 × 3 hurdles jump during the last 4 inter-set rest periods

RM repetition maximum, *RPE* rate of perceived exertion, *RIR* repetitions in reserve, *RFESS* rear foot elevated split squat, ≫ progress to these exercises during next cycle or perform these instead/if preferred and patient/client is competent

with load compromised patients and/or pain interference. Therefore, the adoption of an auto-regulated approach (AR), which is based on RM training zones, rate of perceived exertion (RPE) and repetitions in reserve (RIR) [216, 217], may appear more feasible and clinically advantageous throughout the training cycle. This also accounts for fluctuations in strength capabilities across a training mesocycle [216, 218], which can be influenced by the aforementioned multi-dimensional aspects. In experienced individuals, RPE/RIR scale can be used as a method to assign daily training load, aid in session to session load progression, and monitor individual rates of adaptation [216, 219]. Assessment of movement velocity may also be another valid alternative used to estimate the percentage of loading [220, 221]. This exploits the inverse linear relationship between load and mean concentric velocity (MCV). Indeed, providing that maximal concentric effort is applied during movement, MCV will decrease as magnitude of load increases, thus allowing estimation of relative training loads (%1RM) monitoring movement velocity [222]. In addition, different velocity loss (VL) thresholds across repetitions performed within a set may be also adopted to dictate mechanical and metabolic stress, hormonal responses and neuromuscular fatigue, thus inducing different adaptations. Small to moderate VL threshold (i.e., <20%) are recommended to maximise strength gains in resistance-trained individuals [223, 224]. For clarity of information, examples of loading schemes for strength training are depicted in Table 2. Common subjective and objective variables that contribute to programming and progression decision making are illustrated in Fig. 5.

The frequency and duration of a strength training program might be variable, although position statements and clinical guidelines for specific disorders and targeted populations are clearly outlined in the available literature [77, 122, 126, 127, 176, 189, 217, 225, 226]. However, significant changes in musculoskeletal tissues are generally evident after 8 to 12 weeks, although some studies observed increases in muscle mass after only 2 to 4 weeks [37]. This early increase in strength is likely caused by neuromuscular and connective tissue adaptations [227], whereas the early increases in muscle CSA may be the result of oedema [228]. For tendon adaptations, longer durations ($\geq$ 12 weeks) appear to be more effective [141]. An example of a potential strength training session is outlined in Table 3 and further examples can be found in our recent published work [229].

5 Conclusion

This article has briefly examined the mechanisms underpinning positive adaptations to strength training as well as potential benefits for the musculoskeletal system. An overview of training strategies to target these adaptations has also been discussed in both common musculoskeletal disorders and primary prevention strategies. The concepts expressed in this review may help healthcare professionals in understanding and promoting clear and evidence-based recommendations for strength training in musculoskeletal practice, sports medicine and a wide array of medical specialties. Therefore, shared interdisciplinary recommendations appear vital.

Compliance with Ethical Standards

References

1. Suchomel TJ, Nimphius S, Stone MH. The importance of muscular strength in athletic performance. Sports Med. 2016;46(10):1419–49.
2. Suchomel TJ, Nimphius S, Bellon CR, et al. The importance of muscular strength: training considerations. Sports Med. 2018;48:765–85.
3. Maffiuletti NA, Aagaard P, Blazevich AJ, et al. Rate of force development: physiological and methodological considerations. Eur J Appl Physiol. 2016;116(6):1091–116.
4. Rodriguez-Rosell D, Pareja-Blanco F, Aagaard P, et al. Physiological and methodological aspects of rate of force development assessment in human skeletal muscle. Clin Physiol Funct Imaging. 2018;38(5):743–62.
5. Seitz LB, Reyes A, Tran TT, Saez de Villarreal E, et al. Increases in lower-body strength transfer positively to sprint performance: a systematic review with meta-analysis. Sports Med. 2014;44(12):1693–702.
6. Keiner M, Sander A, Wirth K, et al. Long-term strength training effects on change-of-direction sprint performance. J Strength Cond Res. 2014;28(1):223–31.
7. Storen O, Helgerud J, Stoa EM, et al. Maximal strength training improves running economy in distance runners. Med Sci Sports Exerc. 2008;40(6):1087–92.
8. Haff GG, Nimphius S. Training principles for power. Strength Cond J. 2012;34(6):2–12.
9. Haff GG, Stone MH. Methods of developing power with special reference to football players. Strength Cond J. 2015;37(6):2–16.
10. Cormie P, McGuigan MR, Newton RU. Developing maximal neuromuscular power: part 1—biological basis of maximal power production. Sports Med. 2011;41(1):17–38.
11. Cormie P, McGuigan MR, Newton RU. Adaptations in athletic performance after ballistic power versus strength training. Med Sci Sports Exerc. 2010;42(8):1582–98.

12. Comfort P, Thomas C. Changes in dynamic strength index in response to strength training. Sports. 2018;6(4):176.

13. James LP, Gregory Haff G, Kelly VG, et al. The impact of strength level on adaptations to combined weightlifting, plyometric, and ballistic training. Scand J Med Sci Sports. 2018;28(5):1494–505.

14. Lauersen JB, Bertelsen DM, Andersen LB. The effectiveness of exercise interventions to prevent sports injuries: a systematic review and meta-analysis of randomised controlled trials. Br J Sports Med. 2014;48(11):871.

15. Malone S, Hughes B, Doran DA, et al. Can the workload-injury relationship be moderated by improved strength, speed and repeated-sprint qualities? J Sci Med Sport. 2019;22(1):29–34.

16. Zadro J, O'Keeffe M, Maher C. Do physical therapists follow evidence-based guidelines when managing musculoskeletal conditions? Systematic review. BMJ Open. 2019;9(10):e032329.

17. Bahr R, Thorborg K, Ekstrand J. Evidence-based hamstring injury prevention is not adopted by the majority of Champions League or Norwegian Premier League football teams: the Nordic Hamstring survey. Br J Sports Med. 2015;49(22):1466–71.

