



**NATIONAL AND KAPODISTRIAN UNIVERSITY OF ATHENS**  
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**THESIS**

**“THE CONCURRENT TRAINING EFFECT”**

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## THE CONCURRENT TRAINING EFFECT

### Abstract

Concurrent training (CT) is defined as simultaneously incorporating both resistance and endurance exercise within a periodized training system. Despite the potential additive benefits of combining these divergent exercise modes with regards to disease prevention and athletic performance, there is evidence that this approach may attenuate gains in muscle mass, strength, and power compared with undertaking resistance training alone. This has been described as the interference effect or concurrent training effect (CTE). In recent years, understanding of the molecular mechanisms mediating training adaptation in skeletal muscle has emerged and provided potential mechanistic insight into the concurrent training effect. Although it appears that various molecular signaling responses induced in skeletal muscle by endurance exercise can inhibit pathways regulating protein synthesis and stimulate protein breakdown, human studies to date have not observed such molecular ‘interference’ following acute concurrent exercise that might explain compromised muscle hypertrophy following concurrent training. High volume, moderate, continuous and frequent endurance training, are thought to negatively affect the resistance training-induced adaptations, probably by inhibition of the Protein kinase B—mammalian target of rapamycin (mTOR) pathway activation. Endurance training (ET) activates signaling cascade that regulates metabolic process and mitochondrial biogenesis that comprises adenosine-monophosphate-activated protein kinase (AMPK). AMPK activated by ET is shown to inhibit mTORC1 signaling cascade through tuberous sclerosis complex (TSC) blunting ST-induced protein synthesis. In contrast, it seems that short bouts of high-intensity interval training (HIIT) or sprint interval training (SIT) minimize the negative effects of concurrent training. The nature of the interference effect can be determined by the manipulation of training variables as well as non-training ones.

**Key Words:** concurrent training, strength training, endurance training, adenosine monophosphate-activated protein kinase (AMPK), mammalian target of rapamycin (AKT-mTOR).

**List of Abbreviations**

AMP	adenosine monophosphate
AMPK	adenosine monophosphate-dependent protein kinase
ATP	adenosine triphosphate
Ca <sup>2+</sup>	calcium ion
CaMK	Ca <sup>2+</sup> /calmodulin-dependent protein kinase
CT	concurrent training (CTE – concurrent training effect)
HIIT	high-intensity interval training
LDL	low-density lipoprotein
mTOR(C1C2)	mammalian or mechanistic target of rapamycin (complex 1 and 2)
PGC-1 $\alpha$	peroxisome proliferator-activated receptor- $\gamma$ coactivator-1 $\alpha$
PI3K	phosphoinositide 3-kinases
raptor	regulatory associated protein of mTOR
SERCA	sarcoendoplasmic reticulum calcium ATPase
SIT	sprint interval training
TSC1/2	tuberous sclerosis complex (1/2)
VO <sub>2</sub>	volume of oxygen
VO <sub>2</sub> max	maximal oxygen consumption

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## ***I. Introduction***

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The majority of sports are neither “endurance-based” nor “strength–power-based”, but of a mixed type, with the performance to be determined by the specific contribution of muscle strength/power and endurance, which varies between sports. The inclusion of resistance training (to gain strength, power and muscle mass) combined with aerobic exercise (to enhance endurance) in a single program is known as concurrent training (Wilson et al., 2012). The practice of concurrent training, or simultaneous training of strength and endurance, was first described in 1980 by Robert C. Hickson.

Many studies have investigated the effect of CT programs on both resistance and endurance training adaptations, with many controversial results. Most of them provide evidence that after CT intervention muscle hypertrophy, strength and power adaptations were mostly attenuated, compared with those after isolated strength training stimuli. By contrast, there are several studies providing strong evidence that resistance training adaptations are not suppressed, but further increased after CT. Eventually, it seems that CT interventions do not negatively affect the endurance training adaptations. Furthermore, it seems that after CT, athletes’ endurance capacity is increased to a greater extent compared to when endurance training is performed alone (Berryman et al., 2018). (Methenitis, 2018)

Concurrent training is a matter of great importance not only to sports scientists but also to the coaches as they have to be careful about the training plans/strategies which they are willing to follow, when a combination of endurance and resistance training is needed, by taking into consideration how different training modalities interact between them. Therefore, they can adapt and optimize training methods, thus maximize their athletes’ performance in sports.

