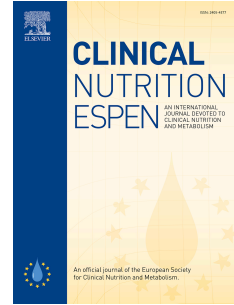


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Basics in clinical sports nutrition: Physical activity, muscle and clinical nutrition

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PII: S2405-4577(26)00016-1

DOI: <https://doi.org/10.1016/j.clnesp.2026.102921>

Reference: CLNESP 102921

To appear in: *Clinical Nutrition ESPEN*

Received Date: 20 December 2025

Accepted Date: 5 January 2026

Please cite this article as: Kozjek NR, Tonin G, Prado C, Maughan RJ, Basics in clinical sports nutrition: Physical activity, muscle and clinical nutrition, *Clinical Nutrition ESPEN*, <https://doi.org/10.1016/j.clnesp.2026.102921>.

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## Basics in clinical sports nutrition: Physical activity, muscle and clinical nutrition

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### Summary

Physical activity and nutrition are inextricably linked in maintaining muscle and overall health, enhancing metabolic resilience, and preventing age-related muscle loss. Exercise acts as a powerful physiological stressor, inducing acute metabolic, endocrine, and immunological responses, while long-term training elicits structural and functional adaptations at the muscular, cardiovascular, and systemic levels. Proper nutritional strategies, aligned with the type, intensity, and duration of physical activity, provide the necessary substrates to fuel exercise, optimize recovery, and support muscle protein synthesis and immune resilience. This integrative approach not only enhances athletic performance but also serves as a critical tool in the prevention and management of chronic diseases, including obesity, type 2 diabetes and cardiovascular disease. Emerging evidence further underscores the importance of skeletal muscle as a metabolic and endocrine organ, producing myokines which, with other exerkines, mediate the systemic health benefits of exercise. As a result, the evolving field of clinical sports nutrition bridges the gap between performance-oriented sports nutrition and evidence-based clinical care for physically active individuals across the health spectrum. By combining exercise physiology, clinical nutrition, and applied sports science, this multidisciplinary model provides a robust framework for advancing metabolic health, functional capacity, and performance in diverse populations.

**Key words:** exercise, exercise endocrinology, exercise physiology, muscle, clinical sports nutrition

### Learning Objectives

- Understand the diverse benefits of physical activity and muscle mass, including their roles in promoting metabolic health, preventing chronic diseases, and optimizing physical and athletic performance.
- Establish a clear and consistent framework for key terminology related to physical activity, exercise, and sports to enhance scientific understanding and practical application.
- Understand how acute exercise induces physiological stress, disrupting homeostasis, and explore how the body adapts to chronic exposure to exercise through recovery mechanisms and improved metabolic function.
- Comprehend how physical activity and structured training modify metabolic pathways and energy utilization at rest and during exercise while examining how targeted nutritional strategies provide essential substrates and other nutrients to support energy demands, recovery, and cognitive function.
- Understand the importance of muscle mass for metabolic health
- Recognize that nutritional requirements are dynamic and depend on the type, intensity, and duration of physical activity, and on individual physiology. This highlights the need for an integrative approach combining an understanding of exercise physiology, metabolism, and nutrition to develop evidence-based, personalized nutritional interventions.

### Key messages

- Exercise is a potent physiological stimulus that creates significant challenges to homeostasis. The metabolic challenges include increased energy demands, shifts in substrate utilization, oxidative stress, and hormonal fluctuations, all of which are key drivers of adaptation of organs, tissues and overall metabolic health.
- To meet the exercise challenge, multiple integrated and often redundant responses operate to diminish the homeostatic stress. The development of fatigue, whether of central or peripheral origin, is strongly influenced by nutrition status.
- Although the benefits of exercise in enhancing health and treating disease are well-acknowledged, the molecular mechanisms underlying exercise-associated benefits remain ill-defined and are actively being investigated.
- The metabolic response to exercise is dictated by energy demand and on the individual's capacity to meet that demand: it depends on the intensity and duration of physical activity and determines the ability to produce muscle power.
- Multiple organ systems, including the cardiometabolic system, nervous system, and immune system, produce exerkinines and are influenced by exerkinines, which probably contributes to the pleiotropic and variable response to exercise.
- 'Exerkinines' encompass various signalling moieties such as hormones, metabolites, proteins, and nucleic acids; they are released in response to acute and/or regular exercise and exert their effects through endocrine, paracrine, and/or autocrine pathways.
- Skeletal muscle is a vital organ that goes beyond strength and movement, playing a crucial role in metabolism, immunity, and overall health, making its preservation essential for longevity and disease prevention.
- Ensuring energy and nutrient availability at critical training points is important for optimal training, regeneration, and competitive performance, immune system protection, injury prevention, and overreaching and overtraining.
- Athletes should be aware of their nutrition goals and should seek professional help to develop an eating strategy that allows those goals to be met.

## 1. Introduction

Physical activity is fundamental for maintaining physical and mental health, preventing chronic diseases, and improving quality of life and longevity [1–4]. A recent comprehensive meta-analysis showed that leisure time physical activity consistently produces substantial dose-related health benefits in men and women: in contrast, higher levels of occupational physical activity are associated with higher all-cause mortality in men but seem to have no effect in women [5].

Exercise is well-established as a potent intervention for promoting cardiovascular health and reducing the risk of cardiovascular disease [6]. Regular running can reduce cardiovascular mortality by approximately 30%, emphasizing its effectiveness in lowering cardiovascular risk and promoting overall longevity [7]. Evidence also indicates that physical activity contributes to cancer prevention, improved metabolic regulation, and enhanced immune function [8–10].

In contrast, physical inactivity is a major public health concern, associated with increased mortality and significant economic costs [11,12]. Loss of muscle mass and muscle strength secondary to inactivity is also a major factor in the loss of the ability to live independently in the older adults [13]. The increasing burden of care for the elderly reflects the growing adoption of a sedentary lifestyle among most populations around the world [14].

Building on this extensive body of scientific research, the World Health Organization (WHO) and various governmental organizations have issued comprehensive global and national reports highlighting the pivotal importance of physical activity in promoting health and preventing disease [15–19]. The recent narrative from the UK government highlights a curvilinear dose-response relationship between physical activity and health outcomes. [19]. The UK report underscores the importance of encouraging all age groups to engage in a variety of physical activities, including strengthening exercises, while emphasizing the additional benefits of balance and flexibility exercises, particularly for older adults. It also highlights the health risks associated with inactivity and sedentary behaviour, citing evidence that prolonged sitting is linked to increased all-cause mortality, cardiovascular mortality, and cancer risk, as well as impacts on cancer survivorship [2,17].

Moreover, numerous reports and expert opinions suggest that exercise may effectively counter some of the adverse consequences of human biological aging [20]. Exercise and physical activity attenuate multiple adverse effects of chronological aging across various physiological systems. Evidence suggests that individuals who engage in lifelong exercise exhibit a more youthful endocrine profile compared to their sedentary counterparts. Furthermore, exercise interventions have been shown to modulate hormonal profiles, resulting in phenotypic characteristics that align more closely with younger biological age [20].

Additionally, meta-analytic findings by Martinez-Vizcaino et al. indicate that moderate to high levels of physical activity are associated with a 21% reduction in all-cause mortality risk and a 24% reduction in cardiovascular disease mortality among adults with obesity (18). These findings highlight the substantial protective effects of physical activity, even within high-risk populations, emphasizing its pivotal role as an evidence-based intervention for enhancing health outcomes and reducing mortality in individuals with obesity.

Therefore, physical activity provides significant health benefits across the entire population spectrum, playing a critical role in enhancing overall well-being and preventing or managing chronic diseases. These benefits are mediated by complex biological responses to physical activity, closely linked to metabolic processes supported by proper nutrition. Skeletal muscle mass serves as a central regulator in these mechanisms, governing energy metabolism, glucose homeostasis, and the secretion of bioactive molecules, such as myokines and other exerkines, which contribute to systemic health improvement [21,22].

Beyond promoting health, regular and planned physical activity also enhances physical performance, particularly in athletes [23]. By improving physiological resilience, metabolic efficiency, recovery, and endurance, exercise enables athletes to achieve peak performance while maintaining long-term health, though injuries associated with intensive training and competition may have some adverse effects in later life [24]. A comprehensive understanding of the terminology of physical activity, the biological principles of exercise physiology and endocrinology, and the central role of muscle mass is essential for developing integrated strategies to improve health and optimize physical performance [25–27]. These strategies, which combine exercise and nutrition, are vital for optimizing health outcomes and maximizing performance across all levels of physical activity [28–30]. Promoting physical activity should be a priority for all healthcare professionals [31].

## 2. Terminology and FITT principle

A clear understanding of the fundamental physical activity and exercise terminology is crucial for designing effective health and performance strategies. The **FITT principle**—Frequency, Intensity, Time, and Type— may serve as a platform for exercise prescription, enabling tailored programs that address individual goals, physiological needs, and health conditions. By systematically applying these components, practitioners can create evidence-based interventions to improve fitness, enhance performance, and promote long-term mental and physical well-being [25,28,32].