18. McCall A, Carling C, Nedelec M, et al. Risk factors, testing and preventative strategies for non-contact injuries in professional football: current perceptions and practices of 44 teams from various premier leagues. Br J Sports Med. 2014;48(18):1352–7.

19. Lowe A, Littlewood C, McLean S, et al. Physiotherapy and physical activity: a cross-sectional survey exploring physical activity promotion, knowledge of physical activity guidelines and the physical activity habits of UK physiotherapists. BMJ Open Sport Exerc Med. 2017;3(1):e000290.

20. Yona T, Ben Ami N, Azmon M, et al. Physiotherapists lack knowledge of the WHO physical activity guidelines. A local or a global problem? Musculoskelet Sci Pract. 2019;43:70–5.

21. Schulz JM, Birmingham TB, Atkinson HF, et al. Are we missing the target? Are we aiming too low? What are the aerobic exercise prescriptions and their effects on markers of cardiovascular health and systemic inflammation in patients with knee osteoarthritis? A systematic review and meta-analysis. Br J Sports Med. 2019. https://doi.org/10.1136/bjsports-2018-100231.

22. Holden S, Barton CJ. 'What should I prescribe?': time to improve reporting of resistance training programmes to ensure accurate translation and implementation. Br J Sports Med. 2019;53:264–5.

23. Czosnek L, Rankin N, Zopf E, et al. Implementing exercise in healthcare settings: the potential of implementation science. Sports Med. 2020;50(1):1–14.

24. Schoenfeld BJ, Grgic J, Ogborn D, et al. Strength and hypertrophy adaptations between low- vs. high-load resistance training: a systematic review and meta-analysis. J Strength Cond Res. 2017;31(12):3508–23.

25. Rey Lopez JP, Gebel K, Chia D, et al. Associations of vigorous physical activity with all-cause, cardiovascular and cancer mortality among 64 913 adults. BMJ Open Sport Exerc Med. 2019;5(1):e000596.

26. Fiuza-Luces C, Garatachea N, Berger NA, et al. Exercise is the real polypill. Physiology (Bethesda). 2013;28(5):330–58.

27. Pickering C, Kiely J. Do non-responders to exercise exist—and if so, what should we do about them? Sports Med. 2019;49(1):1–7.

28. Ruiz-Casado A, Martin-Ruiz A, Perez LM, et al. Exercise and the hallmarks of cancer. Trends Cancer. 2017;3(6):423–41.

29. Stamatakis E, Lee IM, Bennie J, et al. Does strength-promoting exercise confer unique health benefits? A pooled analysis of data on 11 population cohorts with all-cause, cancer, and cardiovascular mortality endpoints. Am J Epidemiol. 2018;187(5):1102–12.

30. Garatachea N, Santos-Lozano A, Sanchis-Gomar F, et al. Elite athletes live longer than the general population: a meta-analysis. Mayo Clin Proc. 2014;89(9):1195–200.

31. Saeidifard F, Medina-Inojosa JR, West CP, et al. The association of resistance training with mortality: a systematic review and meta-analysis. Eur J Prev Cardiol. 2019;26(15):1647–65.

32. Cormie P, Zopf EM, Zhang X, et al. The impact of exercise on cancer mortality, recurrence, and treatment-related adverse effects. Epidemiol Rev. 2017;39(1):71–92.

33. Hayes SC, Newton RU, Spence RR, et al. The Exercise and Sports Science Australia position statement: exercise medicine in cancer management. J Sci Med Sport. 2019;22(11):1175–99.

34. Lauersen JB, Andersen TE, Andersen LB. Strength training as superior, dose-dependent and safe prevention of acute and overuse sports injuries: a systematic review, qualitative analysis and meta-analysis. Br J Sports Med. 2018;52:1557–63.

35. Hornsby WG, Gentles JA, Haff GG, et al. What is the impact of muscle hypertrophy on strength and sport performance? Strength Cond J. 2018;40(6):99–111.

36. Taber CB, Vigotsky A, Nuckols G, et al. Exercise-induced myofibrillar hypertrophy is a contributory cause of gains in muscle strength. Sports Med. 2019;49(7):993–7.

37. Hughes DC, Ellefsen S, Baar K. Adaptations to endurance and strength training. Cold Spring Harb Perspect Med. 2017;8(6):a029769.

38. Henneman E, Somjen G, Carpenter DO. Excitability and inhibitability of motoneurons of different sizes. J Neurophysiol. 1965;28(3):599–620.

39. Clark BC, Mahato NK, Nakazawa M, et al. The power of the mind: the cortex as a critical determinant of muscle strength/weakness. J Neurophysiol. 2014;112(12):3219–26.

40. Del Vecchio A, Negro F, Holobar A, et al. You are as fast as your motor neurons: speed of recruitment and maximal discharge of motor neurons determine the maximal rate of force development in humans. J Physiol. 2019;597(9):2445–56.

41. Goodman CA, Hornberger TA, Robling AG. Bone and skeletal muscle: key players in mechanotransduction and potential overlapping mechanisms. Bone. 2015;80:24–36.

42. Thompson WR, Scott A, Loghmani MT, et al. Understanding mechanobiology: physical therapists as a force in mechanotherapy and musculoskeletal regenerative rehabilitation. Phys Ther. 2016;96(4):560–9.

43. Khan KM, Scott A. Mechanotherapy: how physical therapists' prescription of exercise promotes tissue repair. Br J Sports Med. 2009;43(4):247–52.

44. Warden SJ, Thompson WR. Become one with the force: optimising mechanotherapy through an understanding of mechanobiology. Br J Sports Med. 2017;51(13):989–90.

45. McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. Ann N Y Acad Sci. 1998;840:33–44.

46. McEwen BS. Central effects of stress hormones in health and disease: understanding the protective and damaging effects of stress and stress mediators. Eur J Pharmacol. 2008;583(2–3):174–85.

47. Hannibal KE, Bishop MD. Chronic stress, cortisol dysfunction, and pain: a psychoneuroendocrine rationale for stress management in pain rehabilitation. Phys Ther. 2014;94(12):1816–25.