The present thesis is a brief review on the concurrent training effect. The results of concurrent training on the development of resistance and endurance training adaptations are discussed as well as the molecular responses that regulate them. Moreover, information is provided about the methodological considerations and the multiple benefits of concurrent training.

## ***II. Literature review***

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### **2.1. Resistance vs. Endurance Training**

Exercise training may contain aerobic/endurance and/or strength/resistance components. Endurance exercise is classically performed at low to moderate intensities over a long duration, whereas strength exercise is performed against relatively high loads for a short duration. However, pure endurance and pure strength exercise is rare (Hughes et al., 2018).

Classic endurance training is known to result in enhanced cardiac output, maximal oxygen consumption and mitochondrial biogenesis. The overall improvement in both central and peripheral tissues allows enhanced exercise economy and a greater ability for an individual to run for longer distances and times. In contrast, strength training results in increases in muscle size, neural adaptations, and improved strength (maximal force production). (Hughes et al., 2018).

Strength training leads to an increase in muscle strength and power as a result of neuromuscular adaptations, increases in muscle, and alterations in connective tissue stiffness (Knuttgen & Kraemer, 1987). The hallmark adaptation to resistance training is an increase in maximal strength, which is underpinned by both neural and morphological adaptations. Typically, neural adaptations are considered predominantly responsible for the rapid increases in strength observed during the early phases of a resistance training program prior to notable changes in muscle mass. As training continues, further improvements in strength are associated with muscle hypertrophy. Muscle hypertrophy appears to preferentially occur in type II fibre, however, type I fibre hypertrophy has been observed, albeit to a lesser degree. In addition to fibre area, resistance training may also affect the proportion of fibre types, with evidence of transitions from type IIx to IIa (Lee et al., 2019). Adaptations to resistance training also include increased protein synthesis via regulatory changes in transcriptional and translational mechanisms, and in the proliferation of muscle cells that are added to existing



myofibres or combine and form new contractile filaments, each providing additional contractile machinery with which to generate force (Coffey, 2006).

Endurance exercise training results in profound adaptations of the cardiorespiratory system that enhance the delivery of oxygen from the atmosphere to the mitochondria and enable a tighter regulation of muscle metabolism. These adaptations effect an improvement in endurance performance that is manifested as a rightward shift in the 'velocity-time curve'. This shift enables athletes to exercise for longer at a given absolute exercise intensity, or to exercise at a higher exercise intensity for a given duration (Jones & Carter, 2000). Endurance exercise is characterized by submaximal, high-frequency contractions, which must be maintained for prolonged durations for successful endurance performance. Regular endurance training is associated with central and peripheral adaptations that facilitate greater oxygen delivery and extraction culminating in improvements in whole-body aerobic power and fatigue resistance. One of the hallmark adaptations of endurance training is an increased content and function of the mitochondrial reticulum, via mitochondrial biogenesis (Lee et al., 2019). Repeated bouts of endurance exercise results in altered expression of a multiplicity of gene products, resulting in an altered muscle phenotype with improved resistance to fatigue (Irrcher et al., 2003).

The combination of both resistance and endurance exercise in a training program leads to superior adaptations in health-related and body function variables, independent of age or sex, including increases and/or improvements of basal metabolic rates, insulin sensitivity, glucose/lipids metabolism, lipidemic profile and body composition, while both muscular hypertrophy/strength/power and endurance capacities are increased (Methenitis, 2018). Clearly, both exercise modes have the capacity to improve several aspects of health and wellbeing, and benefit performance in a range of sporting events, thus concurrently training for both modes offers an ideal strategy to maximize adaptations that are considered to sit at opposing ends of the spectrum (Fyfe et al., 2014; Nader, 2006).

## 2.2. Interference effect

The nature of the interference effect may largely be dictated by the manipulation of training variables (e.g., exercise order, intensity, frequency, volume, mode, recovery duration) and non-training variables (e.g., training status, nutrient availability) (Lee et al., 2019). The factors suggested to underpin the interference effect can be classified as ‘acute’ and ‘chronic’, whereby the former relates to the effects of an endurance session on subsequent resistance exercise performance, and the latter concerns the different adaptive demands of long-term endurance and resistance training (Lee et al., 2019).