### 2.1 Terminology

#### 2.1.1 Physical activity, Exercise, Sports

**Physical activity** refers to any bodily movement produced by the contraction of skeletal muscles [25]. Physical activity refers to all movement, including that during leisure time, for transport to and from places, or as part of a person's work or domestic activities [16,33]. As highlighted above, and in contrast to the early studies of occupational physical activity that focused on coronary heart disease, leisure time activities seem to be key to the benefits of exercise. Physical activity plays a crucial role in maintaining overall health by reducing the risk of chronic diseases, such as cardiovascular disease, diabetes, and obesity, through its impact on energy balance and metabolic health (25,26). Both moderate- and vigorous-intensity physical activity improve health. Popular ways to be active include walking, cycling, sports, active recreation, and play. These activities can be done at any skill level and for everybody's enjoyment.

Physical activity benefits health and well-being; conversely, physical inactivity increases the risk for noncommunicable diseases (NCDs) and other poor health outcomes. Together, physical inactivity and sedentary behaviours are contributing to the rise in NCDs and placing a burden on healthcare systems [2,34,35].

**Exercise** is a subcategory of physical activity that is planned, structured, repetitive, and aimed at improving or maintaining physical fitness and health [25]. A characteristic of such activity is the production of force and power through the voluntary, coordinated activation of specific skeletal muscles [36]. However, voluntary exercise is more than just an orchestrated assembly of muscle contractions; a whole-body, voluntary exercise induces a range of additional physiological responses critical for enhancing muscle performance and developing movement skills.

The primary goal of exercise is to enhance physical health and functional capacity, though it can also be used for rehabilitation or to achieve personal fitness goals. It targets specific components of fitness, including cardiovascular endurance, muscular strength, flexibility, and balance. Regular exercise is associated with significant physiological adaptations, such as improved cardiorespiratory function, enhanced muscle strength, and reduced inflammation, collectively decreasing morbidity and mortality [37]. The nature of the adaptation is specific to the type of exercise, and the magnitude of the adaptations is proportional to the training load.

It is critical to differentiate the physiological responses elicited by voluntary, whole-body *in vivo* exercise from those generated by experimental models. For instance, electrical stimulation of isolated skeletal muscle induces activation and initiates intracellular signalling pathways associated with training adaptations [38]. In contrast, voluntary exercise engages complex systemic physiological responses essential for optimizing muscle performance, neuromuscular coordination, and overall homeostasis. Notably, observations derived from animal studies or isolated systems often diverge significantly from human *in vivo* data, underscoring the importance of caution when extrapolating findings across experimental models [39].

**Sport** is defined as a subset of physical activity that involves structured and competitive activities, usually governed by rules (3). The scope of sport is narrower than physical activity and exercise, focusing on structured competition, performance, skill development, achievement of goals, either individually or as a team, and often with entertainment or cultural significance alongside physical fitness. Engaging in sports combines the benefits of exercise with additional psychological and social advantages, such as teamwork, strategic thinking, and stress reduction. However, sport's high intensity and competitiveness may pose risks, such as overuse injuries and psychological stress, particularly at elite levels [40].

Table 1. delineates key characteristics of physical activity, exercise, and sports; Box 1. provides clinical translation strategies.

**Table 1.** Key differences between physical activity, exercises, and sports (Adapted from [25,35,41]).

<b>Aspect</b>	<b>Physical Activity</b>	<b>Exercise</b>	<b>Sports</b>
<b>Definition</b>	General movement by skeletal muscles, incidental or unplanned	Planned and goal-oriented	Organized activity with competition
<b>Purpose</b>	The purpose is usually unrelated to health; functional health maintenance may be unintended outcome	Fitness improvement, health, rehabilitation	Skill, competition, entertainment
<b>Goal</b>	General movement	Fitness or health improvement	Competition, performance, or fun
<b>Definition</b>	General movement by skeletal muscles	Structured, goal-oriented activity	Organized activity with competition
<b>Structure</b>	Unstructured	Structured and repetitive	Structured with formal rules
<b>Health Impact</b>	General improvements in energy balance	Targeted physiological adaptations	Combines fitness with social/mental benefits
<b>Competitive Element</b>	Not present	Optional	Central to sports
<b>Examples</b>	Walking, household chores, or commuting	Running, strength training, or yoga	Football, tennis, basketball

**Box 1.** Tips for clinical practice (Adapted from [11,27,32,42–44]).

#### **Practice Points**

- A clear understanding of *physical activity*, *exercise*, and *sports* is essential for healthcare providers to effectively counsel patients on strategies to enhance or modify their activity levels. Physical inactivity is a significant yet modifiable risk factor for chronic diseases, including

cardiovascular disease, type 2 diabetes, obesity, and certain cancers. Therefore, it is critical to focus on this issue in public health and clinical practice.

- The *health benefits of physical activity and exercise are well-established*; nearly everyone can gain from increasing their physical activity. Research indicates a linear relationship between physical activity and health status, meaning that greater increases in physical activity and fitness lead to further improvements in health.
- Addressing physical inactivity requires clear, evidence-based communication to help patients understand its health implications and practical applications. Interventions may include structured exercise programs or encouragement to increase daily physical movement tailored to individual needs. The evidence shows that clinically relevant health benefits can be achieved simply by becoming more physically active. Recognizing the differences between *physical activity*, *exercise*, and *sports* allows for a more personalized approach to tackling nutritional and health-related challenges.

In the following text, the terms *physical activity* and *exercise* are used interchangeably, primarily referring to regular physical activity or exercise, as defined in the cited literature, unless specified otherwise [3,4].

### 2.1.2 Types of physical activity

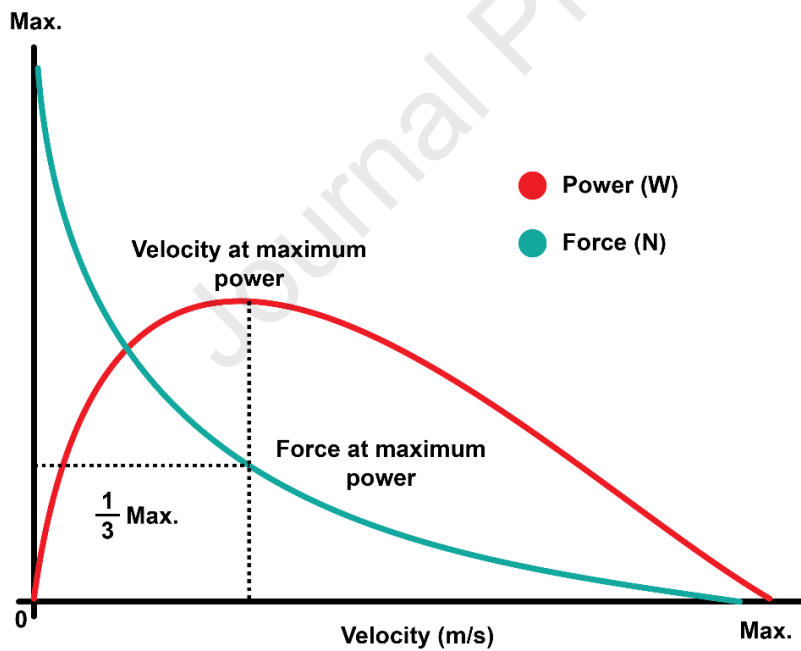
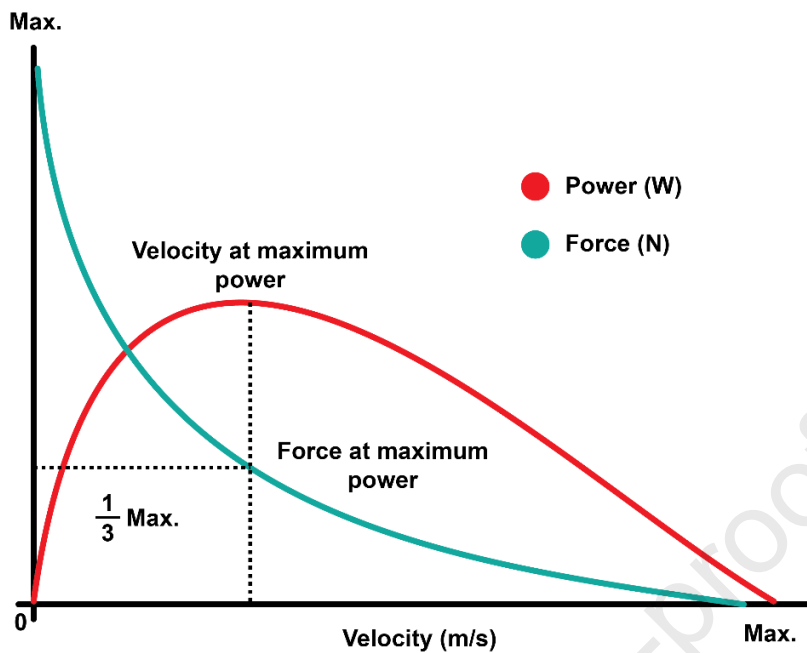
Physical activity is generally categorized into four primary types [2,34,45]:

1. **Aerobic or endurance physical activity:** Activities that increase heart rate and improve cardiovascular endurance, such as walking, jogging or swimming.
2. **Muscle-strengthening activity:** Exercises that enhance muscular strength and endurance, such as resistance training or push-ups.
3. **Bone-strengthening activity:** Movements that apply impact or force to bones, promoting bone density and health, like jumping or brisk walking.
4. **Flexibility and balance activity:** Exercises to improve range of motion and stability, such as yoga or tai chi.

