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CURRENT OPINION

Skeletal Muscle Hypertrophy with Concurrent Exercise Training: Contrary Evidence for an Interference Effect

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Abstract Over the last 30+ years, it has become axiomatic that performing aerobic exercise within the same training program as resistance exercise (termed concurrent exercise training) interferes with the hypertrophic adaptations associated with resistance exercise training. However, a close examination of the literature reveals that the interference effect of concurrent exercise training on muscle growth in humans is not as compelling as previously thought. Moreover, recent studies show that, under certain conditions, concurrent exercise may augment resistance exercise-induced hypertrophy in healthy human skeletal muscle. The purpose of this article is to outline the contrary evidence for an acute and chronic interference effect of concurrent exercise on skeletal muscle growth in humans and provide practical literature-based recommendations for maximizing hypertrophy when training concurrently.

Key Points

In response to acute concurrent exercise, various lines of evidence suggest blunted hypertrophic potential compared to resistance exercise alone, but the concurrent training data in humans do not fully support this notion.

Aerobic exercise training alone can induce hypertrophy, and concurrent exercise training may augment the hypertrophic response to resistance exercise training in some circumstances.

Maximal hypertrophic potential with concurrent exercise training may be achieved by (1) separating exercise bouts by 6–24 h, (2) adopting strategies that minimize overall exercise volume (i.e., utilizing high-intensity intervals, 2–3 days of aerobic exercise, and $\leq$2 days of leg lifting), and (3) favoring cycling as opposed to running.

1 Introduction

Combining aerobic and resistance exercise within the same training program (termed concurrent exercise training) is a practical paradigm that conforms to the American College of Sports Medicine's guidelines for general health and fitness [1]. While this strategy is often employed by athletes, rehabilitation clinicians, and recreational exercisers, the 'specific adaptation to imposed demands' principle of exercise training stipulates that optimal adaptation from a

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given exercise type is achieved by mode-specific training (i.e., not combined training) [2]. In 1980, Dr. Robert Hickson [3] was the first to experimentally evaluate the effects of combining aerobic and resistance exercise within the same training regimen. Following 10 weeks of training, Hickson reported that concurrent training (6 days per week of aerobic exercise, 5 days of leg resistance exercise, all at high intensity) interfered with leg strength improvements (specifically beyond 7 weeks of training) relative to resistance exercise alone but yielded similar aerobic capacity improvements as aerobic exercise alone. Since this seminal investigation, it has become axiomatic that concurrent exercise training interferes with resistance exercise adaptations.

A hallmark adaptation to resistance exercise training is skeletal muscle hypertrophy. As such, a number of investigations since Hickson's original study have compared the hypertrophic response from resistance to concurrent exercise training. Few studies have reported blunted growth at the cellular level with concurrent training, and this was only in slow-twitch muscle fibers of the thigh [4–6]. Similar to Hickson's study, an important factor to consider for these investigations was that overall training stress on the legs was considerable (30–50 min of continuous and/or interval aerobic exercise at least 3 days per week and multiple sets of four different lower-body resistance exercises performed at least 3 days per week). Greater overall exercise volume may have caused chronic fatigue and/or overtraining in the concurrent versus resistance training groups. Interestingly, to our knowledge, no study to date has reported attenuated whole muscle growth of the legs with concurrent versus resistance exercise training. Even Hickson [3] reported a similar increase in leg circumference with concurrent compared with resistance exercise (albeit leg circumference is a crude measure of muscle size). The experimental evidence for blunted hypertrophy with concurrent exercise is therefore circumstantial and limited.