Several explanations have been offered to explain the interference effects seen. One of the more popular theories is the chronic interference hypothesis, which postulates that the addition of endurance training results in overreaching and overtraining and stimulates competing adaptations over a long-term training program (Leveritt et al., 1999). Overreaching is currently thought to be caused by high-volume, high-intensity, or high-frequency training bouts, particularly when bouts of exercise result in large amounts of skeletal muscle damage (Halson & Jeukendrup, 2004). It is likely that elements of endurance training, which lead to overreaching, would in theory result in greater interference effects (Wilson et al., 2012).

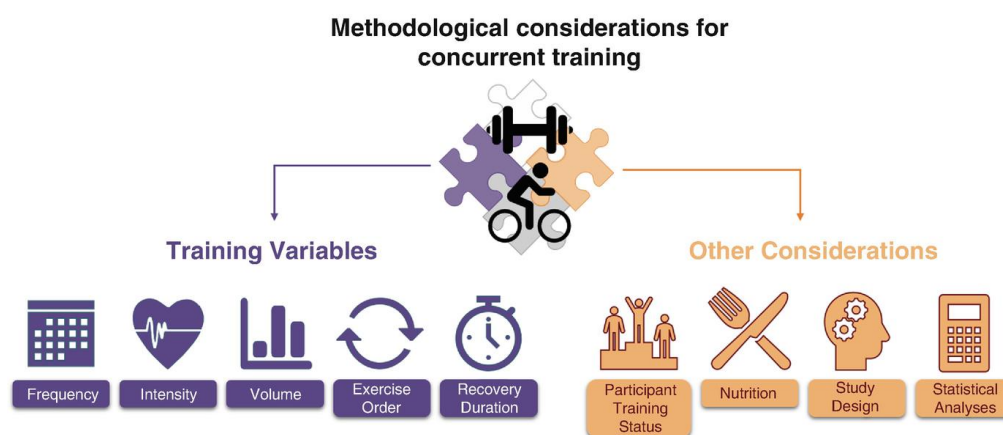
It was observed that both aerobic and resistance exercises interfere in their responses when performed together (Apró et al., 2013). Muscle growth was not inhibited by subsequent endurance exercise. Similarly, it has been found that the rate of strength development is similar for the CT group and strength training group for the first 7 weeks of training (Hickson, 1980). A positive interference effect was seen with CT, i.e., gain in lower body muscle strength was higher than for the strength training group alone (Hickson, 1980). Secondly, a combination of resistance training with high-intensity endurance training led to increases in strength and explosive strength and power, but not to the same extent (Chtara et al., 2008) (Jha et al., 2018). It has been evaluated that when volume and/or frequency of exercises are high in CT, it may adversely affect the gains observed during strength and power training, thus favoring positive interference phenomena (Kraemer et al., 2004). Several studies have supported that a low

volume of CT produced similar muscle strength and hypertrophy increments when compared to low-volume strength training alone and does not support an interference effect (Davis et al., 2008; Glowacki et al., 2004; Libardi et al., 2012; Silva et al., 2012). Additionally, (Wilson et al., 2012) said that the interference effects of endurance training with strength training outcomes are body part specific and not systemic, because primarily lower-body modalities do not interfere with upper-body strength training outcomes.

In conclusion, it is undeniable that resistance and endurance training interfere and sometimes can have negative effects. However, in a suitably designed training program and after taking into consideration the parameters discussed below as well as the individuality of each athlete, concurrent training is proved to have even greater results than when the two different modes are performed alone.

### 2.3. Methodological considerations

The presence and magnitude of the interference effect is likely dependent upon several methodological factors pertaining to the training program and study design. These include training variables such as the choice of exercise session order (i.e., endurance prior to, or after resistance exercise), between-session recovery duration, training frequency, volume, intensity, and exercise modality, plus other ‘non-training’ variables such as the participant training status, nutrient availability, and individual responses to training (Lee et al., 2019).



**Figure 1.** Methodological considerations for concurrent training. A schematic highlighting the key training and ‘non-training’ variables that dictate the potential presence and magnitude of the interference effect. Produced by the author from Bishop et al., (2019a).

Concurrent training offers a time-efficient alternative to single-mode training, particularly if both modes are performed within the same session or separated by short recovery periods. The ‘acute’ interference hypothesis suggests that one exercise session may induce residual fatigue and substrate depletion, hindering the quality and performance of a subsequent bout, and may induce unfavorable neuromuscular, hormonal, and molecular milieus for adaptation (Fyfe et al., 2014).