**The endurance type of physical activity** represents a spectrum of rhythmic exercises like walking, running, cycling, swimming, skiing, and others. Within these sports, there are different disciplines. During sustained rhythmic exercise like cycling or running, the contraction times are short; there is increased muscle blood flow and minimal perturbations in blood pressure (BP).

Intense exercise events that require high power outputs of relatively shorter duration for success are considered **power sports** [46]. Typical power sports are middle-distance running, track cycling, Olympic rowing, canoeing/kayaking, and most swimming events. An isometric or static contraction of high force but a short duration may compress blood vessels within the contracting musculature and limit blood flow and oxygen delivery to those muscles while increasing BP [47].

**Power** is defined as the amount of work performed per unit of time [36]. It reflects the ability to exert high forces instantly or in an explosive burst of movements. The two power components are *strength* and *speed* (e.g., jumping or a sprint start). From an energetic and nutritional perspective, it is crucial to understand that power refers to the rate at which work is performed or the rate at which metabolic potential energy is transformed into work and/or heat. The force-velocity curve of skeletal muscle shows a characteristic shape: the highest force is generated in an isometric contraction, where speed of contraction is zero, and declines progressively as speed increases (Figure 1) [48]. Peak power is achieved at about one third of the maximum speed of shortening.



**Figure 1.** The force - velocity curve: Peak power is achieved at about one third of the maximum speed of shortening (adapted from [48]).

Typically, the efficiency of converting metabolic energy to work during exercise is slightly over 20%. This means that almost 80% of the energy from metabolic fuels is released as heat.

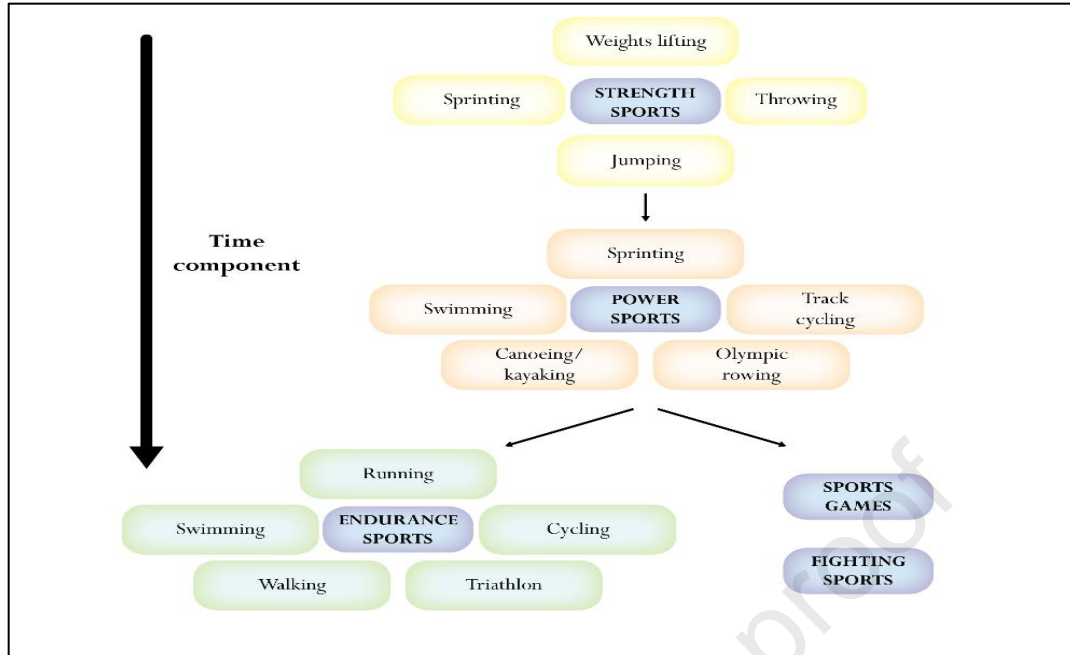
Optimal power output demands effective muscle coordination and mechanical efficiency of limb movement, meaning optimal sports performance requires the consideration of both mechanical (e.g., best gear ratio in cycling) and biomechanical (e.g., stride length in running and stroke length in swimming) factors. The best choice of gear ratio, step or stroke length, etc., is the one that allows muscles to contract with optimum combination of speed and force, which results in maximum mechanical muscle power. In complex motor tasks, the resulting power is influenced by the qualities of individual muscles and tendons, muscle coordination, the relationship between muscles and external forces, and the nervous system's activity [49].

**Strength** is defined as the ability to carry out work against a high resistance. Muscle strength represents the maximal force generated by muscle contracting against a load (e.g., holding or restraining an object or person) [36]. Muscles may contract maximally during isometric, concentric, eccentric, or stretch-shortening cycles. A typical example of muscle strength is the force and velocity of the weightlifter's motion on the barbell. Generating explosive muscle power and strength is essential for weightlifting, sprinting, throwing events, and bodybuilding [50].

The assessment and quantification of these physical abilities are described by the use of the International System of Measurements (SI) for force (Newton), energy, work, heat (Joules), torque (Newton-meters), and power (Watts).

Training methods to increase maximal muscle force (strength) and power are developed by employing resistance exercise programs. These programs use high opposing force (routinely termed resistance); the training includes lifting weights or increasing the resistance against which it is worked. Power and strength athletes incorporate resistance exercise programs in their yearly training plans. Resistance training is frequently included in the training of endurance athletes, too [51]. It was shown that the focus on a more explosive type of lifting (Olympic lifting) results in better power and strength gain compared to more traditional strength-based lifting, mainly because of inducing neural adaptation [52,53]. Early (within days or a few weeks) adaptations to training are achieved largely through changes in muscle recruitment patterns with later changes resulting from increases in muscle mass and changes in muscle composition [54].

From a clinical perspective, it is essential to recognize that an arbitrary threshold of 8 -10 minutes is often used to differentiate endurance exercises from power exercises, aiding in the classification of physical and nutritional activities based on their physiological and metabolic demands for tailoring effective training regimens (Figure 2)[46,55]. However, elements of strength and power sports are also included in sports games (e.g., football, tennis) and fighting sports (e.g., jiu-jitsu, boxing), where specific cyclic movements are interrupted with acyclic movements such as jumps, throws, and hits.



**Figure 2.** Classification of types of sports.

High-intensity interval training has recently become popular as a time-efficient way of promoting many of the physiological benefits associated with traditional endurance training [56,57]. As few as three training sessions per week, involving  $\leq 10$  min of intense exercise within a time commitment of  $\leq 30$  min per session, has been shown to improve aerobic capacity, skeletal muscle oxidative capacity, exercise tolerance and markers of disease risk after only a few weeks in both healthy individuals and people with cardiometabolic disorders [58].

## 2.2 FITT model

Scientifically, the FITT model - Frequency, Intensity, Time, and Type - provides a structured approach to optimizing exercise prescription. It allows integration of FITT components into personalized training plans, with health status and nutrition related strategies to enhance performance, recovery, and overall health [28] (Table 2).

**Table 2.** Examples of clinical integration of personalized training plans with health status and nutritional strategies. RM – repetition maximum.

FITT Component	Clinical Application	Clinical Integration with Nutrition
<b>Frequency</b> <i>Refers to how often an exercise or physical activity is performed within a specific time frame (e.g., per week).</i>	<b>Health Promotion:</b> Regular frequency (e.g., 3–5 sessions per week) improves cardiovascular health and reduces chronic disease risk.	Athletes training multiple times daily require staggered
	<b>Rehabilitation:</b> Adjusted frequency aids recovery for patients.	
	<b>Performance Optimization:</b> Athletes may train multiple times daily with rest periods during peak cycles.	

		meals to replenish glycogen and support recovery.
<b>Intensity</b>  <i>Refers to the level of effort required to perform an activity, often measured as a percentage of the individual's maximum capacity.</i>	<b>Cardiorespiratory Training:</b> Moderate to vigorous intensity exercise improves cardiovascular fitness.	High-intensity sessions necessitate increased carbohydrate intake for energy and protein intake for muscle repair.
	<b>Strength Training:</b> Prescribed as a percentage of one-repetition maximum (e.g., 60–80% 1RM).	
	<b>Chronic Disease Management:</b> Low intensity minimizes strain while promoting adaptations.	
	<b>Nutrition Link:</b> Intensity influences energy substrate utilization.	
<b>Time</b>  <i>Refers to the duration of each exercise session or the time spent on a specific activity.</i>	<b>General Guidelines:</b> Adults should aim for 150–300 minutes of moderate-intensity aerobic activity weekly.	Prolonged activities may require hydration and electrolyte management (depending on sweat losses), while shorter, high-intensity workouts may benefit from pre-workout meals.
	<b>Chronic Conditions:</b> Shorter bouts (10–20 minutes) suit beginners or those with limited tolerance.	
	<b>Endurance Athletes:</b> Extended durations (e.g., long runs) improve stamina.	
	<b>Nutrition Link:</b> Longer durations increase energy expenditure and require carbohydrate replenishment.	
<b>Type</b>  <i>Refers to the specific mode of exercise performed (e.g., aerobic, resistance, flexibility, or neuromuscular training).</i>	<b>Aerobic Exercise:</b> Improves cardiovascular and metabolic health.	Nutritional strategies vary; for example, in resistance training there are benefits from protein timing, while carbohydrate loading is emphasized in endurance sports.
	<b>Resistance Training:</b> Enhances muscle mass, strength, and bone density.	
	<b>Flexibility and Mobility:</b> Improves range of motion and may reduce injury risk.	
	<b>Neuromuscular Training:</b> Restores balance and coordination in rehabilitation.	
	<b>Nutrition Link:</b> Exercise type determines macronutrient requirements.	