Numerous human and animal investigations have nevertheless sought to identify the underpinnings of an 'interference effect' on hypertrophy following acute and chronic concurrent exercise (see reviews by Fyfe et al. [7] and Baar [8]). However, study design limitations (animal versus human, unrealistic training model, etc.) in many studies preclude extrapolation of findings to practical exercise training paradigms. Moreover, an acute (minutes to hours) interference effect of concurrent exercise at the cellular level does not necessarily translate to habitual (weeks to months) training responses. Within the last few years, a growing body of literature suggests concurrent exercise does not interfere with resistance exercise-induced hypertrophy. When aerobic and resistance exercise are performed in low volumes and with adequate rest of the targeted muscle groups between bouts (i.e., hours to days), concurrent exercise may actually augment whole muscle growth. This Current Opinion paper presents the contrary evidence for an acute and chronic interference effect of concurrent exercise on skeletal muscle hypertrophy. We also provide practical considerations for applying aerobic and resistance exercise within the same training program.

2 Signaling and Molecular Bases for an Interference Effect with Concurrent Exercise

Building from the pioneering work of Nader and Esser [9], Atherton et al. [10] proposed in 2005 that adenosine monophosphate kinase (AMPK, or the 'energy sensor' of the cell that triggers mitochondrial biogenesis) signaling selectively mediates aerobic exercise adaptations, the mechanistic target of rapamycin (mTOR, member of a major hypertrophy pathway) signaling facilitates resistance exercise adaptations, and that these signals could be in competition. Shortly thereafter, Thomson et al. [11] experimentally showed that AMPK up-regulation indeed interfered with mTOR signaling. In short, the energetic perturbation caused by aerobic exercise favors mitochondrial adaptation and theoretically overrides growth signaling from resistance exercise. While elegant and insightful, these landmark investigations were performed in rodents, utilized acute aerobic- and resistance exercise-like stimuli, and did not include a concurrent exercise analog. A few subsequent investigations in humans suggested inhibition of skeletal muscle growth processes [12, 13] or showed increased protein turnover/breakdown markers at the molecular level with acute concurrent exercise [14–16]. However, most acute concurrent exercise studies have failed to observe interference of growth processes at the transcriptional [16, 17] or protein [14–20] level compared with resistance exercise alone. This is true even when aerobic and resistance exercise is in close temporal proximity (Table 1). A noteworthy recent investigation reported no molecular differences in response to acute concurrent versus resistance exercise, and mTOR activation was amplified with concurrent exercise [16].

Contentious findings for an acute concurrent interference effect on hypertrophy in humans are not surprising since skeletal muscle signaling pathways following exercise are often not discrete in vivo. For example, resistance exercise can up-regulate AMPK [13, 16, 21–25], while aerobic exercise can increase mTOR activity [26–28].

Table 1 Summary of contrary evidence for an acute interference effect on hypertrophic signaling caused by CE in human skeletal muscle

Study	Subjects	Exercise stimulus	Relevant findings (concurrent vs. resistance)
Carrithers et al., 2007 [18]	12 moderately active males and females[a]	RE: 3 × 10 repetitions leg press and extension at 80 % 1RM CE: RE + 90 min of cycling at 60 % wattage at max, 30 min apart	⇔ myofibrillar FSR
Donges et al., 2012 [19]	8 sedentary middle-aged males	RE: 8 × 8 leg extension at 70 % 1RM CE: 4 × 8 leg extension at 70 % 1RM immediately followed by 20 min of cycling at 55 % VO_{2max}	⇔ myofibrillar FSR ⇔ mitochondrial FSR ⇔ Akt or mTOR signaling
Lundberg et al., 2012 [20]	9 healthy moderately active males[a]	RE: 2 × 7 maximal repetitions on an isoinertial leg extension and leg press CE: RE + 40 min single leg cycling at 70 % max wattage, then wattage incrementally increased until exhaustion, 6 h apart	⇑ mTOR and p70S6K signaling ⇔ rpS6 and eEF2 ⇓ myostatin mRNA 1 h
Apro et al., 2013 [17]	10 healthy moderately active males	RE: 10 sets progressive leg press CE: Leg press + 30 min cycling at 70 % VO_{2max}, 15 min apart	⇔ mTOR, S6K1, and eEF2 signaling as well as various molecular growth markers
Fernandez-Gonzalo et al., 2013 [15]	10 healthy moderately active males[a]	RE: 4 × 7 maximal repetitions on an isoinertial leg extension CE: RE + 40 min single leg cycling at 70 % max wattage, then wattage incrementally increased until exhaustion, 6 h apart	⇔ mTOR, rpS6, and eEF2, signaling, p70S6K signaling tended to be ⇑ and myostatin mRNA ⇓ ⇑ MuRF-1 and atrogin mRNA expression
Pugh et al., 2015 [16]	10 healthy males	RE: 4 × 7 repetitions of leg extension at 70 % 1RM CE: RE immediately followed by 10 × 1 min cycling at 90 % HRmax	⇑ mTOR signaling ⇔ eEF2, p70S6K, rpS6, and 4EBP1 signaling ⇑ atrogin mRNA, ⇔ IGF or MGF mRNA
Apro et al., 2015 [14]	8 moderately active males	RE: 10 × 8–10 repetitions leg press at varying intensity CE: 5 × 4 cycling bouts at 85 % VO_{2max} immediately followed by RE	⇔ AMPK and mTOR signaling ⇔ FSR ⇑ MuRF-1 mRNA and protein