*Exercise session.* Greater reductions in acute strength performance (i.e., the ability to maintain a required load or volume) have been observed when resistance exercise was conducted immediately ( $\leq 10$  minutes) after both steady-state (Jones et al., 2017) and high-intensity intermittent running (Inoue et al., 2016) compared with the reverse order. As such, a reduction in the resistance training stimulus, such as the volume, may compromise the potential for adaptation. The findings indicate some degree of exercise order-dependent adaptations, thus prioritizing the exercise order according to the primary goals of training may help to maximize the quality of the exercise session and the stimulus for adaptation.

Indeed, two recent meta-analyses concluded that for same-session concurrent training (<15 minutes of between-mode recovery) performing resistance exercise before endurance exercise has a greater effect on maximal dynamic strength compared to the reverse order. However, no order effect was evident for changes in aerobic capacity, nor static strength, hypertrophy, or body fat percentage (Eddens et al., 2018; Murlasits et al., 2018). It may be concluded that when the aim of the training is to maximize adaptations of muscle mass—strength—power, as well as to improve body composition, resistance exercises should be performed prior to endurance exercises, and vice versa when increases in endurance capacity are necessary or when the resistance training adaptations are of lower importance (Leveritt et al., 1999).

*Between-Mode Recovery Length.* The length of recovery allowed between undertaking concurrent exercise sessions is another important practical consideration. Potentially, residual fatigue and/or substrate (i.e., muscle glycogen) depletion from endurance training bouts may impact negatively upon force/power production and anabolic signaling responses to subsequent resistance exercise, respectively. For example, following a bout of endurance exercise, force production of the exercised musculature is reduced for at least 6 h, returning to baseline by 24h post-exercise. Allowing adequate recovery between concurrent exercise sessions may therefore attenuate any negative residual effects from endurance exercise on subsequent training bouts, consequently alleviating any interference (Fyfe et al., 2014).

*Endurance Training Intensity.* Another important practical consideration is the intensity of endurance training employed in a concurrent training regimen. High-intensity interval training

(HIIT) has emerged as a potent exercise strategy for inducing signaling related to mitochondrial biogenesis and health benefits (e.g., improved insulin sensitivity) typically associated with longer-duration, lower-intensity endurance exercise. HIIT represents a time-efficient strategy for promoting mitochondrial biogenesis and associated improvements in oxidative capacity and metabolic health (Fyfe et al., 2014). It seems that low volume, maximum and supramaximal HIIT/SIT ( $\geq 85\%$  of  $\text{VO}_2\text{max}$  velocity or peak power) endurance training, during CT, did not result in decrements of strength, power and muscle hypertrophy adaptations, and in many cases they induce greater resistance training adaptations, while significantly increasing endurance capacity and performance, compared with high-volume low-moderate intensity endurance training (Wilson et al., 2012). Thus, inclusion of high intensity (maximal or supramaximal;  $\geq 100\%$  of  $v\text{VO}_2\text{max}$  or  $W_{\text{peak}}$ ), low-volume ( $< 20\text{min}$ ) HIIT and SIT endurance training seems to minimize the negative CTE (Methenitis, 2018).

*Endurance Training Volume and Frequency.* Greater attenuation of strength and hypertrophy (estimated via limb girth) has been shown to occur with greater frequencies of concurrent endurance exercise (3 days per week for each mode) than when endurance exercise was performed once per week (Jones et al., 2013). Nevertheless, it remains to be determined whether the total weekly endurance training volume, or the training frequency, is the more critical factor mediating concurrent interference (Fyfe et al., 2014). It has been suggested that a training frequency ratio between 2:1 and 3:1 (frequency of resistance training per week: frequency of endurance training per week), leads to higher resistance training-induced adaptations, compared with when a ratio of 1:1 was performed (Fisher et al., 2013).

*Endurance Training Modality.* The endurance training modality employed in a concurrent training regime may also modulate interference following long-term concurrent training. Interestingly, the majority of concurrent training studies reporting an interference effect have incorporated running, and less often cycling, as the endurance training modality (Wilson et al., 2012). Nevertheless, it seems that when cycling is used, compared with running, there are even lower or no negative results on resistance exercise-induced adaptations, especially on strength and power performances, indicating that cycling is superior to running (Gergley, 2009).

To conclude, consideration must be given to both individual and collective roles of other training variables, as the interference effect is unlikely attributable to the manipulation of one training variable alone; a change to one may alter the effect of another. Other factors inherent to training programs and research study designs also moderate the training effects and observed outcomes. Such ‘non-training’ variables include the participant training status and nutrient availability, as well as other factors such as the sample size, individual responses, and choice of statistical analyses (Lee et al., 2019).