48. Heim C, Ehlert U, Hellhammer DH. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. Psychoneuroendocrinology. 2000;25(1):1–35.

49. Alford L. Psychoneuroimmunology for physiotherapists. Physiotherapy. 2006;92(3):187–91.

50. Deschenes MR, Kraemer WJ, Maresh CM, et al. Exercise-induced hormonal changes and their effects upon skeletal muscle tissue. Sports Med. 1991;12(2):80–93.

51. Crowther B, Keogh J, Cronin J, et al. Possible stimuli for strength and power adaptation: acute hormonal responses. Sports Med. 2006;36(3):215–38.

52. Kraemer WJ, Ratamess NA. Hormonal responses and adaptations to resistance exercise and training. Sports Med. 2005;35(4):339–61.

53. Kraemer WJ, Staron RS, Hagerman FC, et al. The effects of short-term resistance training on endocrine function in men and women. Eur J Appl Physiol Occup Physiol. 1998;78(1):69–76.

54. Kraemer WJ, Fry AC, Warren BJ, et al. Acute hormonal responses in elite junior weightlifters. Int J Sports Med. 1992;13(2):103–9.

55. Williams AG, Ismail AN, Sharma A, et al. Effects of resistance exercise volume and nutritional supplementation on anabolic and catabolic hormones. Eur J Appl Physiol. 2002;86(4):315–21.

56. Fry AC, Schilling BK. Weightlifting training and hormonal responses in adolescent males: implications for program design. Strength Cond J. 2002;24(5):7–12.

57. Fry AC, Kraemer WJ. Resistance exercise overtraining and overreaching. Neuroendocrine responses. Sports Med. 1997;23(2):106–29.

58. Pedersen BK. The diseasome of physical inactivity—and the role of myokines in muscle–fat cross talk. J Physiol. 2009;587(Pt 23):5559–68.

59. Pedersen BK. The physiology of optimizing health with a focus on exercise as medicine. Annu Rev Physiol. 2019;10(81):607–27.

60. McLeod JC, Stokes T, Phillips SM. Resistance exercise training as a primary countermeasure to age-related chronic disease. Front Physiol. 2019;10:645.

61. Strasser B, Pesta D. Resistance training for diabetes prevention and therapy: experimental findings and molecular mechanisms. Biomed Res Int. 2013;2013:805217.

62. Wedell-Neergaard AS, Eriksen L, Gronbaek M, et al. Low fitness is associated with abdominal adiposity and low-grade inflammation independent of BMI. PLoS ONE. 2018;13(1):e0190645.

63. Pedersen BK, Febbraio MA. Muscles, exercise and obesity: skeletal muscle as a secretory organ. Nat Rev Endocrinol. 2012;8(8):457–65.

64. Evans PL, McMillin SL. Regulation of skeletal muscle glucose transport and glucose metabolism by exercise training. Nutrients. 2019;11(10):2432.

65. Galvao DA, Nosaka K, Taaffe DR, et al. Resistance training and reduction of treatment side effects in prostate cancer patients. Med Sci Sports Exerc. 2006;38(12):2045–52.

66. Newton RU, Galvao DA, Spry N, et al. Exercise mode specificity for preserving spine and hip bone mineral density in prostate cancer patients. Med Sci Sports Exerc. 2019;51(4):607–14.

67. Hojman P. Exercise protects from cancer through regulation of immune function and inflammation. Biochem Soc Trans. 2017;45(4):905–11.

68. Campbell JP, Turner JE. Debunking the myth of exercise-induced immune suppression: redefining the impact of exercise on immunological health across the lifespan. Front Immunol. 2018;9:648.

69. Gordon BR, McDowell CP, Lyons M, et al. The effects of resistance exercise training on anxiety: a meta-analysis and meta-regression analysis of randomized controlled trials. Sports Med. 2017;47(12):2521–32.

70. Gordon BR, McDowell CP, Hallgren M, et al. Association of efficacy of resistance exercise training with depressive symptoms: meta-analysis and meta-regression analysis of randomized clinical trials. JAMA Psychiatry. 2018;75(6):566–76.

71. Singh NA, Stavrinos TM, Scarbek Y, et al. A randomized controlled trial of high versus low intensity weight training versus general practitioner care for clinical depression in older adults. J Gerontol A Biol Sci Med Sci. 2005;60(6):768–76.

72. O'Connor PJ, Herring MP, Caravalho A. Mental health benefits of strength training in adults. Am J Lifestyle Med. 2010;4(5):377–96.

73. Chrousos GP. Stress and disorders of the stress system. Nat Rev Endocrinol. 2009;5(7):374–81.

74. Strickland JC, Smith MA. The anxiolytic effects of resistance exercise. Front Psychol. 2014;5:753.

75. Cassilhas RC, Antunes HKM, Tufik S, et al. Mood, anxiety, and serum IGF-1 in elderly men given 24 weeks of high resistance exercise. Percept Mot Skills. 2010;110(1):265–76.

76. Kovacevic A, Mavros Y, Heisz JJ, et al. The effect of resistance exercise on sleep: a systematic review of randomized controlled trials. Sleep Med Rev. 2018;39:52–68.

77. Booth J, Moseley GL, Schiltenwolf M, et al. Exercise for chronic musculoskeletal pain: a biopsychosocial approach. Musculoskelet Care. 2017;15(4):413–21.

78. Babatunde OO, Jordan JL, Van der Windt DA, et al. Effective treatment options for musculoskeletal pain in primary care: a systematic overview of current evidence. PLoS ONE. 2017;12(6):e0178621.

79. Kristensen J, Franklyn-Miller A. Resistance training in musculoskeletal rehabilitation: a systematic review. Br J Sports Med. 2012;46(10):719.

80. Deleo JA, Tanga FY, Tawfik VL. Neuroimmune activation and neuroinflammation in chronic pain and opioid tolerance/hyperalgesia. Neuroscientist. 2004;10(1):40–52.

81. Sluka KA, Frey-Law L, Hoeger Bement M. Exercise-induced pain and analgesia? Underlying mechanisms and clinical translation. Pain. 2018;159(Suppl 1):S91–7.