All positive/negative results are statistically significant unless otherwise stated. Moderately active means subjects generally participated in weight lifting, aerobic exercise, and/or team sports for recreational purposes but were not competitive athletes

1RM 1 repetition maximum, *Akt* protein kinase B, *AMPK* adenosine monophosphate kinase, *CE* concurrent exercise, *eEF2* eukaryotic elongation factor 2, *FSR* fractional synthetic rate, *HRmax* maximum heart rate, *IGF* insulin-like growth factor, *max* maximum, *MGF* mechanogrowth factor, *mRNA* messenger ribonucleic acid, *mTOR* mechanistic target of rapamycin, *MuRF-1* muscle ring finger protein 1, *p70S6K* p70S6 kinase, *RE* resistance exercise, rpS6 ribosomal protein S6, VO_{2max} maximal aerobic capacity, ⇑ indicates greater,⇓ indicates reduced, ⇔ indicates no difference

[a] Unilateral leg training study design

Acute exercise responses provide insight into adaptive processes [29], but intracellular signaling events following exercise in humans may [30] or may not [15, 31, 32] predict adaptation. Factors such as heredity [33], training status [21, 34–36], and nutrition [37–39] can complicate chronic outcomes. However, the immediate fatigue associated with intense aerobic exercise may affect the quality of a subsequent resistance exercise bout if performed in close temporal proximity [40]. Signaling interference may impact long-term concurrent exercise training results in certain cases, but compromised training quality due to fatigue is also of major concern. Thus, the timing and volume of aerobic and resistance exercise within a concurrent training paradigm are important considerations for maximizing adaptation.

3 Timing of Aerobic and Resistance Exercise within a Concurrent Training Paradigm

Minutes, hours, and/or days of rest between aerobic and resistance exercise can be utilized when designing a concurrent exercise protocol. If an acute interference phenomenon exists in humans, it is predicted that close-succession aerobic and resistance exercise (e.g., ≤30 min apart) would elicit the strongest competing intracellular signals and affect hypertrophic adaptive potential [41]. Interestingly, the human literature does not support this concept. Human training studies employing close-succession concurrent exercise reveal no difference in whole muscle hypertrophy compared with resistance exercise alone [31, 42–44]. Lundberg et al. [44] reported modest but

greater whole-muscle hypertrophy with close-succession concurrent exercise (~40 min of cycling and maximal isoinertial leg extension) compared with resistance exercise over 5 weeks despite greater concurrent exercise-induced AMPK up-regulation. This finding challenges the role of acute AMPK stimulation as a growth-confounding molecular agent in humans.