#### **2.4. Molecular Adaptations**

According to recent studies, it appears that CTE is mostly affected by the interference of molecular pathways underlying adaptations from each type of training segment (Coffey & Hawley, 2017; Hawley, 2009; Shamim et al., 2018), and the extent of muscle damage (Lee et al., 2019; Wilson et al., 2012)

The mechanistic (or mammalian) target of rapamycin (mTOR) is a central mediator of protein synthesis in skeletal muscle (Song et al., 2017). The mTOR is a highly conserved serine/threonine protein kinase that forms part of a multicomponent protein complex that occurs as two distinct variants. Each variant is composed of different accessory proteins, which dictate their respective structure, function, and location (Hall, 2008). Both complexes differ structurally and functionally; mTORC1 plays a central role in regulating translation and cell growth, whilst mTORC2 regulates actin cytoskeleton organization, cell proliferation and survival, and targets different proteins from mTORC1.

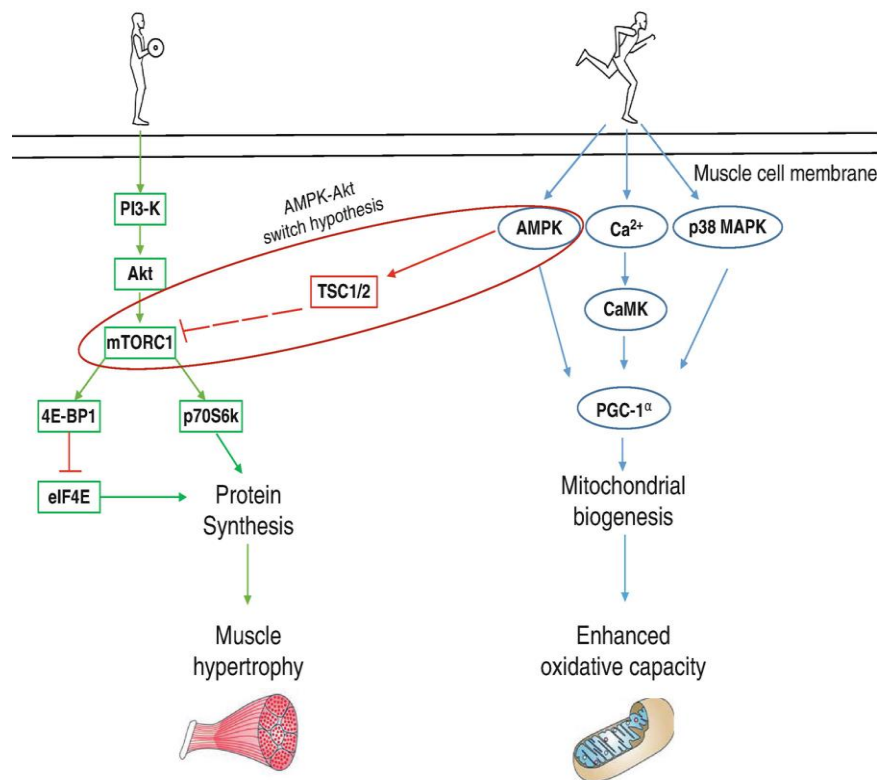
Adenosine monophosphate-activated protein kinase (AMPK) is a principal intracellular energy sensor which activates energy producing pathways and inactivates energy-requiring pathways when the cellular AMP/ATP ratio increases. (Pantovic et al., 2013) AMPK has been linked to control of gene expression by activating transcription factors associated with mitochondrial

fatty acid oxidation and the inhibition of protein synthesis by down-regulating components of the insulin / insulin-like growth factor signaling pathway (Coffey, 2006).

The PI3K-AKT-mTOR pathway mediates hypertrophy in skeletal muscle via activation of translation initiation and increased ribosomal protein content. A significant inhibitor of AKT-mTOR pathway activation, and thus of muscle hypertrophy, is AMPK (Hawley et al., 2014), which is a key energy sensor of the cells, as well as a significant regulator of mitochondrial protein synthesis–biogenesis. This probably occurs by the activation of its downstream target, peroxisome proliferator-activated receptor- $\gamma$  coactivator (PGC-1a) (Coffey & Hawley, 2017; Hawley, 2009; Shamim et al., 2018). Low energy availability, increased energy deficit and/or an increase of Adenosine monophosphate/ triphosphate (AMP/ATP) ratio in the cells, results in AMPK phosphorylation, and thus, in increased activation of tuberous sclerosis complex 1/2 (TSC1/2) which in turn causes the inhibition of mTOR signaling, probably by the de-phosphorylation of Raptor-mTOR. Increases in AMPK phosphorylation, during and after a continuous endurance training seems to have a dose–response relationship with the endurance training load/volume and/or intensity, e.g., higher loads, longer durations and higher intensities resulting in higher increases of AMP/ATP ratio and thus higher activation of AMPK (Coffey & Hawley, 2017; Fisher et al., 2013; Fyfe et al., 2014; Silva, 2012).