82. Rice D, Nijs J, Kosek E, et al. Exercise induced hypoalgesia in pain-free and chronic pain populations: state of the art and future directions. J Pain. 2019;20(11):1249–66.

83. Naugle KM, Fillingim RB, Riley Iii JL. A meta-analytic review of the hypoalgesic effects of exercise. J Pain. 2012;13(12):1139–50.

84. Lannersten L, Kosek E. Dysfunction of endogenous pain inhibition during exercise with painful muscles in patients with shoulder myalgia and fibromyalgia. Pain. 2010;151(1):77–86.

85. Smith BE, Hendrick P, Bateman M, et al. Musculoskeletal pain and exercise—challenging existing paradigms and introducing new. Br J Sports Med. 2019;53(14):907.

86. Hollings M, Mavros Y, Freeston J, et al. The effect of progressive resistance training on aerobic fitness and strength in adults with coronary heart disease: a systematic review and meta-analysis of randomised controlled trials. Eur J Prev Cardiol. 2017;24(12):1242–59.

87. Frodermann V, Rohde D, Courties G, et al. Exercise reduces inflammatory cell production and cardiovascular inflammation via instruction of hematopoietic progenitor cells. Nat Med. 2019;25(11):1761–71.

88. Cornelissen VA, Fagard RH, Coeckelberghs E, et al. Impact of resistance training on blood pressure and other cardiovascular risk factors: a meta-analysis of randomized, controlled trials. Hypertension. 2011;58(5):950–8.

89. Guilak F, Fermor B, Keefe FJ, et al. The role of biomechanics and inflammation in cartilage injury and repair. Clin Orthop Relat Res. 2004;423:17–26.

90. Bohndorf K. Imaging of acute injuries of the articular surfaces (chondral, osteochondral and subchondral fractures). Skelet Radiol. 1999;28(10):545–60.

91. Eckstein F, Hudelmaier M, Putz R. The effects of exercise on human articular cartilage. J Anat. 2006;208(4):491–512.

92. Vanwanseele B, Eckstein F, Knecht H, et al. Longitudinal analysis of cartilage atrophy in the knees of patients with spinal cord injury. Arthritis Rheum. 2003;48(12):3377–81.

93. Hudelmaier M, Glaser C, Englmeier KH, et al. Correlation of knee-joint cartilage morphology with muscle cross-sectional areas vs. anthropometric variables. Anat Rec A Discov Mol Cell Evol Biol. 2003;270(2):175–84.

94. Ericsson YB, Roos EM, Owman H, et al. Association between thigh muscle strength four years after partial meniscectomy and radiographic features of osteoarthritis 11 years later. BMC Musculoskelet Disord. 2019;20(1):512.

95. Cicuttini FM, Wluka AE. Is OA a mechanical or systemic disease? Nat Rev Rheumatol. 2014;10(9):515–6.

96. Skou ST, Pedersen BK, Abbott JH, et al. Physical activity and exercise therapy benefit more than just symptoms and impairments in people with hip and knee osteoarthritis. J Orthop Sports Phys Ther. 2018;48(6):439–47.

97. Caneiro JP, Sullivan PB, Roos EM, et al. Three steps to changing the narrative about knee osteoarthritis care: a call to action. Br J Sports Med. 2019. https://doi.org/10.1136/bjsports-2019-101328.

98. Hunter DJ, Bierma-Zeinstra S. Osteoarthritis. Lancet. 2019;393(10182):1745–59.

99. Oiestad BE, Juhl CB, Eitzen I, et al. Knee extensor muscle weakness is a risk factor for development of knee osteoarthritis. A systematic review and meta-analysis. Osteoarthr Cartil. 2015;23(2):171–7.

100. Segal NA, Torner JC, Felson D, et al. Effect of thigh strength on incident radiographic and symptomatic knee osteoarthritis in a longitudinal cohort. Arthritis Rheum. 2009;61(9):1210–7.

101. Thorstensson CA, Petersson IF, Jacobsson LT, et al. Reduced functional performance in the lower extremity predicted radiographic knee osteoarthritis five years later. Ann Rheum Dis. 2004;63(4):402–7.

102. Pietrosimone B, Thomas AC, Saliba SA, et al. Association between quadriceps strength and self-reported physical activity in people with knee osteoarthritis. Int J Sports Phys Ther. 2014;9(3):320–8.

103. Lin I, Wiles L, Waller R, et al. What does best practice care for musculoskeletal pain look like? Eleven consistent recommendations from high-quality clinical practice guidelines: systematic review. Br J Sports Med. 2019;54(2):79–86.

104. Juhl C, Christensen R, Roos EM, et al. Impact of exercise type and dose on pain and disability in knee osteoarthritis: a systematic review and meta-regression analysis of randomized controlled trials. Arthritis Rheumatol. 2014;66(3):622–36.

105. Bricca A, Juhl CB, Steultjens M, et al. Impact of exercise on articular cartilage in people at risk of, or with established, knee osteoarthritis: a systematic review of randomised controlled trials. Br J Sports Med. 2019;53(15):940–7.

106. Turner MN, Hernandez DO, Cade W, et al. The role of resistance training dosing on pain and physical function in individuals with knee osteoarthritis: a systematic review. Sports Health. 2019. https://doi.org/10.1177/1941738119887183.

107. Bricca A, Juhl CB, Grodzinsky AJ, et al. Impact of a daily exercise dose on knee joint cartilage—a systematic review and meta-analysis of randomized controlled trials in healthy animals. Osteoarthr Cartil. 2017;25(8):1223–37.

108. Amin S, Baker K, Niu J, et al. Quadriceps strength and the risk of cartilage loss and symptom progression in knee osteoarthritis. Arthritis Rheum. 2009;60(1):189–98.

109. de Paula FJA, Rosen CJ. Bone remodeling and energy metabolism: new perspectives. Bone Res. 2013;1(1):72–84.