The FITT principle applies broadly across clinical and performance domains, aiding health improvement, chronic condition management, and rehabilitation (Table 3).

**Table 3.** The framework of comprehensive integration of the FITT principle across both clinical and performance-oriented domains [9,32,59–62]. FITT – Frequency, Intensity, Time, and Type.

1. **Chronic Conditions:** The FITT principle is applied to create individualized exercise programs for diabetes, hypertension, heart disease, and arthritis.
2. **Post-Injury Rehabilitation:** Tailored plans help restore function and strength while preventing recurrence of injury.
3. **Mental Health:** Exercise programs improve mood, reduce anxiety, and enhance cognitive function.
4. **Prehabilitation in Surgery:** Exercise programs designed using the FITT framework help to improve physical and functional capacity before surgery, reducing postoperative complications, enhancing recovery, and improving outcomes in patients undergoing major surgical procedures.
5. **Paediatric and Geriatric Care:** Adjustments in the FITT framework accommodate age-specific needs, ensuring safe and effective physical activity.
6. **Nutritional Strategies:** The FITT principle helps to guide energy and nutrient requirements by aligning dietary plans with exercise demands.
7. **Training for Performance:** The FITT principle is used to optimize athletic performance by fine-tuning exercise frequency, intensity, time, and type to meet sport-specific goals, improve endurance, build strength, and prevent overtraining.

### 3. Basic concepts in exercise physiology and endocrinology

Exercise physiology studies the acute responses to single exercise exposures and the adaptations that occur at whole body and at tissue level in response to repeated exercise bouts [63]. Exercise endocrinology explores the inter-organ communication pathways activated by exercise to regulate metabolism, physiology, behaviour, and survival [26]. Historically, the classical view of exercise signalling focused on autonomic nerves releasing norepinephrine (noradrenaline), which triggered the release of cortisol and adrenal catecholamines [64]. These hormones are essential in the regulation of fuel metabolism according to the type, duration, and intensity of exercise, while also activating the physiological and behavioural responses characteristic of the fight-or-flight mechanism [65–67]. Key hormonal messengers include norepinephrine, epinephrine (adrenaline), cortisol, growth hormone (GH), insulin, insulin-like growth factor (IGF), and glucagon [68]. Later, it was discovered that exercise also stimulates paracrine and autocrine signalling, where locally produced molecules act on nearby cells and tissues rather than relying on the systemic circulation [69,70]. Recent research has expanded this view, identifying diverse organ-derived messengers now called exerkins [3,71]. They represent a group of bioactive molecules released from muscles, organs and other tissues. These diverse messengers are variably influenced by exercise and collectively underscore its role in inter-organ communication, metabolic regulation, and overall health (see section on exerkins 3.3).

#### 3.1 Acute response to exercise

Acute exercise is a potent physiological stressor, inducing widespread perturbations in numerous cells, tissues and organs. Physiological and metabolic responses to exercise share significant similarities with the general stress response observed in various biological systems [67]. The acute cardiovascular and metabolic responses to exercise depend on many factors, such as intensity, duration, type of exercise, and the person's health, fitness level, and psychological status. These responses are driven by the increased metabolic demands of contracting skeletal muscle and are modified by environmental stressors such as heat and altitude [1,72–74]. Various systems in the body, including the cardiovascular, respiratory, and endocrine systems, are activated to help maintain energy supply, oxygen delivery, and thermoregulation.

Common signs of activation in the cardiorespiratory system include increased heart and breathing rates to allow for greater cardiac output and oxygen intake. As exercise continues, the metabolic rate rises in response to the increased demands of physical activity, leading to heightened heat production. This elevated energy turnover and heat generation can be sustained throughout the duration of the exercise and for some time afterwards. The extent of this increase depends mainly on the intensity and duration of the exercise and the individual's fitness level. The metabolic rate can increase during moderate exercise to 5 to 20 times the resting energy requirement. In the case of very high-intensity exercise in elite athletes, this increase may exceed 100 times the resting level but such exercise is necessarily of very short duration.

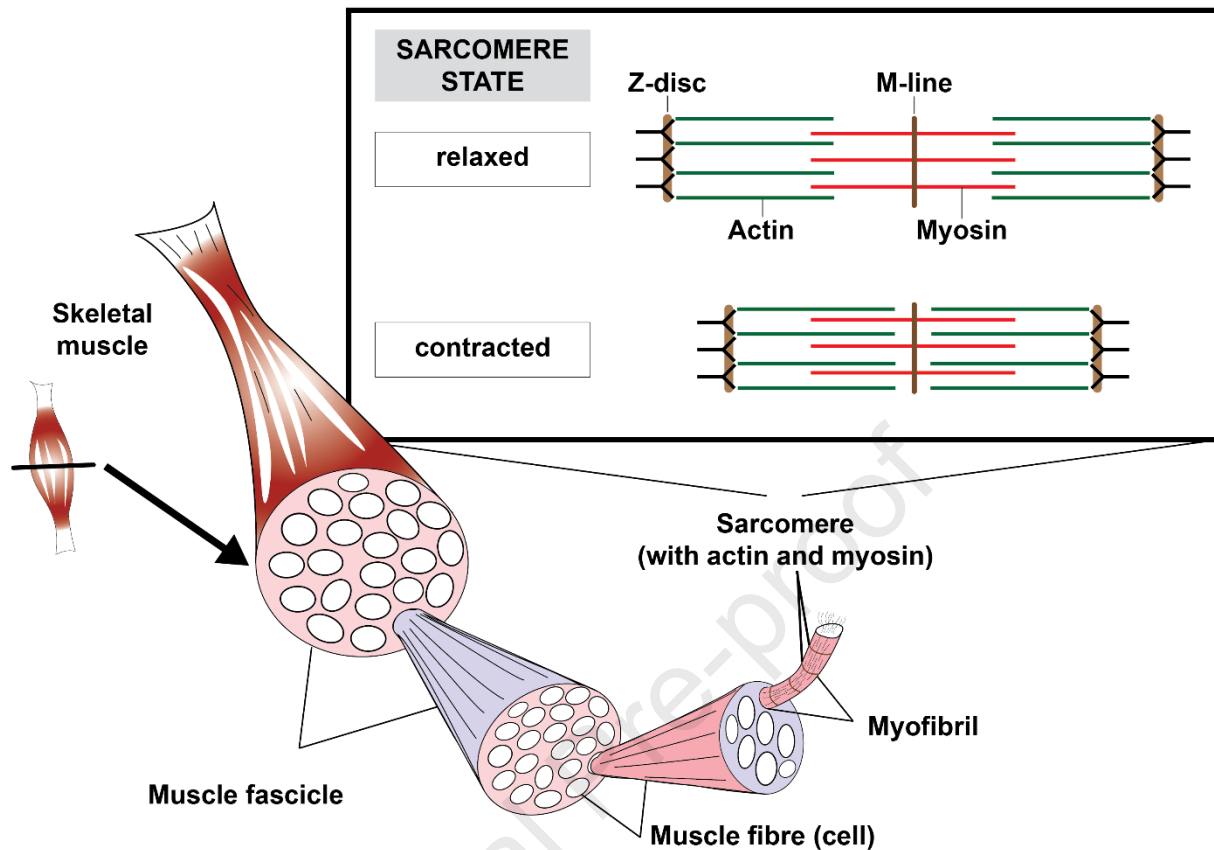
Physiological and metabolic responses are generally proportional to the relative stress as a fraction of the individual's maximum capacity rather than to the absolute energy demand [72]. Although there will be some individual variability, walking at a speed of 6 km/h requires the same amount of energy for the sedentary individual as for the trained athlete if body mass is the same, but the relative stress is very different [75]. Similarly, lifting a mass of 20 kg may be the maximum force achievable by an elderly sarcopenic individual, but is a small fraction of the force generating capacity of a trained power athlete.

### 3.1.1 Muscle

Skeletal muscle typically makes up about 40% of total body mass in a young lean male and about 35% in a young lean female, though this varies greatly, depending primarily on adiposity; muscle mass progressively declines with age and is strongly influenced by both genetic endowment and by habitual use [76]. Muscle responds to regular use with an increase in mass and in functional properties, and disuse leads to a loss of mass and function. The nature of the adaptation is specific to the stimulus (strength or endurance training, for example) and the magnitude of the adaptation is proportional to the training load (frequency, intensity and duration).

Each muscle contains hundreds or even many thousands of individual muscle fibres, each fibre being a single specialised cell. Two key proteins, actin and myosin, make up much of the muscle mass, and the interaction of these two proteins allows muscle cells to generate force [77]. These proteins form long overlapping microfilaments within the skeletal muscle fibres and these filaments can slide past each other, allowing the muscle to shorten; they interact to generate force (Figure 4). Other proteins provide supporting structures and are involved in the regulation of the interaction of these filaments, allowing precise control of activation and relaxation. Part of the myosin molecule functions as an ATPase, breaking down adenosine triphosphate (ATP) and so making chemical energy available to power muscle activity [77].