Endurance training stimulates molecular pathways [like those of PGC-1, Ca<sup>2+</sup>/calmodulin-dependent kinases (CaMK), calcineurin, adenosine monophosphate-activated protein kinase (AMPK) and mitogen-activated protein kinases (ERK1/2, p38 MAPK)] underlying the cellular processes that promote mitochondrial protein synthesis–biogenesis and angiogenesis, and thus providing metabolic adaptations leading to an increase in endurance capacity. In contrast, resistance training promotes mostly the increase of muscle hypertrophy, strength and power via the AKT-mTOR R (mammalian target of rapamycin) pathway that stimulates myofibrillar protein synthesis (Hawley et al., 2014).





**Figure 2.** Schematic diagram summarizing signaling pathways activated by strength (ST) and endurance training (ET) and the AMPK-Akt switch hypothesis. ST induces an increase in the activity of protein kinase B (Akt), mammalian target of rapamycin complex 1 (mTORC1), that modulates rates of protein synthesis through phosphorylation of eukaryotic initiation factor 4E-binding protein (4E-BP1) that promotes dissociation between 4E-BP1 and eukaryotic initiation factor 4E- (eIF4E) and activation of 70 kDa ribosomal protein kinase (p70S6K). ET activates signaling cascade that regulates metabolic process and mitochondrial biogenesis that comprises adenosine-monophosphate-activated protein kinase (AMPK), p38 mitogen-activated protein kinase (MAPK), calmodulin-dependent protein kinase (CaMK) and proliferator-activated receptor gamma coactivator 1-alpha (PGC-1 $\alpha$ ). In the Akt-AMPK switch hypothesis model, AMPK activated by ET may inhibit mTORC1 signaling cascade through tuberous sclerosis complex (TSC) blunting ST-induced protein synthesis. Figure was adapted from: Hawley, JA. (2009) Molecular adaptations to strength and endurance: Are they incompatible? Atherton et al. (2005). Selective activation of AMPK-PGC-1 or PKB-TSC2-mTOR signaling can explain specific adaptive responses to endurance or resistance training-like electrical muscle stimulation. From Coffey and Hawley (2017)

## 2.5. Concurrent training benefits

### Health

Both aerobic exercise training at either high or moderate intensities and high-intensity strength training improve endothelial function and decrease the cardiovascular risk profile in obese adults. However, high-intensity aerobic interval training results in a greater improvement in endothelial function and a decrease in the cardiovascular risk profile than moderate-intensity aerobic training or strength training in conjunction with improved antioxidant status and decreased levels of oxidized LDL. Moreover, enhanced VO<sub>2</sub>max after strength training and high-intensity aerobic interval training was associated with higher expression of PGC1 $\alpha$  and improved SERCA activity in the skeletal muscle (Schjerve et al., 2008). Also, it might be possible to reverse impaired endothelial function, decrease cardiovascular risk and improve exercise capacity in those that have difficulty performing whole-body aerobic training. CT is also shown to improve the cardiometabolic risk factors and reduce the likelihood of developing metabolic syndrome. That happens because higher levels of muscular strength are associated with a lower cardiometabolic risk, and with reductions in abdominal body fat, plasma concentration of triglycerides and HDL-cholesterol, blood pressure, or glycemia (Álvarez et al., 2019).

### Performance

The combination of cardiovascular and neuromuscular exercise stress within the same training program, and occasionally within the same training session, appears to be a compulsory path to achieve high performance in many sports. The overall improvement in both central and peripheral tissues allows for enhanced exercise economy and a greater ability for an individual to run for longer distances and times (Brooks, 2011). In contrast, strength training results in increases in muscle size, neural adaptations (motor output), and improved strength (maximal force production). These positive alterations in physical capacity allow an individual to be stronger, more powerful, and thus have a greater performance. (Sedano et al., 2013) found that

CT, including both explosive and endurance strength training, led to improved maximal strength, running economy, and peak velocity with no significant effects on the VO<sub>2</sub> kinetics pattern. It is also found that CT results in improvement in performance of vertical jump height, ball throwing speed, and standing long jump (Marta et al., 2013). In addition, positive results were also shown for agility, flexibility, muscular endurance and strength in adolescents (Arazi et al., 2011).