110. Rubin J, Rubin C, Jacobs CR. Molecular pathways mediating mechanical signaling in bone. Gene. 2006;15(367):1–16.

111. Uda Y, Azab E, Sun N, et al. Osteocyte mechanobiology. Curr Osteoporos Rep. 2017;15(4):318–25.

112. Burr DB, Milgrom C, Fyhrie D, et al. In vivo measurement of human tibial strains during vigorous activity. Bone. 1996;18(5):405–10.

113. You L, Cowin SC, Schaffler MB, et al. A model for strain amplification in the actin cytoskeleton of osteocytes due to fluid drag on pericellular matrix. J Biomech. 2001;34(11):1375–86.

114. Weinbaum S, Duan Y, Thi MM, et al. An integrative review of mechanotransduction in endothelial, epithelial (renal) and dendritic cells (osteocytes). Cell Mol Bioeng. 2011;4(4):510–37.

115. Frost HM. Wolff's law and bone's structural adaptations to mechanical usage: an overview for clinicians. Angle Orthod. 1994;64(3):175–88.

116. O'Connor JA, Lanyon LE, MacFie H. The influence of strain rate on adaptive bone remodelling. J Biomech. 1982;15(10):767–81.

117. Rubin CT, McLeod KJ. Promotion of bony ingrowth by frequency-specific, low-amplitude mechanical strain. Clin Orthop Relat Res. 1994;298:165–74.

118. Turner CH. Three rules for bone adaptation to mechanical stimuli. Bone. 1998;23(5):399–407.

119. Martyn-St James M, Carroll S. Meta-analysis of walking for preservation of bone mineral density in postmenopausal women. Bone. 2008;43(3):521–31.

120. Watson SL, Weeks BK, Weis LJ, et al. High-intensity resistance and impact training improves bone mineral density and physical function in postmenopausal women with osteopenia and osteoporosis: the LIFTMOR randomized controlled trial. J Bone Miner Res. 2018;33(2):211–20.

121. Watson SL, Weeks BK, Weis LJ, et al. High-intensity exercise did not cause vertebral fractures and improves thoracic kyphosis in postmenopausal women with low to very low bone mass: the LIFTMOR trial. Osteoporos Int. 2019;30(5):957–64.

122. Zhao R, Zhao M, Xu Z. The effects of differing resistance training modes on the preservation of bone mineral density in postmenopausal women: a meta-analysis. Osteoporos Int. 2015;26(5):1605–18.

123. Gomez-Cabello A, Ara I, Gonzalez-Aguero A, et al. Effects of training on bone mass in older adults: a systematic review. Sports Med. 2012;42(4):301–25.

124. Westcott WL. Resistance training is medicine: effects of strength training on health. Curr Sports Med Rep. 2012;11(4):209–16.

125. Cauley JA, Giangregorio L. Physical activity and skeletal health in adults. Lancet Diabetes Endocrinol. 2019;8(2):150–62.

126. Fragala MS, Cadore EL, Dorgo S, et al. Resistance training for older adults: position statement from the national strength and conditioning association. J Strength Cond Res. 2019;33(8):2019–52.

127. Beck BR, Daly RM, Singh MA, et al. Exercise and Sports Science Australia (ESSA) position statement on exercise prescription for the prevention and management of osteoporosis. J Sci Med Sport. 2017;20(5):438–45.

128. Maddalozzo GF, Snow CM. High intensity resistance training: effects on bone in older men and women. Calcif Tissue Int. 2000;66(6):399–404.

129. Ryan AS, Ivey FM, Hurlbut DE, et al. Regional bone mineral density after resistive training in young and older men and women. Scand J Med Sci Sports. 2004;14(1):16–23.

130. Hansen M, Kjaer M. Influence of sex and estrogen on musculotendinous protein turnover at rest and after exercise. Exerc Sport Sci Rev. 2014;42(4):183–92.

131. MacKelvie KJ, Khan KM, McKay HA. Is there a critical period for bone response to weight-bearing exercise in children and adolescents? a systematic review. Br J Sports Med. 2002;36(4):250–7 (discussion 7).

132. Tenforde AS, Fredericson M. Influence of sports participation on bone health in the young athlete: a review of the literature. PM R. 2011;3(9):861–7.

133. Warden SJ, Mantila Roosa SM, Kersh ME, et al. Physical activity when young provides lifelong benefits to cortical bone size and strength in men. Proc Natl Acad Sci USA. 2014;111(14):5337–42.

134. Hernandez CJ, Beaupre GS, Carter DR. A theoretical analysis of the relative influences of peak BMD, age-related bone loss and menopause on the development of osteoporosis. Osteoporos Int. 2003;14(10):843–7.

135. Courteix D, Lespessailles E, Peres SL, et al. Effect of physical training on bone mineral density in prepubertal girls: a comparative study between impact-loading and non-impact-loading sports. Osteoporos Int. 1998;8(2):152–8.

136. Kahanov L, Eberman LE, Games KE, et al. Diagnosis, treatment, and rehabilitation of stress fractures in the lower extremity in runners. Open Access J Sports Med. 2015;6:87–95.

137. Schnackenburg KE, Macdonald HM, Ferber R, et al. Bone quality and muscle strength in female athletes with lower limb stress fractures. Med Sci Sports Exerc. 2011;43(11):2110–9.

138. Clark EM, Tobias JH, Murray L, et al. Children with low muscle strength are at an increased risk of fracture with exposure to exercise. J Musculoskelet Neuronal Interact. 2011;11(2):196–202.

139. Popp KL, Hughes JM, Smock AJ, et al. Bone geometry, strength, and muscle size in runners with a history of stress fracture. Med Sci Sports Exerc. 2009;41(12):2145–50.

140. Hoffman JR, Chapnik L, Shamis A, et al. The effect of leg strength on the incidence of lower extremity overuse injuries during military training. Mil Med. 1999;164(2):153–6.

141. Bohm S, Mersmann F, Arampatzis A. Human tendon adaptation in response to mechanical loading: a systematic review and meta-analysis of exercise intervention studies on healthy adults. Sports Med Open. 2015;1(1):7.

142. Turner AN, Jeffreys I. The stretch-shortening cycle: proposed mechanisms and methods for enhancement. Strength Cond J. 2010;32(4):87–99.