In alignment with the close-succession aerobic and resistance exercise findings, a number of concurrent exercise investigations with modes separated by hours to days do not report blunted whole muscle growth [45–48]. Moreover, notably greater whole muscle growth was found after 5 weeks of concurrent (bouts separated by 6 h) versus resistance exercise training in isolation (14 vs. 8 %, respectively [$p < 0.05$]) [45]. Whole muscle hypertrophy from combined aerobic and resistance training in this investigation (the highest rate reported in the human exercise literature) could be attributed to a more anabolic cellular milieu [15, 20] and was paradoxically driven by the more frequently activated and less growth-oriented slow-twitch muscle fibers [45]. Similarly, Mikkola et al. [48] reported almost double the quadriceps growth (11 vs. 6 %, $p < 0.05$) following concurrent exercise (~60 min of variable-intensity cycling and progressive resistance, separated by 24 h) compared with resistance exercise alone after 21 weeks of training [48] (Fig. 1).

Dr. Keith Baar [8] recently provided an in-depth overview of the various molecular events that could potentially mediate acute concurrent exercise interference. He submitted that, based mainly on the time course of AMPK recovery (and subsequent mTOR antagonization) as well as sirtuin 1 activity after exercise [49–51], at least 3 h should separate aerobic from resistance exercise when exercising concurrently. However, from a practical and applied standpoint, strength impairments following an endurance exercise bout (high intensity or submaximal continuous)