It may be concluded that CT should be an integral component of well-trained athletes' practice regimen because of its potential to improve the performance with respect to the individualization as a crucial factor for designing their training protocol.

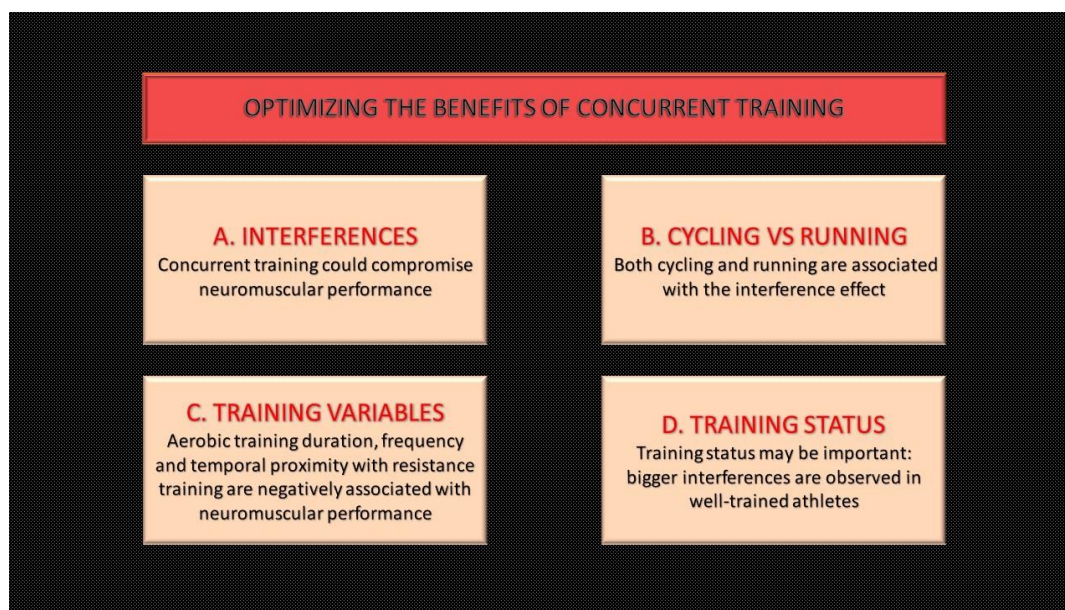


### ***III. Discussion***

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#### **Conclusions**

CTE is a multidimensional phenomenon, affected by various physiological and other factors (such as the exercise characteristics, training background, genetics, muscle groups that are trained, inter-individual variations and others). Concurrently performing endurance and resistance exercise within the same training program presents a theoretically optimal training method for improving athletic performance, as well as attaining the multiple health benefits from both modes of training. It is evident that under certain conditions (discussed above), the concurrent performance of endurance and resistance training may interfere with the adaptive potential of hallmark resistance training adaptations such as strength, muscle hypertrophy, and power. The precise causes of the interference effect remain a contentious area of research, but are likely the product of both ‘acute’ and ‘chronic’ factors, such as residual fatigue, potential incompatibility between the molecular pathways induced by endurance and resistance exercise respectively, and the distinct structural and functional adaptations to both modes.