143. Mersmann F, Bohm S, Arampatzis A. Imbalances in the development of muscle and tendon as risk factor for tendinopathies in youth athletes: a review of current evidence and concepts of prevention. Front Physiol. 2017;8:987.

144. Scott A, Backman LJ, Speed C. Tendinopathy: update on pathophysiology. J Orthop Sports Phys Ther. 2015;45(11):833–41.

145. Kjaer M, Langberg H, Heinemeier K, et al. From mechanical loading to collagen synthesis, structural changes and function in human tendon. Scand J Med Sci Sports. 2009;19(4):500–10.

146. Docking SI, Cook J. How do tendons adapt? Going beyond tissue responses to understand positive adaptation and pathology development: a narrative review. J Musculoskelet Neuronal Interact. 2019;19(3):300–10.

147. Ryan M, Bisset L, Newsham-West R. Should we care about tendon structure? The disconnect between structure and symptoms in tendinopathy. J Orthop Sports Phys Ther. 2015;45(11):823–5.

148. Drew BT, Smith TO, Littlewood C, et al. Do structural changes (eg, collagen/matrix) explain the response to therapeutic exercises in tendinopathy: a systematic review. Br J Sports Med. 2014;48(12):966–72.

149. Cook JL, Rio E, Purdam CR, et al. Revisiting the continuum model of tendon pathology: what is its merit in clinical practice and research? Br J Sports Med. 2016;50(19):1187.

150. Murphy M, Travers M, Gibson W, et al. Rate of Improvement of pain and function in mid-portion achilles tendinopathy with loading protocols: a systematic review and longitudinal meta-analysis. Sports Med. 2018;48(8):1875–91.

151. Scott A, Docking S, Vicenzino B, et al. Sports and exercise-related tendinopathies: a review of selected topical issues by participants of the second International Scientific Tendinopathy Symposium (ISTS) Vancouver 2012. Br J Sports Med. 2013;47(9):536.

152. Geremia JM, Baroni BM, Bobbert MF, et al. Effects of high loading by eccentric triceps surae training on Achilles tendon properties in humans. Eur J Appl Physiol. 2018;118(8):1725–36.

153. Reeves ND, Maganaris CN, Narici MV. Effect of strength training on human patella tendon mechanical properties of older individuals. J Physiol Lond. 2003;548(Pt 3):971–81.

154. Malliaras P, Barton CJ, Reeves ND, et al. Achilles and patellar tendinopathy loading programmes: a systematic review comparing clinical outcomes and identifying potential mechanisms for effectiveness. Sports Med. 2013;43(4):267–86.

155. Beyer R, Kongsgaard M, Hougs Kjaer B, et al. Heavy slow resistance versus eccentric training as treatment for achilles tendinopathy: a randomized controlled trial. Am J Sports Med. 2015;43(7):1704–11.

156. Couppé C, Svensson RB, Silbernagel KG, et al. Eccentric or concentric exercises for the treatment of tendinopathies? J Orthop Sports Phys Ther. 2015;45(11):853–63.

157. O'Neill S, Watson PJ, Barry S. Why are eccentric exercises effective for achilles tendinopathy? Int J Sports Phys Ther. 2015;10(4):552–62.

158. Suchomel TJ, Wagle JP, Douglas J, et al. Implementing eccentric resistance training—part 1: a brief review of existing methods. J Funct Morphol Kinesiol. 2019;4(2):38.

159. Suchomel TJ, Wagle JP, Douglas J, et al. Implementing eccentric resistance training—part 2: practical recommendations. J Funct Morphol Kinesiol. 2019;4(3):55.

160. O'Neill S, Radia J, Bird K, et al. Acute sensory and motor response to 45-s heavy isometric holds for the plantar flexors in patients with Achilles tendinopathy. Knee Surg Sports Traumatol Arthrosc. 2019;27(9):2765–73.

161. Holden S, Lyng K, Graven-Nielsen T, et al. Isometric exercise and pain in patellar tendinopathy: a randomized crossover trial. Med Sci Sports Exerc. 2019;23(3):208–14.

162. Gravare Silbernagel K, Vicenzino BT. Isometric exercise for acute pain relief: is it relevant in tendinopathy management? Br J Sports Med. 2019;53(21):1330–1.

163. Malliaras P. Understanding mechanisms to improve exercise interventions in tendinopathy. Phys Ther Sport. 2017;27:50–1.

164. Head J, Mallows A, Debenham J, et al. The efficacy of loading programmes for improving patient-reported outcomes in chronic midportion Achilles tendinopathy: a systematic review. Musculoskelet Care. 2019;17(4):283–99.

165. Mahieu NN, Witvrouw E, Stevens V, et al. Intrinsic risk factors for the development of achilles tendon overuse injury: a prospective study. Am J Sports Med. 2006;34(2):226–35.

166. O'Neill S, Watson PJ, Barry S. A Delphi study of risk factors for achilles tendinopathy—opinions of world tendon experts. Int J Sports Phys Ther. 2016;11(5):684–97.

167. O'Neill S, Barry S, Watson P. Plantarflexor strength and endurance deficits associated with mid-portion Achilles tendinopathy: the role of soleus. Phys Ther Sport. 2019;37:69–76.

168. McAuliffe S, Tabuena A, McCreesh K, et al. Altered strength profile in achilles tendinopathy: a systematic review and meta-analysis. J Athl Train. 2019;54(8):889–900.

169. Peters JA, Zwerver J, Diercks RL, et al. Preventive interventions for tendinopathy: a systematic review. Med Sci Sports Exerc. 2016;19(3):205–11.

170. Fredberg U, Bolvig L, Andersen NT. Prophylactic training in asymptomatic soccer players with ultrasonographic abnormalities in Achilles and patellar tendons: the Danish Super League Study. Am J Sports Med. 2008;36(3):451–60.

171. Heinemeier KM, Schjerling P, Heinemeier J, et al. Lack of tissue renewal in human adult Achilles tendon is revealed by nuclear bomb (14)C. FASEB J. 2013;27(5):2074–9.