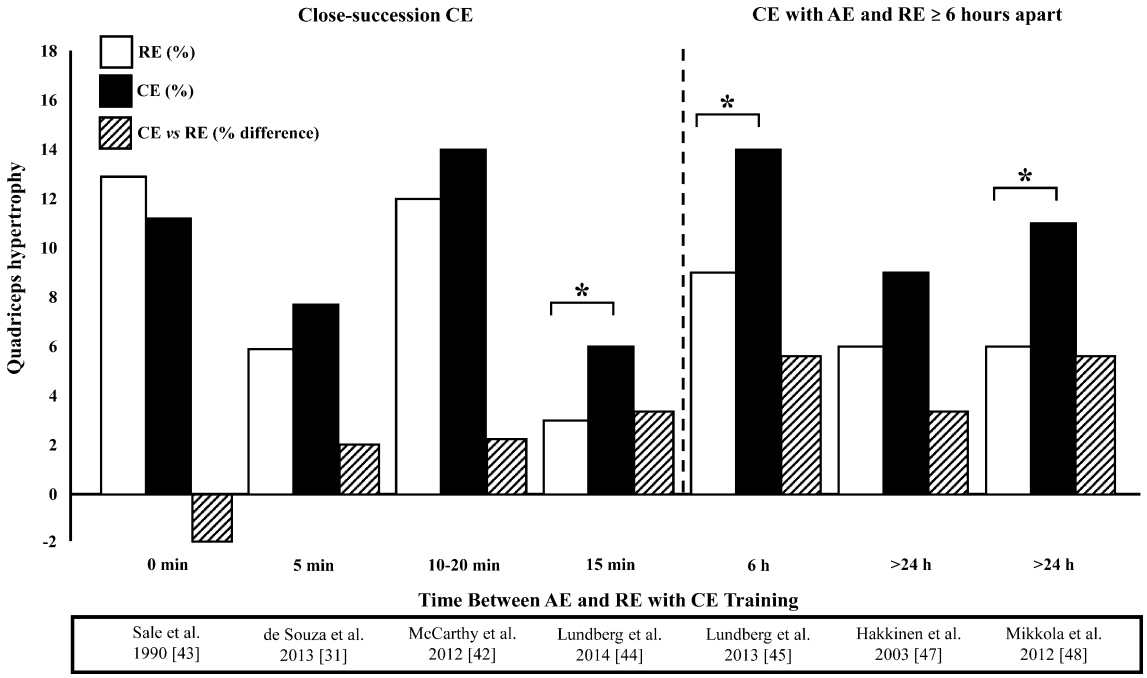

Fig. 1 Equivalent or greater whole muscle hypertrophy with CE vs. RE training, stratified by recovery period between AE and RE. Sale et al. [43]—11-week unilateral leg training, RE: 6 sets × 15–20 leg presses 3 days/week, CE: RE + five 3-min cycling bouts at 90–100 % maximal aerobic capacity 3 days/week; de Souza et al. [31]—8-week training, RE: 6–12 maximal repetitions leg press/knee extension/knee flexion 2 days/week, CE: RE + 20 × 1-min high-intensity run sprints 2 days/week; McCarthy et al. [42]—10-week training, RE: 8 upper/lower body exercises, 3 sets × 5–7 repetitions 3 days/week, CE: RE + 50 min cycling at 70 % heart rate reserve 3 days/week; Lundberg et al. [44]—5-week unilateral leg training, RE: 4 sets × 7 repetitions maximal isoinertial leg extensions 2–3 days/week, CE: RE + ~40 min cycling at ≥70 % wattage at max 2–3 days/week; Lundberg et al. [45]—5-week unilateral leg training, RE: 4 sets × 7 repetitions maximal isoinertial leg extensions 2–3 days/week, CE: RE + ~40 min cycling at ≥70 % wattage at max 2–3 days/week; Hakkinen et al. [47]—21-week training, RE: upper and lower body progressive overload training 2 days/week, CE: RE + 30–90 min variable intensity cycling 2 days/week per mode; Mikkola et al. [48]—21-week training, RE: strength/power focused upper and lower body exercises 2 days/week, CE: RE + 30–90 min variable intensity cycling 2 days/week per mode. Bell et al. [46] reported no difference in growth between CE and RE, but data were not presented. Quadriceps size measured by computed tomography or magnetic resonance imaging for all studies. No sample size was smaller than eight subjects for any group in any study. *AE* aerobic exercise, *CE* concurrent exercise, *RE* resistance exercise, *Asterisks* CE statistically greater than RE

can last for at least 6 h [40, 52–54]. Indeed, Robineau et al. [55] recently reported that mean maximal strength increased in a stepwise fashion as time between aerobic and resistance exercise (0, 6, and 24 h) in the concurrent training program was extended. Thus, to maximize the training response, aerobic and resistance exercise within the concurrent training program should be separated by a minimum of 3 h, but preferably 6–24 h. Allotting this recovery timeframe between modes increases the likelihood of a high-quality resistance exercise stimulus.

4 Volume, Intensity, and Mode Considerations for Concurrent Exercise Training

An important feature of the aforementioned investigations that reported greater hypertrophy with concurrent versus resistance exercise training was the overall exercise volume. Both investigations utilized two to four sets of ≤2 leg resistance exercises performed 2 (or occasionally 3) days per week while aerobic exercise was performed ≤3 days per week for ≤60 min per session. Training was vigorous, but overall exercise duration was relatively low, and frequency did not exceed 4 days per week in either study [45, 48]. Consistent with reports regarding muscle strength and power adaptations [56, 57], minimizing the overall volume of exercise (by manipulating total exercise duration or, perhaps more importantly, frequency) is likely an important consideration for maximizing the hypertrophic potential of concurrent training.

It is well established that short-duration high-intensity interval training induces robust skeletal muscle adaptations that mimic the effects of prolonged endurance training [58–60]. However, high-intensity interval aerobic exercise can increase adenosine triphosphate (ATP) demand 100-fold above rest [61, 62]. If subscribing to the acute AMPK/mTOR interference hypothesis, the significant energetic perturbation from high-intensity aerobic exercise could exacerbate potential interference phenomena, especially if performed in close temporal proximity to resistance exercise [13, 63, 64]. However, no attenuation of whole-muscle hypertrophy has been found with concurrent exercise characterized by high-intensity low-volume interval aerobic exercise performed in close-succession [31, 43] or on separate days from resistance exercise [47]. High-intensity aerobic and resistance exercise both involve forceful contractions that heavily recruit growth-oriented fast-twitch fibers. The similarity in loading pattern could collaboratively support hypertrophy despite local energetic challenges. In keeping with this logic, cycling may also be preferable to running as the aerobic exercise mode based on the resistance-like loading stimulus. Cycling alone can induce leg muscle hypertrophy [42, 43, 65–67]; this may in part account for robust growth observed in recent concurrent exercise investigations [45, 48]. Conversely, the eccentric damage associated with intense running may impede recovery and affect hypertrophic potential of concurrent training (see Wilson et al. [57]). Taking volume, frequency, intensity, and mode considerations together, 2 (or at most 3) days per week of short-duration aerobic training (i.e., 30–40 min per bout) that may include high-intensity interval cycling combined with ≤2 days per week of lifting per day (4–8 sets) is a reasonable recommendation for facilitating maximal hypertrophic adaptations from concurrent exercise training.