The functional unit of the muscle fibre is the sarcomere (Figure 3), and the functional properties of each fibre are closely related to the number of sarcomeres it contains: the more sarcomeres in parallel - i.e. the greater the physiological cross-sectional area - the greater the maximum force that a muscle can generate, but the number of sarcomeres in series will dictate the maximum speed of shortening [78]. Short, thick muscles are therefore good for strength, but long thin muscles are good for speed. Within the muscle, a number of muscle fibres, ranging from only a few in small muscles suited to delicate tasks to more than 1000 in large muscles, are innervated by a single nerve fibre: the motor nerve and the fibres it innervates form a motor unit, but each fibre is innervated by only one neuron [79].



**Figure 3.** Schematic presentation of muscle structure.

Individual fibres are activated on an all-or-none basis, and not all of the muscle fibres are recruited in submaximal contractions: only enough to generate the force necessary [80]. The higher the force required, the greater the number of individual muscle fibres that must be recruited. Those motor units with the lowest activation threshold (i.e. the first to be recruited) are those with a low speed of contraction and a high fatigue resistance: this makes sense as these fibres will be used most often in daily tasks [81]. As the weight to be moved is increased or the power output increases (i.e. an increased speed in running or cycling), progressively more motor units are recruited. In maximal efforts, all the muscle fibres are likely to be active. In prolonged exercise, some of the fibres that were recruited in the early stages may become fatigued and will cease to contribute to work performance and be replaced by others.

Receptors in the muscle and in the tendons can sense the length of the muscle and the force being generated; this allows the brain to know how many fibres need to be recruited and how to coordinate the recruitment of the different muscles. Muscle spindles signal information about the length and velocity of a muscle, while Golgi tendon organs signal information about the load or force applied to a muscle [82]. In well-trained and highly motivated individuals, it can be demonstrated that the muscle is working maximally - adding an electrical stimulus to the nerve or directly to the muscle does not generate any additional force - but in untrained individuals, there is usually some functional reserve [83].

Human muscle fibres can be classified in a number of ways, depending on their maximum speed of contraction, their biochemical characteristics, their molecular composition and their resistance to fatigue [84]. Contraction occurs by interaction of actin and myosin filaments within the fibres, and the speed of contraction is determined largely by the ATPase activity of the myosin: the faster ATP can be hydrolysed to release energy, the faster contraction can occur. Three main fibre types are generally recognised in

skeletal muscle, but there may be overlap in some characteristics, and substantial changes can be induced by training. The type II muscle fibres of an elite marathon runner may have a higher oxidative capacity than the type I fibres of the sprinter, but they are still distinct from the type I fibres present in the same muscle.

**Box 2.** The primary muscle fibre types.

**Type I slow oxidative (also called slow twitch or fatigue-resistant fibres)**

These muscle fibres are dark red because of their high myoglobin content and high density of blood capillaries; they contain many mitochondria and so have a high oxidative capacity, have a slow peak contraction velocity and are relatively fatigue resistant. They are present in higher numbers in postural muscle. Elite endurance athletes have higher than normal numbers of these fibres.

**Type IIa fast oxidative (also called fast twitch, 2A or fatigue-resistant fibres)**

These muscle fibres also have a high myoglobin content and high density of blood capillaries, contain many mitochondria and so have a high oxidative capacity, but they can hydrolyse ATP at a high rate and so have a fast peak contraction velocity. They are resistant to fatigue, but less so than the type I fibres.

**Type IIx fast glycolytic (also called II B, 2B or fatigable fibres).**

These muscle fibres have a low myoglobin content, low capillary density and fewer mitochondria. They can hydrolyse ATP at a high rate and so have a fast peak contraction velocity. They have a high activity of glycolytic enzymes and contain large amount of glycogen. They are useful when high power outputs are needed but they fatigue rapidly. The muscles of elite sprinters have high proportions of these fibres.

The distribution of muscle fibre types is important in determining not only function and metabolism, but also the sports for which the athlete is most suited. The individual with mostly slow twitch muscle fibres will never be a world-class sprinter.

### 3.1.2 Respiratory function

The major source of energy from all but the briefest of exercise comes from oxidative metabolism, requiring a constant supply of both oxygen and substrates to the exercising muscles (see Module 37.2). An individual's maximum oxygen uptake ( $VO_{2max}$ ) represents the highest rate of aerobic energy production that can be achieved but it has been debated for more than a century whether the primary limitation is the capacity of the cardiorespiratory systems to deliver oxygen or of the muscle to use that oxygen [85]. The importance of  $VO_{2max}$  for endurance athletes such as marathon runners lies in the fact that endurance capacity/performance is largely a function of the rate of aerobic energy metabolism that can be sustained for prolonged periods: the higher the fraction of aerobic capacity that must be used, the shorter the time for which a given pace can be sustained. Improving performance requires either an increase in  $VO_{2max}$ , an increase in the fraction of  $VO_{2max}$  that can be sustained for the duration of the race, or a decrease in the energy cost of running. In practice, all of these can be achieved with suitable training and nutrition strategies, but only relatively small improvements in running economy (the oxygen cost of running) are possible [86]. It is important to note that the athlete engaged in very prolonged exercise can compensate to some degree for a low  $VO_{2max}$  by working at a high fraction of that aerobic capacity. For the middle-distance runner and other athletes in sports lasting a few minutes (e.g. rowing, pursuit in cycling) a high  $VO_{2max}$  is an absolute requirement for successful performance.

Limiting factors to  $VO_{2max}$  have been discussed and debated over the years. Part of the reason for the debate is that the limiting factor seems to vary in different types of exercise, in different environments and in different individuals. Typically, the lungs are not considered to limit performance at sea level in the absence of lung disease, so attention has focused primarily on whether the limitation lies in the delivery of oxygen by the cardiovascular system or in the ability of the working muscles themselves to utilise oxygen. However, at altitude, the picture is clearer: the oxygen content of the inspired air falls as the elevation above sea level increases, leading to a progressive fall in arterial oxygen saturation, decreased oxygen transport and a fall in  $VO_{2max}$ . This accounts for the reduction in running performance in events lasting more than a few minutes that is generally seen at altitudes above about 1500 metres. At higher speeds involved in cycling, the decreased air resistance at moderate altitude more than compensates for the decreased partial pressure of oxygen, and world records for the distance covered in 1 hour are typically set at moderate altitude, with an optimum elevation of about 2000-2500 m [87]. Some highly trained runners, however, show arterial desaturation - clear evidence of a limitation at the level of the lungs - in maximal exercise even at sea level. The effect can be reversed, and  $VO_{2max}$  increased, by breathing air with an increased  $O_2$  content [88]. This effect is not normally seen in trained but non-elite runners, suggesting there may be a pulmonary limitation in elite endurance-trained runners, perhaps because all other steps in the oxygen transport chain have adapted maximally in response to training.

Research investigating the responses to training of the inspiratory muscles also provides some support to the idea that there may be a pulmonary limitation. For example, 4 weeks of inspiratory muscle training for 30 min/day has been shown to increase time to fatigue in both trained and untrained subjects [89]. Not all subsequent research has reported the same findings, but the current consensus is that beneficial effects on performance in both normoxia and hypoxia can be obtained by even small amounts of respiratory muscle training [90]. As in other cases where divergent results are found, this may relate to the exercise task or to the specific subject population. Both factors are likely to affect the limitations both to  $VO_{2max}$  and to endurance performance.

### 3.1.3 Cardiovascular function

Effective delivery of oxygen and nutrients to the working muscles while maintaining adequate blood flow to other essential organs, including the brain, requires the ability to develop a high cardiac output. A high flow through the muscles is also necessary to remove the products of metabolism from those tissues, to carry heat from the active muscles to the skin, and to transport hormones from the sites of their production to the sites of their action.  $VO_{2max}$  may be limited by the delivery of oxygen to the active muscles or by the ability of those muscles to use oxygen. The findings in this research area are conflicting, perhaps because the underlying factors might differ in various exercise contexts. It will depend, for example, on whether the exercise involves large muscle groups, such as in cross-country skiing, or whether only a small muscle mass is involved. In the latter case, the heart is clearly capable of delivering more oxygenated blood than the muscle can accommodate, but in the former example, it is easier to believe that oxygen supply might be limiting.

There is strong experimental evidence to support the idea that the limitations to oxygen uptake in most whole-body exercise situations are imposed by the cardiovascular system and that the limitation may lie at any one or more of several stages. The key element seems to be the maximum cardiac output that can be achieved [91]. The size of the heart, and more specifically the size of the left ventricle, is important as it determines the stroke volume - the amount of blood ejected with each beat [91]. Maximum heart rate is often slightly lower in trained athletes than in sedentary individuals [92], but peak stroke volume is the primary determinant of the maximum cardiac output. Cross-sectional studies of athletes of varying levels of ability from different sports, and comparisons with sedentary individuals show that the maximum cardiac output that can be achieved is closely correlated with both  $VO_{2max}$  and endurance performance. In elite endurance athletes, the cardiac output can reach 40 L/min, compared with the maximum of about 20 L/min that the sedentary individual can achieve (Table 5). The high stroke volume of the endurance athlete is also responsible for the characteristically low heart rate displayed by these individuals at rest. Resting cardiac output is the same for the elite athlete as for the sedentary individual. As the stroke volume is high, the

same cardiac output can be achieved at a much lower heart rate. A high blood volume will also benefit the endurance athlete by helping to maintain the central venous pressure, thus maintaining stroke volume [93].