172. Kubo K, Teshima T, Hirose N, et al. Growth changes in morphological and mechanical properties of human patellar tendon in vivo. J Appl Biomech. 2014;30(3):415–22.

173. Waugh CM, Blazevich AJ, Fath F, et al. Age-related changes in mechanical properties of the Achilles tendon. J Anat. 2012;220(2):144–55.

174. Behringer M, Vom Heede A, Yue Z, et al. Effects of resistance training in children and adolescents: a meta-analysis. Pediatrics. 2010;126(5):e1199–210.

175. Faigenbaum AD, Myer GD. Resistance training among young athletes: safety, efficacy and injury prevention effects. Br J Sports Med. 2010;44(1):56–63.

176. Faigenbaum AD, Kraemer WJ, Blimkie CJ, et al. Youth resistance training: updated position statement paper from the national strength and conditioning association. J Strength Cond Res. 2009;23(5 Suppl):S60–79.

177. Lloyd RS, Oliver JL. The youth physical development model: a new approach to long-term athletic development. Strength Cond J. 2012;34(3):61–72.

178. Jarvinen TA, Jarvinen TL, Kaariainen M, et al. Muscle injuries: biology and treatment. Am J Sports Med. 2005;33(5):745–64.

179. Santilli V, Bernetti A, Mangone M, et al. Clinical definition of sarcopenia. Clin Cases Miner Bone Metab. 2014;11(3):177–80.

180. Clark BC, Manini TM. What is dynapenia? Nutrition. 2012;28(5):495–503.

181. Aagaard P, Suetta C, Caserotti P, et al. Role of the nervous system in sarcopenia and muscle atrophy with aging: strength training as a countermeasure. Scand J Med Sci Sports. 2010;20(1):49–64.

182. Tinetti ME. Preventing falls in elderly persons. N Engl J Med. 2003;348(1):42–9.

183. Pijnappels M, van der Burg PJ, Reeves ND, et al. Identification of elderly fallers by muscle strength measures. Eur J Appl Physiol. 2008;102(5):585–92.

184. Boyle PA, Buchman AS, Wilson RS, et al. Association of muscle strength with the risk of Alzheimer disease and the rate of cognitive decline in community-dwelling older persons. Arch Neurol. 2009;66(11):1339–44.

185. Ruiz JR, Sui X, Lobelo F, et al. Association between muscular strength and mortality in men: prospective cohort study. BMJ. 2008;337:a439.

186. Goodpaster BH, Park SW, Harris TB, et al. The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. J Gerontol A Biol Sci Med Sci. 2006;61(10):1059–64.

187. Garcia-Hermoso A, Cavero-Redondo I, Ramirez-Velez R, et al. Muscular strength as a predictor of all-cause mortality in an apparently healthy population: a systematic review and meta-analysis of data from approximately 2 million men and women. Arch Phys Med Rehabil. 2018;99(10):2100–2113.e5.

188. Li R, Xia J, Zhang XI, et al. Associations of muscle mass and strength with all-cause mortality among US older adults. Med Sci Sports Exerc. 2018;50(3):458–67.

189. Avers D, Brown M. White paper: strength training for the older adult. J Geriatr Phys Ther. 2009;32(4):148–52, 58.

190. Guizelini PC, de Aguiar RA, Denadai BS, et al. Effect of resistance training on muscle strength and rate of force development in healthy older adults: a systematic review and meta-analysis. Exp Gerontol. 2018;102:51–8.

191. Moore SA, Hrisos N, Errington L, et al. Exercise as a treatment for sarcopenia: an umbrella review of systematic review evidence. Physiotherapy. 2020;107:189–201.

192. van der Horst N, Smits DW, Petersen J, et al. The preventive effect of the nordic hamstring exercise on hamstring injuries in amateur soccer players: a randomized controlled trial. Am J Sports Med. 2015;43(6):1316–23.

193. Mueller-Wohlfahrt HW, Haensel L, Mithoefer K, et al. Terminology and classification of muscle injuries in sport: the Munich consensus statement. Br J Sports Med. 2013;47(6):342.

194. Arnason A, Andersen TE, Holme I, et al. Prevention of hamstring strains in elite soccer: an intervention study. Scand J Med Sci Sports. 2008;18(1):40–8.

195. Petersen J, Thorborg K, Nielsen MB, et al. Preventive effect of eccentric training on acute hamstring injuries in Men's soccer: a cluster-randomized controlled trial. Am J Sports Med. 2011;39(11):2296–303.

196. Cuthbert M, Ripley N, McMahon JJ, et al. The effect of nordic hamstring exercise intervention volume on eccentric strength and muscle architecture adaptations: a systematic review and meta-analyses. Sports Med. 2019;50(1):83–99.

197. Bourne MN, Timmins RG, Opar DA, et al. An evidence-based framework for strengthening exercises to prevent hamstring injury. Sports Med. 2018;48(2)251–67.

198. van Dyk N, Behan FP, Whiteley R. Including the Nordic hamstring exercise in injury prevention programmes halves the rate of hamstring injuries: a systematic review and meta-analysis of 8459 athletes. Br J Sports Med. 2019;53(21):1362.

199. Zouita S, Zouita AB, Kebsi W, et al. Strength training reduces injury rate in elite young soccer players during one season. J Strength Cond Res. 2016;30(5):1295–307.

200. Haroy J, Clarsen B, Wiger EG, et al. The Adductor Strengthening Programme prevents groin problems among male football players: a cluster-randomised controlled trial. Br J Sports Med. 2019;53(3):150–7.

201. Halson SL. Sleep monitoring in athletes: motivation, methods, miscalculations and why it matters. Sports Med. 2019;49(10):1487–97.

202. Lovibond PF, Lovibond SH. The structure of negative emotional states: comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. Behav Res Ther. 1995;33(3):335–43.