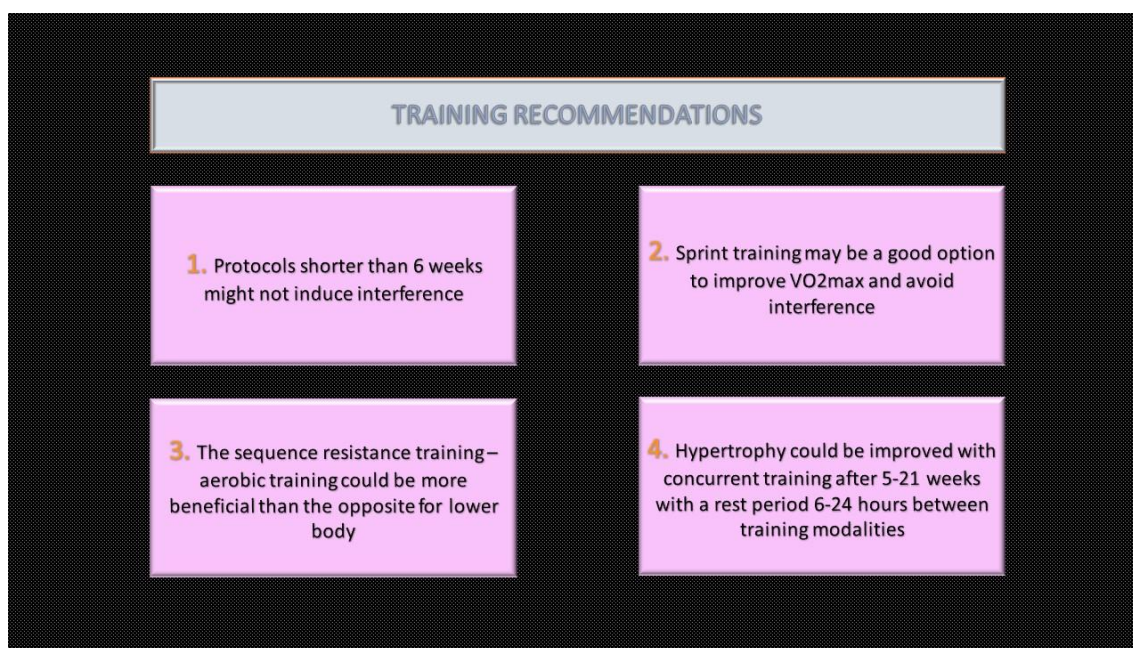


***Figure 3. Optimizing the benefits of CT. Adapted by Berryman, Mujika & Bosquet I (2018)***

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## Limitations - Recommendations

The reasons for the differential responses/adaptations between participants after CT have not been fully investigated. Therefore, there are significant questions about the molecular events underlying the CTE that remain unanswered. Furthermore, future studies should look to adopt an integrated approach, combining assessments of acute molecular responses with measures of protein synthesis, as well as multiple measures of hypertrophy (assessing whole-body, muscle- and fibre-specific changes mass, volume and area), taken over a longer training period, to provide greater insight into the temporal relationship between exercise-induced responses and skeletal muscle adaptations to concurrent training. Research has to be longitudinal, with the training interventions to exceed three months, and should most probably aim to answer questions like the meaning of the acute molecular events that are present after the initial training session for the training-induced adaptations as well as the critical time-point of a training intervention after which the CTE is stronger. Lastly, it would also be interesting to examine nutrient strategies that may affect the CT responses by replenishing and/or depleting muscle glycogen.



**Figure 4.** *Training Recommendations. Adapted by Berryman, Mujika & Bosquet (2018)*

## **Practical Applications**

The level of fatigue from both modules and the need of inter-stimulus or inter-session time intervals to minimize the training induced overall fatigue. The training volume of each training regimen of a CT must be considered, in an effort to minimize muscle fatigue and energy expenditure. Furthermore, it is suggested to incorporate low-volume, high-intensity (maximum and supra-maximum) HIIT or SIT endurance exercises, in order to keep low the activation of AMPK. Also, it's beneficial to separate training bouts by 3–6 to 24 h, even if this is not always practical to the “real” world of athletes’ training. When the goal is to maximize the resistance training adaptations on muscle mass—strength—power, as well as to improve body composition, resistance exercises should be performed prior to endurance exercises. On the contrary, when the goal of the training is to increase the endurance capacity or when the resistance training adaptations are of lower importance, then endurance exercises should be performed prior to resistance exercises. Finally, attention must be given to frequency of each training stimulus. If resistance training-induced adaptations are of importance, consider using a ratio of 2:1 or 3:1 between resistance training sessions per week: endurance training sessions per week. In contrast, it seems that a ratio of 1:1 or 1:2 leads to a better improvement of endurance capacity. Training experience and background are of high importance for the CTE, which is stronger for experienced participants, while in novice or recreational individuals it is lower. Thus, training plans aiming to maximize performance in well-trained individuals or athletes, through a CT intervention, should be designed very carefully, based on the specific requirement of each sport as well as on the evidence-based suggestions (Methenitis, 2018).

Careful consideration of the individual and collective roles of all training and non-training variables is required by anyone engaging in concurrent training to achieve the desired adaptations.

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