**Table 5.** Cardiovascular characteristics at rest and during maximal exercise at varying levels of endurance fitness.

Training Status	Condition	Heart Rate (beats/min)	Stroke Volume (mL)	Cardiac Output (L/min)
<b>Sedentary</b>	Rest	70	70	5
	Max	200	100	20
<b>Moderately Trained</b>	Rest	40–60	100	5
	Max	195	150	29
<b>Elite Endurance</b>	Rest	30–40	150	5
	Max	190	200	38

The oxygen-carrying capacity of the blood is important, and this is determined by the haemoglobin concentration and the total blood volume. Almost all the oxygen in the blood is transported bound to haemoglobin and each gram of haemoglobin can bind 1.34 mL of oxygen, far exceeding the solubility of oxygen in plasma. Normal reference ranges will depend on the population studied and the laboratory methodology, but the average male has a higher haemoglobin concentration (140-170 g/L) than the average female (120-140 g/L) and so has about 15% more oxygen in the blood when it leaves the lungs [94]. This difference accounts in part for the generally higher aerobic capacity of males. The importance of the oxygen carrying capacity of the blood is also the reason for the negative effects of anaemia on performance and for the various strategies used by athletes to increase the haemoglobin content of the blood: these strategies include high altitude training, the use of agents such as erythropoietin and its analogues that stimulate the formation of new red blood cells, and the use of blood transfusions prior to competition [95]. Even though some of these strategies are prohibited by the World Anti-Doping Agency (WADA), they have been used - and almost certainly still are being used - by some athletes.

The delivery of oxygen to the muscles is also influenced by the density of the capillary network within the muscles and by the size of the individual muscle fibres, which determine the diffusion distance from the capillary to the site of utilisation in the mitochondria. An increase in the number of capillaries or smaller muscle fibres means less distance for oxygen (and blood-borne substrates) to diffuse. Muscles that rely on aerobic metabolism are generally characterised by smaller fibres and by a large number of capillaries: in response to endurance training, the capillary density can increase substantially [96].

At the cellular level, exercise activates stress signalling pathways, including the hypothalamic-pituitary-adrenal (HPA) axis, producing stress hormones such as cortisol and catecholamines (epinephrine and norepinephrine). These hormones are critical in mobilizing energy substrates, including glucose and free fatty acids, to sustain the heightened metabolic rate [65,97]. Moreover, exercise triggers the release of various molecules, known as exerkines, which facilitate communication between organs, tissues, and cells during exercise. [3]. They are crucial in coordinating the body's response to exercise and adaptation to exercise training. The discovery of these components has shifted our understanding from a muscle-centric view of the body's response to exercise to a broader perspective that considers how the entire body responds to physical activity [72].

When the energy demands of active muscles for exercise are not met, the intensity of the exercise must be reduced or the activity must be stopped. Limiting factors are usually complex but mainly depend on the nature of the activity and an individual's physiological characteristics.

The energy metabolism in exercise is described in Module **37.2**.

### 3.1.4 Fatigue

All exercise, if it is sufficiently intense or prolonged, will result in a subjective sensation of fatigue. From studies of muscle fatigue carried out in the late nineteenth century, it was generally concluded that fatigue was in part a local phenomenon occurring within the active muscle but the primary cause of fatigue was attributed to events within the central nervous system that acted to prevent irreversible damage [98]. This conclusion was largely based on the observation that direct electrical stimulation of small muscles of the hand muscle or their motor nerve could still produce strong contractions even when voluntary activation of the muscle was ineffective. Technical developments that allowed the collection and analysis of samples from muscle are perhaps responsible for the focus on muscle fatigue that developed in second half of the twentieth century. Results of muscle biopsy analysis, for example, showed a clear link between the depletion of the muscle glycogen store and the onset of fatigue, at least in prolonged cycling exercise [99]. More recently, however, there has been a renewed recognition of the role of the brain in fatigue, even though the mechanisms remain uncertain. This has been described as the action of a 'central governor' that acts to regulate pace and effort to optimize performance [100]. This is reminiscent of the work of Lagrange, who, in 1889, referred to fatigue as a *"regulator, warning us that we are exceeding the limits of useful exercise, and that work will soon become dangerous"* [101]. The danger referred to is that of irreversible damage to the muscles or other tissues, with the attainment of excessively high body temperatures being a real concern.

Many different factors can contribute to fatigue, and at some point, the intensity must be reduced or the exercise terminated. There are, however, some interventions that can enhance performance and, by implication, affect specific aspects of the fatigue process, and these observations may give some clues as to where the limitation lies. Each of these interventions, however, will usually produce only a small benefit, so a limitation remains – this may be the same limitation that has simply been postponed or it may be that a different limitation is invoked.

In very high intensity exercise that leads to fatigue within 1–2 minutes, there is a rapid decline in the intracellular concentration of creatine phosphate (CP) in the active muscles as high energy phosphate groups are transferred to ADP to maintain the muscle ATP concentration [102]. ATP concentration will fall, but only slightly, and increasing concentrations of ADP may impair muscle contractility. Increasing the pre-exercise muscle CP content by feeding creatine supplements for a few days can lead to higher power outputs and a delay in fatigue, suggesting that the fall in the contribution of CP to energy supply is a factor in fatigue [103,104]. In exercise that causes fatigue within about 1–10 minutes, the high rates of glycolysis produce pyruvate at a rate faster than the ability of oxidative metabolism to remove it, and a marked acidosis develops within the muscle cells as the high rate of hydrogen ion formation overwhelms the buffer capacity of the muscle. Increasing the body's capacity to buffer the acidity (by pre-exercise ingestion of bicarbonate or citrate) can allow greater amounts of lactate to be formed before the pH within the cell becomes limiting [105]. In contrast to the old idea that lactate production was *"a bad thing"* that caused fatigue, it is now recognized that without producing the lactate, there will not be sufficient power to allow high power outputs to be achieved. If the pre-exercise muscle glycogen content is low, performance of exercise lasting a few minutes is impaired, even though lactate levels in the muscle and blood are lower at the point of fatigue: in contrast elevated pre-exercise muscle glycogen content results in better performance even though lactate levels are higher [106]. Lactate can therefore be dissociated from whatever is causing fatigue. There also seems to be a "functional reserve" within the muscle that cannot be accessed by voluntary activation [107].

In prolonged exercise, it is more difficult to identify a single factor that might be responsible for fatigue. We know that performance in cycling tests lasting about 1–3 hours can be improved by increasing the muscle glycogen store and is impaired if exercise begins in a glycogen-depleted state [99]. Feeding CHO during this type of exercise can also delay fatigue, and these findings suggest that there is a metabolic component to fatigue. These findings are true for exercise in a temperate environment but endurance exercise capacity is progressively impaired as the ambient temperature increases above about 10°C [108]. When the temperature is high, there seems still to be an adequate amount of glycogen remaining in the muscle at the point of fatigue, suggesting that glycogen depletion is unlikely to be the cause of fatigue in prolonged exercise in the heat, even though this may be the case in cooler environments. Nevertheless, feeding a

high-CHO diet in the days prior to exercise can improve endurance performance in the heat even when glycogen availability should not be limiting, so other factors must be involved [109]. Pre-exercise cooling, either by immersion in cold water or by ingestion of cold drinks, can improve endurance performance in warm environments, apparently by delaying the time until a critical elevation of core temperature occurs [110]. Repeated exposure to exercise in the heat improves performance in the heat by a number of mechanisms, including an increased sweating sensitivity, an increased plasma volume and also a lowering of the basal pre-exercise core temperature [111]. Endurance exercise performance in cool conditions is also improved by short periods of heat acclimation [112].

The role of the central nervous system (CNS) in exercise-associated fatigue is best demonstrated by several pharmacological interventions that have been shown to affect exercise performance without any obvious cardiovascular or metabolic effects. Paroxetine, a drug that acts on CNS neurons to prolong the action of the neurotransmitter serotonin, can reduce performance [113]. Subsequent studies using drugs with opposing actions have been shown to enhance performance in both human and animal models [114]. A range of stimulant drugs, including amphetamines, can enhance performance by actions on neurons in the brain that use dopamine as a neurotransmitter [115]. This effect seems to be particularly marked in endurance exercise in the heat, and one of the dangers associated with the use of drugs that can override the sensation of fatigue is that the outcome that Lagrange referred to may occur [116]. Fatal hyperthermia during hard exercise in the heat is rare, but it can occur, as in the case of Tom Simpson in the 1967 Tour de France.

### **3.2 Adaptation to exercise training**

The aim of training is, or at least should be, to increase functional capacity and to bring about improvements in performance. Exercise impacts every organ and tissue in the body; however, the induced adaptive responses to training are specific to the training stimulus, the muscles being trained, and individual physiological characteristics, including the genetic response [73].