203. Smarr KL, Keefer AL. Measures of depression and depressive symptoms: Beck Depression Inventory-II (BDI-II), Center for Epidemiologic Studies Depression Scale (CES-D), Geriatric Depression Scale (GDS), Hospital Anxiety and Depression Scale (HADS), and Patient Health Questionnaire-9 (PHQ-9). Arthritis Care Res (Hoboken). 2011;63(Suppl 11):S454–66.

204. Linton SJ. A transdiagnostic approach to pain and emotion. J Appl Biobehav Res. 2013;18(2):82–103.

205. Foster NE, Hill JC, O'Sullivan P, et al. Stratified models of care. Best Pract Res Clin Rheumatol. 2013;27(5):649–61.

206. Appleby BB, Cormack SJ, Newton RU. Specificity and transfer of lower-body strength: influence of bilateral or unilateral lower-body resistance training. J Strength Cond Res. 2019;33(2):318–26.

207. Lasevicius T, Schoenfeld BJ, Silva-Batista C, et al. Muscle failure promotes greater muscle hypertrophy in low-load but not in high-load resistance training. J Strength Cond Res. 2019. https://doi.org/10.1519/JSC.0000000000003454.

208. Morton RW, Colenso-Semple L, Phillips SM. Training for strength and hypertrophy: an evidence-based approach. Curr Opin Physiol. 2019;10:90–5.

209. Martorelli S, Cadore EL, Izquierdo M, et al. Strength training with repetitions to failure does not provide additional strength and muscle hypertrophy gains in young women. Eur J Transl Myol. 2017;27(2):6339.

210. Kubo K, Ikebukuro T, Yata H. Effects of 4, 8, and 12 repetition maximum resistance training protocols on muscle volume and strength. J Strength Cond Res. 2020. https://doi.org/10.1519/JSC.0000000000003575.

211. Schoenfeld BJ, Grgic J. Does training to failure maximize muscle hypertrophy? Strength Cond J. 2019;41(5):108–13.

212. Davies T, Orr R, Halaki M, et al. Effect of training leading to repetition failure on muscular strength: a systematic review and meta-analysis. Sports Med. 2016;46(4):487–502.

213. Stone MH, Sands WA, Carlock J, et al. The importance of isometric maximum strength and peak rate-of-force development in sprint cycling. J Strength Cond Res. 2004;18(4):878–84.

214. Androulakis-Korakakis P, Fisher JP, Steele J. The minimum effective training dose required to increase 1RM strength in resistance-trained men: a systematic review and meta-analysis. Sports Med. 2020;50(4):751–65.

215. Rhea MR, Alvar BA, Burkett LN, et al. A meta-analysis to determine the dose response for strength development. Med Sci Sports Exerc. 2003;35(3):456–64.

216. Zourdos MC, Klemp A, Dolan C, et al. Novel resistance training-specific rating of perceived exertion scale measuring repetitions in reserve. J Strength Cond Res. 2016;30(1):267–75.

217. Thompson SW, Rogerson D. The effectiveness of two methods of prescribing load on maximal strength development: a systematic review. Sports Med. 2020;50(5):919–38.

218. Graham T, Cleather DJ. Autoregulation by "repetitions in reserve" leads to greater improvements in strength over a 12-week training program than fixed loading. J Strength Cond Res. 2019. https://doi.org/10.1519/JSC.0000000000003164.

219. Mann JB, Thyfault JP, Ivey PA, et al. The effect of autoregulatory progressive resistance exercise vs. linear periodization on strength improvement in college athletes. J Strength Cond Res. 2010;24(7):1718–23.

220. Gonzalez-Badillo JJ, Sanchez-Medina L. Movement velocity as a measure of loading intensity in resistance training. Int J Sports Med. 2010;31(5):347–52.

221. Picerno P, Iannetta D, Comotto S, et al. 1RM prediction: a novel methodology based on the force-velocity and load-velocity relationships. Eur J Appl Physiol. 2016;116(10):2035–43.

222. Dorrell HF, Smith MF, Gee TI. Comparison of velocity-based and traditional percentage-based loading methods on maximal strength and power adaptations. J Strength Cond Res. 2020;34(1):46–53.

223. Pareja-Blanco F, Alcazar J, Sanchez-Valdepenas J, et al. Velocity loss as a critical variable determining the adaptations to strength training. Med Sci Sports Exerc. 2020. https://doi.org/10.1249/MSS.0000000000002295

224. Weakley J, McLaren S, Ramirez-Lopez C. Application of velocity loss thresholds during free-weight resistance training: responses and reproducibility of perceptual, metabolic, and neuromuscular outcomes. J Sports Sci. 2019;22:1–9.

225. Grgic J, Schoenfeld BJ, Latella C. Resistance training frequency and skeletal muscle hypertrophy: a review of available evidence. J Sci Med Sport. 2019;22(3):361–70.

226. Androulakis-Korakakis P, Fisher JP, Steele J. The minimum effective training dose required to increase 1RM strength in resistance-trained men: a systematic review and meta-analysis. Sports Med. 2020;50(4):751–65.

227. Sale DG. Neural adaptation to resistance training. Med Sci Sports Exerc. 1988;20(5 Suppl):S135–45.

228. Damas F, Phillips SM, Lixandrao ME, et al. Early resistance training-induced increases in muscle cross-sectional area are concomitant with edema-induced muscle swelling. Eur J Appl Physiol. 2016;116(1):49–56.

229. Maestroni L, Read P, Bishop C, et al. Strength and power training in rehabilitation: underpinning principles and practical strategies to return athletes to high performance. Sports Med. 2020;50(2):239–52.

230. Physical activity guidelines Advisory Committee. 2018 Physical activity guidelines Advisory Committee scientific report. Washington, DC: U.S. Department of Health and Human Services; 2018.

231. Bull FC, Groups EW. Physical activity guidelines in the U.K.: review and recommendations. Loughborough: School of Sport, Exercise and Health Sciences, Loughborough University; 2010.

232. Department of Health. Australia's physical activity and sedentary behaviour guidelines. Canberra: Department of Health; 2014.

233. World Health Organization. Global recommendations on physical activity for health. Geneva: World Health Organization; 2010.

234. American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. Philadelphia: Wolters Kluwer; 2018.