5 Nutritional Considerations for Concurrent Exercise Training

The relationship between protein synthesis and breakdown over days and weeks largely determines the magnitude of adaptation, and a positive protein balance can only be achieved with feeding [68, 69]. A recent report shows that, similar to what occurs with resistance exercise [68–70], protein ingestion after acute concurrent exercise has a positive effect on protein synthesis for at least 4 h [71]. Congruent with these findings, Donges et al. [19] evaluated the acute molecular, signaling, and protein synthesis responses to concurrent versus resistance exercise in the fed state and found them to be virtually identical. These acute results suggest that properly managed nutrition (i.e., sufficient overall calorie and protein ingestion around the time of exercise) promotes a favorable growth environment with concurrent exercise that is akin to what is found with resistance exercise. However, the impact of proper nutrition on hypertrophic potential may be even more important with concurrent training given the greater caloric demand associated with adding volume (i.e., an additional mode of exercise) to a training program.

Unique insight into how concurrent versus resistance training affects muscle mass when dietary intake is completely accounted for can be found in the bed rest literature. One investigation involved a concurrent exercise training intervention with complete nutritional oversight in women during prolonged bed rest (60 days) [72]. A previous study involving the same resistance exercise intervention and nutritional oversight in men during 84 days of bed rest serves as a basis for comparison [73]. Prolonged unloading is associated with pronounced skeletal muscle atrophy due to complete inactivity, and resistance exercise is generally the preferred countermeasure [74]. However, high-intensity resistance exercise in men and high-intensity resistance exercise combined with variable intensity aerobic exercise on alternating days in women (≤80 % maximal aerobic capacity for 40 min) similarly protected thigh muscle mass

when strictly adhering to balanced eucaloric diets (~55 % carbohydrate, ~30 % fat, and ~15 % protein) provided by the investigative teams [72, 73]. Admittedly, these studies are not directly comparable and sex differences cannot be precluded. However, the concurrent training findings are compelling since fast-twitch fibers appear to atrophy more during bed rest in women than in men [73, 75]. So long as diet is properly managed, aerobic exercise does not seem to interfere with the effectiveness of resistance training at preserving muscle mass in the face of muscle disuse.

Perez-Schindler et al. [76] recently published recommendations for nutrition with concurrent training based on the independent aerobic and resistance exercise literature. The authors assert that, in addition to consuming 1.2–1.7 g/kg of body weight per day of protein when training concurrently, protein should be consumed with carbohydrate if aerobic exercise is prolonged (≥1.5 h). This may help limit endogenous amino acid usage, expedite glycogen re-synthesis, promote a positive protein balance, and aid in skeletal muscle recovery processes [77, 78]. Following aerobic exercise, the concurrent exerciser should not neglect replenishing glycogen by eating adequate carbohydrate in the hours after exercise (e.g., 1–1.5 g/kg) and should attempt to restore skeletal muscle energy status so that resistance exercise is performed in a fed, pro-anabolic state [69, 79–81]. Separating aerobic and resistance exercise by 6–24 h may help maximize the re-fueling process. Although more research on nutritional interventions with concurrent exercise training is warranted, these aforementioned strategies should help promote optimal long-term adaptation to both aerobic and resistance exercise and facilitate high-quality training.