A well-designed strength training program will have little effect on endurance and vice versa; one leg can be specifically trained for strength and the other for endurance with relatively little cross-over [117]. Training should therefore be designed to address the event-specific limitations to performance, and this will differ between individuals as well as between events. It is important to note, however, that some adaptations to training are not entirely specific, as the effects on the central cardiovascular system will be similar whether the athlete is running, cycling or performing any other activity that engages a large muscle mass. The performance improvement in response to a training program is generally proportional to the training load (i.e. the intensity, duration and frequency of the training sessions) (see 2.2). Generally, the harder an athlete trains, the greater the improvements in performance that result. There is a limit, however, beyond which further increases in training will result in poorer performances: this is usually referred to as an overtraining syndrome, but few athletes ever reach this level of training. Where it does happen, overtraining is associated with impaired performance, chronic fatigue leading to an inability to sustain the training program, and an increased risk of infectious illness [118].

It used to be thought that a primary role of nutrition in the athlete's diet was to support consistent, intensive training by promoting recovery between training sessions. While recovery is a key aim of post-exercise nutrition strategies during periods of intensive training, nutrition also has a key role in promoting the adaptations that take place in muscle and other tissues in response to each training session [119]. Training provides the stimulus to turn on the genes responsible for the expression of functional proteins: strength training leads to synthesis of more actin and myosin, making muscles bigger and stronger, while endurance training leads to synthesis of more oxidative enzymes and of all the other components necessary for endurance performance. Increased rates of the synthesis and breakdown of specific proteins must be taking place in the post-exercise period. The response is modulated by the nutrients, metabolic and hormonal environment, and this in turn can be modified by food intake before, during and after training. Ingestion of a small amount (typically 20-40 g) of mixed protein or a corresponding amount of essential amino acids (especially leucine) after a training session can stimulate protein synthesis for up to 24 hours after resistance training [120]. Early studies focused on measurement of rates of protein synthesis or turnover,

but more recent studies have shown that these acute responses translate to functional outcomes [119]. Without the training stimulus, though, it is clear that adaptation will not take place in the muscle.

Training should aim to address the factors that limit exercise performance, shifting the barriers to better health or performance. In the case of strength training, a significant part of the adaptation that takes place, especially in the early stages, is within the nervous system: strength improves after only a few training sessions, before any measurable changes in muscle structure have taken place [76]. In the case of endurance training, a large number of adaptations have been identified, both in the central circulation and in the muscles themselves. The pumping capacity of the heart is increased, primarily by an increase in stroke volume as a result of an increase in left ventricular volume. Blood volume and red blood cell mass both increase, thus increasing the total oxygen carrying capacity. New capillaries grow in the endurance-trained muscle, shortening the diffusion distance for oxygen and nutrients between the circulation and the muscle fibres. Mitochondrial mass increases, and with it the activity of the enzymes involved in the oxidation of CHO and fat. There is, in particular, an increase in the capacity of the trained muscle to oxidise fat, thus decreasing the reliance on CHO during exercise, though this adaptation can be reversed to some extent by feeding a high-CHO diet [121]. As mentioned above, this may be beneficial when the availability of CHO is limited but is of questionable value in other situations as it will lead to an increased energy cost of exercise. Tissues respond to disuse with a reversal of the central and local adaptations caused by training [122].

Regular training also has important effects on the brain, though these are less well understood than many of the peripheral adaptations. One important learned response is the ability to judge pace, so that effort can be distributed evenly across the whole duration of an event: this is not an innate ability but is learned by repeated experience [123]. A common mistake of novice athletes is to set off too fast and then to fade badly in the later stages or to finish with too much still in reserve. The limitation to performance in this situation may be very different from that affecting the experienced athlete.

### 3.3 Exerkines

Recent progress in exercise physiology and endocrinology has revealed that exercise triggers communication between organs and tissues through endocrine, paracrine, and autocrine signalling pathways [26]. This process is mediated, at least in part, by exerkines, a diverse group of bioactive molecules released by muscles and other tissues in response to physical activity [3,10,124–127]. While myokines, produced during muscle contractions, are the most extensively studied, other exerkines - such as cardiokines (from the heart), hepatokines (from the liver), adipokines (from white adipose tissue), bapokines (from brown adipose tissue), osteokines (from bone) and neurokines (from neurons) - are less well understood.

Currently, exerkines are thought to be key drivers of the systemic health benefits of exercise, facilitating communication between cells, tissues, and organs. They play crucial roles in improving cardiovascular, metabolic, immune, and neurological health and hold promise for managing conditions such as cardiovascular disease, type 2 diabetes, cancer, and obesity and promoting healthy aging [1,32].

## 4. Muscle mass

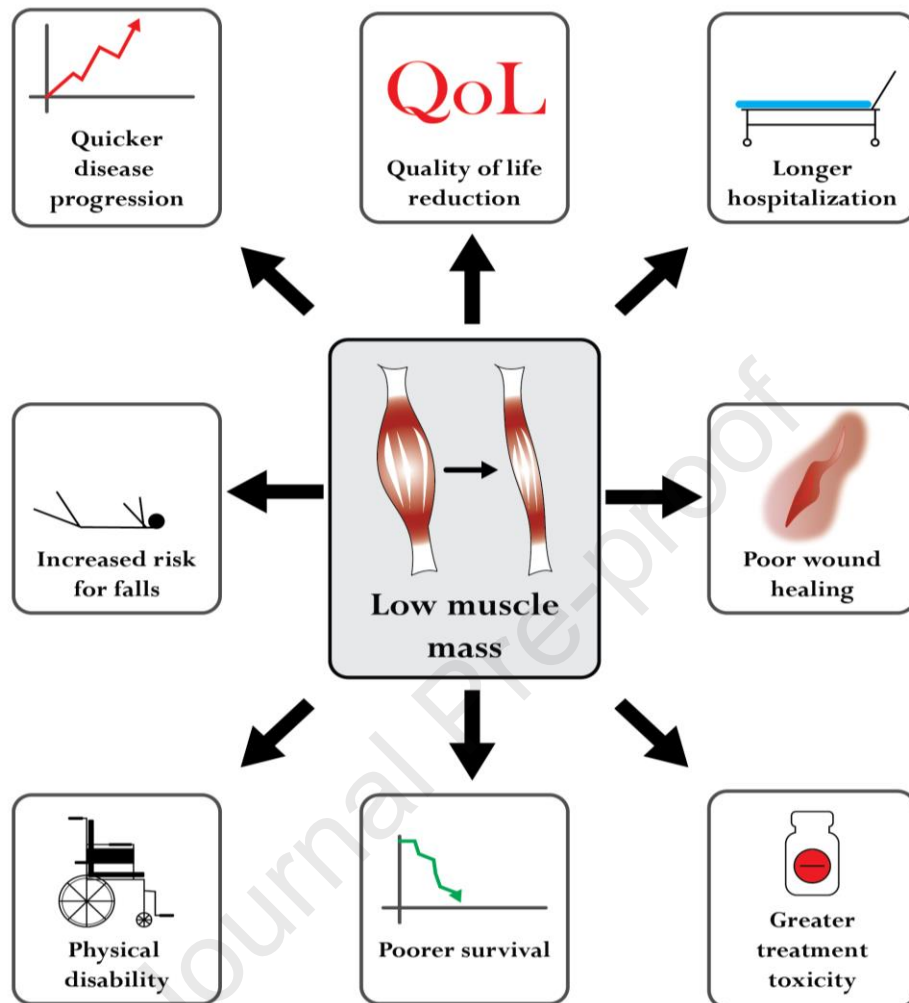
Muscle health has become an increasingly prominent topic in recent years due to several converging factors. As the global population ages, the prevalence of muscle loss continues to rise, leading to higher rates of sarcopenia, sarcopenic obesity, and frailty – interrelated conditions associated with functional and clinical impairment [27]. Additionally, a growing number of chronic diseases - including cancer, kidney disease, and metabolic disorders - are recognized for their detrimental effects on muscle mass and function. More recently, the widespread use of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) for obesity management has further highlighted the importance of preserving muscle during weight loss [128]. As these medications gain popularity, concerns about their potential impact on muscle mass underscore the need

for strategies to mitigate muscle deterioration and optimize long-term health outcomes. Consequently, research on muscle preservation, targeted nutrition, and resistance training has gained significant traction, emphasizing the crucial role of muscle in overall health, longevity, and disease management.

Muscle is essential for overall health and well-being. Muscles serve vital metabolic functions that extend well beyond movement and strength [128]. Often overlooked in discussions on weight loss, muscle acts as a key metabolic organ, functioning as an amino acid reservoir crucial for responding to stress, trauma, and infection. It also synthesizes and stores glutamine, a key amino acid essential for nitrogen transport and immune function. Additionally, muscle mass plays a significant role in glucose homeostasis, facilitating glucose uptake in response to insulin to maintain normoglycaemia. Muscle-derived myokines, signalling molecules released by muscle cells, function as endocrine factors that regulate systemic metabolism, energy balance, and inflammation (see 3.3). Given these roles, muscles are integral to immune function, as myokines influence immune cell proliferation, activation, and distribution. This interdependence between muscle and the immune system underscores the significance of muscle mass for overall health [129–131].