6 Insight on Concurrent Training from Competitive Endurance Athletes

Most concurrent exercise training studies involve untrained or recreationally active subjects who are generally unaccustomed to consistent or intense exercise. For instance, high-intensity cycling exercise can be modestly hypertrophic in previously untrained but healthy individuals [42, 43, 65, 66], a phenomenon that supports the notion that aerobic exercise (specifically cycling) could supplement hypertrophic adaptation to resistance exercise in the exercise naïve. Conversely, intensified aerobic exercise training in well-conditioned endurance athletes' regimens would likely not result in appreciable or sustained muscle mass increases. Signaling flexibility following unfamiliar acute exercise points to adaptive potential in well-trained endurance and resistance athletes [21]. However, it is posited that muscle of highly trained endurance athletes who resistance train may not hypertrophy to the same degree as untrained individuals who train concurrently [82]. Given the abnormally high duration and frequency of exercise as well as caloric expenditure characteristic of competitive endurance athletes, the hypertrophic potential of concurrent exercise training in this population is worth further consideration.

In strength training-naïve elite cross-country skiers, Losnegard et al. [83] did not report quadriceps growth when 1–2 days per week of half squats were included during 3 months of pre-season endurance training. These athletes utilized very high aerobic training volumes (~60 h per month) that, if performed at a modest 2 l per minute of oxygen consumption, equates to an additional ~9000 calories of energy expenditure per week. It is possible that the half squats were not challenging enough to induce growth, but it is also conceivable that caloric/nutritional deficits due to high volumes of aerobic exercise influenced these findings [83]. Ronnestad et al. [84] subsequently compared muscle growth after 12 weeks of resistance exercise training in untrained individuals and highly trained endurance athletes while quantifying dietary intake. The endurance athletes resistance trained twice per week in conjunction with their routine high-volume aerobic exercise (~10 h per week) while the non-athletes conducted resistance training only. The resistance trained non-athletes had almost twice the whole muscle growth as the concurrently trained endurance athletes (8.0 vs. 4.3 % [$p < 0.05$], respectively), and this response was not attributable to differences in baseline muscle size. However, the athletes had the same total energy, protein, carbohydrate, and fat intake as the untrained individuals during a representative portion of the training period despite similar body mass and significantly greater overall training volume. While the different magnitudes of growth with resistance exercise training could be solely related to training status, it is also possible that nutritional intake was inadequate to support hypertrophy in the endurance athletes while it was sufficient in the non-athletes [84]. Concurrent training outcomes in highly trained endurance athletes provide preliminary evidence that concurrent exercise can be confounded by inadequate compensation of dietary intake.

Another factor that may impede hypertrophic adaptation to concurrent exercise is overtraining due to high training duration and frequency. The etiology of overtraining is not well elucidated, but is common in endurance athletes and characterized by muscle dysfunction and under-performance [85, 86]. A competitive endurance athlete may train six to seven times per week for ≥10 h per week. The addition of intense resistance exercise to such demanding training could conceivably elicit a negative effect on recovery from aerobic exercise and cause or exacerbate

overtraining, thereby influencing adaptive ability. Consistent with this hypothesis, Izquierdo-Gabarren et al. [87] reported that low-volume resistance exercise (392 contractions over 6 weeks at 75–92 % 1 repetition maximum [RM] not to fatigue) conferred greater strength and power benefits than higher-volume resistance exercise (784 contractions to fatigue) in well-conditioned rowing athletes during competition training. These data align with muscle growth findings of previously mentioned studies that utilized modest resistance exercise durations and frequencies to induce greater growth with concurrent versus resistance training in normal healthy individuals [45, 48]. The information derived from high-level endurance athletes that concurrently train supports the recommendations for regular exercisers and recreational athletes. Ensuring adequate dietary intake and not overtraining (i.e., reasonable training durations and incorporating rest days) is important for maximizing hypertrophic adaptations to concurrent training.