Muscle loss can occur independently or in the context of malnutrition, sarcopenia, sarcopenic obesity or frailty, each of which exacerbates its impact on health and functional outcomes. A particularly significant aspect of low muscle mass is that it can affect individuals of any age or body mass, including those with a normal weight or obesity [132]. Additionally, muscle mass loss may be concealed behind an apparently stable body weight, making it difficult to detect through weight monitoring alone. We direct the reader to additional publications for a more in-depth discussion on body composition assessment methods [30,133].

Across different conditions and clinical populations, low muscle mass has been shown to be a significant predictor of poor health outcomes, leading to reduced physical function, impaired immunity and wound healing, increased infection risk, disability, lower quality of life, and shorter survival, among other outcomes (Figure 4) [30]. This can be worsened by the accelerated muscle loss observed in certain conditions or clinical situations. As individuals age or experience prolonged immobility, muscle protein synthesis declines while catabolic processes accelerate, leading to a progressive reduction in muscle mass and function. Chronic diseases further exacerbate this process by inducing metabolic stress and inflammatory responses that promote muscle degradation. Emerging research underscores the role of mitochondrial dysfunction in muscle atrophy, particularly in aging and disease-related catabolic states [134]. Impaired mitochondrial bioenergetics and reduced capacity for energy production contribute to muscle wasting, highlighting the need for targeted interventions to preserve muscle health.



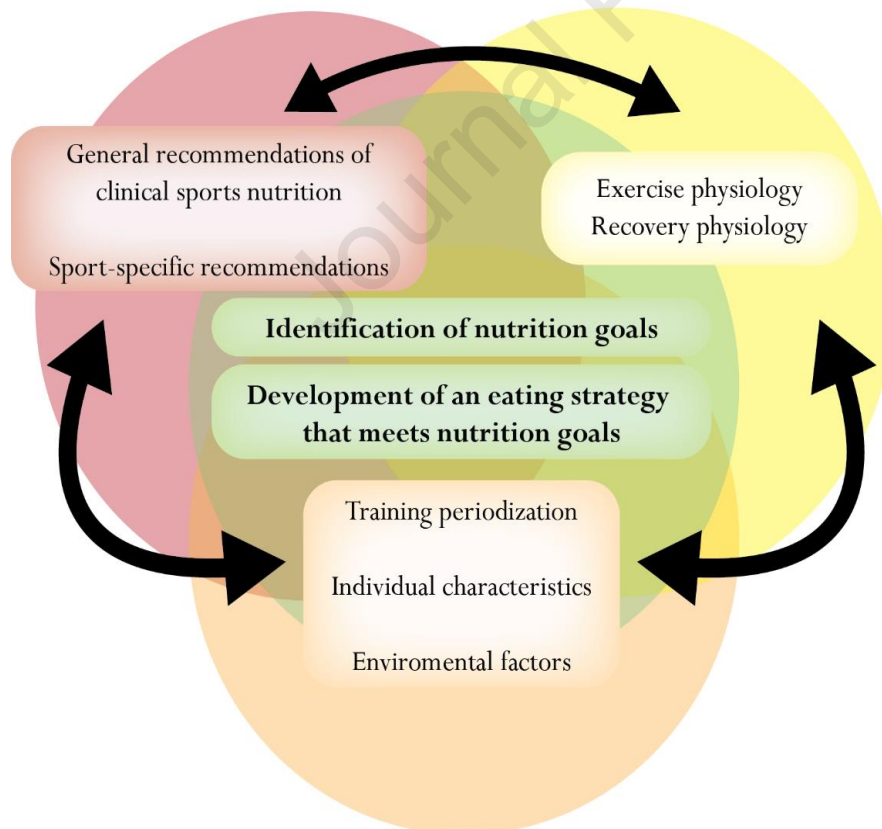
**Figure 4.** Clinical outcomes associated with low muscle mass (Adapted from [30]).

Exercise interventions, particularly resistance and endurance training, play a crucial role in enhancing muscle mass, function, and overall metabolic health. A multimodal strategy that combines nutrition with structured physical activity is essential for optimal outcomes [30]. To effectively prevent and manage muscle loss, clinical practice should prioritize early screening, assessment, and personalized nutrition interventions. Increasing awareness among healthcare providers, patients, and caregivers is critical to closing the gap between research and practical application. Additionally, a multidisciplinary approach that includes dietitians, physicians, physical therapists, and exercise physiologists should be integrated into patient care to ensure comprehensive and effective management of muscle health.

## 5. Concepts in sports nutrition

## 5.1 Sports nutrition

Sports nutrition is intrinsically linked to understanding the body's physiological processes during exercise and recovery after exercise. Nutrition serves as a fundamental determinant of individual health, influencing physiological adaptation to exercise training, regulation of body mass and composition, and optimisation of athletic performance [135]. This concept highlights the primary distinction between sports nutrition strategies and the general nutritional recommendations outlined in public health guidelines. Therefore, sports nutrition is not merely about prescribed diets but a dynamic nutritional strategy designed to support the physiological demands imposed by exercise [136]. It is based on scientific understanding of exercise-induced metabolic, molecular and systemic adaptations, encompassing processes such as energy metabolism, substrate availability, muscle recovery, immune function and performance optimization. Currently, an appropriate strategy for nutritional support is based on evidence provided by general scientific recommendations and then applied to various forms of endurance and dynamic voluntary exercise as well as to specific forms of power and strength sports. Therefore, the guidelines of sports nutrition represent various general recommendations for energy intake, amounts and composition of nutrients and fluid intake, and also specific recommendations for the type of sport and/or exercise activity and for different phases in the training process (Figure 5). Integrating scientific findings with insights from applied nutrition on how to cover individual metabolic needs of exercise has led to an increasingly sophisticated practice of sports nutrition. The concept of nutrition periodization is used in performance-oriented physical activities [137,138]. It enables strategically adjusted consumption of key nutrients depending on an individual's specific needs, and aims to enhance athletic performance and regeneration, thus allowing an athlete to reach his or her full genetic potential and to benefit maximally from physical activities that alternate in duration and intensity [139].



**Figure 5.** Personalized sports nutritional strategy.

Ultimately, the most critical aspect of sports nutrition is translating these scientific principles into practical, actionable nutrition plans. Athletes and physically active individuals must be equipped to apply food, fluid, and supplement strategies that align with their personal goals, training schedules and health needs. Furthermore, these plans must be culturally sensitive, practically feasible and tailored to the individual's lifestyle, food preferences and social environment. The culture of the sport itself, alongside the influence of coaches, teammates, and media, also shapes athletes' nutritional beliefs and behaviours. Therefore, effective sports nutrition education must be delivered in ways that are personalized and responsive to both individual and sport-specific contexts.

## **5.2 Clinical sports nutrition**

In recent years, knowledge in the fields of sports science and nutrition has expanded significantly, fostering a dynamic environment for applied research in sports nutrition and exercise medicine. To maximize both health and performance, nutritional strategies must be tailored to the specific physiological demands of each individual engaged in physical activity. Recent advances in research have further emphasized the essential role of personalized nutrition, not only in optimizing athletic performance, but also in enhancing recovery, preventing injuries, supporting rehabilitation and optimizing metabolic health. This growing body of evidence has led to the development of the concept of clinical sports nutrition, a specialized field that integrates performance nutrition of athletes with individualized nutritional care for all physically active individuals managing medical conditions, injuries, or chronic health concerns [140–143]. An individualized approach is fundamental to the concept of clinical sports nutrition, which integrates principles from both sports nutrition and clinical nutrition. To ensure consistency and alignment with established medical and nutritional frameworks, sports nutrition interventions for physically active individuals should be guided by evidence-based principles and, where appropriate, align with ESPEN terminology in clinical nutrition [144]. Furthermore, when metabolic demands of exercise are not met by appropriate nutritional intake of energy and nutrients, this can lead to physiological dysfunction and clinical manifestations as different disease states (e.g., The relative energy deficiency in sport [REDS] syndrome - see Topic 42 and Module 42.2). REDs represents a form of nutritional disorder known as energy malnutrition, with consequences for both performance and health [145].

The combination of exercise and tailored nutrition is a powerful tool to enhance metabolic health and physical performance in both healthy and clinical populations [135,140]. Exercise-induced metabolic demands are influenced by the type, intensity, duration and frequency of activity, as well as by the total muscle mass engaged [146]. These factors determine energy expenditure and substrate utilization, and the physiological adaptations necessary for performance, recovery and long-term muscle health. Maintaining muscle health requires a combination of regular exercise and targeted nutritional support, including adequate protein intake—particularly leucine and other essential amino acids—alongside  $\beta$ -hydroxy- $\beta$ -methylbutyrate (HMB), vitamin D, and n-3 polyunsaturated fatty acids (n-3 PUFA), all of which have been shown to support muscle preservation, enhance recovery, and improve functional outcomes, especially in populations at risk of muscle loss [30,147–150]. In both clinical practice and exercise settings, evidence supports the inclusion of selected oral nutritional supplements during periods of increased nutritional risk or reduced dietary intake, as they provide targeted nutritional support to optimize immune function, muscle preservation, recovery and overall metabolic health [104,151].

## **Funding**

There was no funding support.

## **Conflict of interest**

Authors have no conflicts of interest.

## Authors contributions

NRK, CP, RJM Conceptualization; NRK Project administration; NRK Resources; NRK Supervision; GT Visualization; NRK, GT, CP, RJM Writing- original draft; NRK, GT, CP, RJM Writing- review & editing.

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