7 Muscle Strength and Power with Concurrent Exercise Training

Skeletal muscle size and strength are highly correlated [88, 89], and hypertrophy largely explains the increase in strength following resistance training (after neural adaptations have occurred) [90, 91]. Muscle strength is also an important contributor to whole muscle power since power is the product of force and velocity. Muscle mass increases from concurrent exercise should therefore parallel whole muscle force- and power-producing capacity. However, muscle strength and power improvements are not always commensurate with hypertrophy from concurrent training [45, 57]. The evidence for any possible explanation of this disparity is limited, but differential muscle architectural adaptations [92, 93] or maximal neuromuscular activation [42, 47] do not seem to play a role. Greater tendon thickness following resistance versus concurrent training may contribute to relative functional deficits with concurrent exercise, but the evidence is limited [94]. Lundberg and colleagues [44, 45] suggested that non-contractile volume expanded more than myofibrillar volume with 5-week concurrent versus resistance exercise training. The ultimate result was strength and power adaptations with concurrent training that did not comport with the magnitude of hypertrophy. Interestingly, strength decrements have not been reported after longer durations of concurrent training, meaning this phenomenon could characterize the early adaptive response to concurrent exercise [31, 42, 95]. However, compromised rate of force development [47, 48, 84] as well as force at high velocities [96], further underscores diminished maximal power production with concurrent training [57], which could be of particular concern for competitive athletes.

The recommendation for curtailing muscle function deficits with concurrent exercise is fundamentally similar to the strategy for optimizing hypertrophy. Minimizing aerobic exercise volume (i.e., utilizing high-intensity intervals with $\geq$6 h of rest between modes or only aerobic training twice per week) [97], resistance exercise volume (i.e., limiting the total number of leg exercises and/or the number of reps/sets) [56, 87], and concurrent exercise volume in general ($\leq$3 days per week if aerobic and resistance exercise are in close succession) [42, 43, 98–101] seems best for preserving muscle strength and power. Although limited, data from power-oriented team sport athletes point to the same conclusion: focusing on high-velocity movements within the aerobic mode (i.e., sprint training) is more beneficial for power production than lower-intensity endurance training (i.e., $\sim$45 min of submaximal exercise) [102].

8 Future Directions

From an applied perspective, future in vivo human research on concurrent exercise training may focus on exploring the most appropriate nutritional strategies to support hypertrophic adaptations with concurrent training. Mode of aerobic exercise (e.g., cycling, running, rowing, etc.) most compatible with resistance exercise is also worth further consideration. To this point, a recent investigation that employed concurrent training and is consistent with the strategies outlined in this Current Opinion but used rowing as the aerobic exercise mode reported among the highest quadriceps hypertrophy rate found in the human literature (2 % per week over five weeks) [103]. More comprehensive concurrent training investigations should also be carried out in competitive endurance athletes, who represent a natural experimental model of extreme endurance exercise duration and frequency. If attenuated hypertrophy can occur with concurrent training, it may be most apparent in this population. Mechanistically, further empirical evidence is needed to corroborate the existence of the previously proposed molecular or signaling 'interference effect' of concurrent exercise in human skeletal muscle and whether it could inhibit hypertrophy. Namely, studies in human skeletal muscle cell culture involving AMPK up-regulation and mTOR signaling and perhaps in vitro work using human biopsies would be insightful. Concurrent training studies in rodents (specifically rats) that can be subjected to translatable analogues of resistance and endurance exercise as well as tolerate pharmacological manipulation of AMPK are also warranted.

9 Summary and Conclusions

The experimental evidence for aerobic exercise training interfering with the hypertrophic potential of resistance exercise training in humans is limited at the cellular level and non-existent at the whole-muscle level (regardless of how training is conducted). Based on the literature, we conclude that aerobic exercise training may facilitate resistance-training mediated hypertrophy if (1) exercise bouts are separated by 6–24 h, (2) concurrent exercise is performed using strategies that minimize overall exercise volume (i.e., utilizing high-intensity intervals, 2–3 days of aerobic exercise, $\leq$2 days of leg lifting), and (3) the mode of aerobic exercise favors cycling as opposed to running. Additional research is warranted on the roles of nutrition and exercise mode in optimizing hypertrophic adaptation to concurrent training. Future mechanistic work may involve human muscle cell culture and practical animal models of concurrent exercise training.

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Compliance with Ethical Standards

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