

A Proposed Model for Examining the Interference Phenomenon between Concurrent Aerobic and Strength Training

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Abstract

A review of the current research on the interference phenomenon between concurrent aerobic and strength training indicates modest support for the model proposed in this article. However, it is clear that without a systematic approach to the investigation of the phenomenon there is lack of control and manipulation of the independent variables, which makes it difficult to test the validity of the model. To enhance the understanding of the interference phenomenon, it is important that researchers are precise and deliberate in their choice of training protocols.

Clear definition of the specific training objectives for strength (muscle hypertrophy or neural adaptation) and aerobic power (maximal aerobic power or anaerobic threshold) are required. In addition, researchers should equate training volumes as much as possible for all groups. Care needs to be exercised to avoid overtraining individuals. There should be adequate recovery and regeneration between the concurrent training sessions as well as during the training cycle. The model should be initially tested by maintaining the same protocols throughout the duration of the study. However, it is becoming common practice to use a periodised approach in a training mesocycle in which there is a shift from high volume and moderate intensity training to lower volume and higher intensity. The model should be evaluated in the context of a periodised mesocycle provided the investigators are sensitive to the potential impact of the loading parameters on the interference phenomenon. It may be that the periodised approach is one way of maintaining the training stimulus and minimising the amount of interference.

The effects of gender, training status, duration and frequency of training, and the mode of training need to be regarded as potential factors effecting the training response when investigating the interference phenomenon. Other experimental design factors such as unilateral limb training or training the upper body for one attribute and the lower body for another attribute, may help establish the validity of the model.

Many sports require athletes to develop high levels of several physical and fitness attributes to compete at the elite level. Ideally, athletes use a

'periodised' approach to training for optimal long term development which allows them to sequentially develop the fitness requirements for their

sport over a period of time.^[1] However, because of lack of time and the demands of competitive schedules, a sequential, periodised approach is not always possible. Consequently, athletes are often required to train different physiological systems during the same training cycle. This appears particularly true for the foundation fitness attributes of strength and aerobic power. It has generally been concluded that strength gains will be compromised when trained simultaneously with aerobic power, and this has been referred to as the 'interference phenomenon'.^[2]

Several hypotheses have been proposed to explain the interference phenomenon or the decrement in gains in strength when simultaneously trained with aerobic power compared with strength training alone. The hypotheses suggest combined training results in excess fatigue, a greater catabolic state, differences in motor unit recruitment patterns and a possible shift in fibre type.^[2-4] In a recent review,^[5] the different mechanisms thought to contribute to the inhibition of strength development when concurrently training strength and endurance were defined as either chronic or acute hypotheses. The chronic hypothesis proposes that the muscle cannot adapt metabolically or morphologically to concurrent training because of the different adaptations that are being demanded. The acute hypothesis contends that strength training is compromised by the residual fatigue resulting from the endurance training. In their review, Kraemer and Nindl^[6] concluded that the different hypotheses that attempt to explain the interference phenomenon can be linked. They contend that simultaneous training produces an 'over-training' state such that the training stimuli exceed the maximal adaptive response of a given physiological system. However, their conclusion would appear to be an oversimplification, especially in relation to simultaneously developing strength and aerobic power, based on the inconclusive findings of studies that have investigated this question.

The equivocal findings have shown that combined training of strength and aerobic power results in compromised strength gains,^[7-9] uncompromised strength gains^[10-12] and uncompromised gains in muscular power,^[10,13] with no apparent compromise

in the development of aerobic power. In examining the methodology of the current literature, it is apparent that a number of different training protocols have been used to concurrently develop strength and aerobic power, which may account for the equivocal findings.^[5]

Researchers have attempted to improve aerobic power using submaximal^[8,10,11] and maximal^[7,12,14] training intensities. Some studies have implemented long, continuous training,^[8,10,15] other studies used short, interval training,^[7,13,14] and some utilised a combination of continuous and interval training.^[2,9] Duration of the training programmes has varied from 7 weeks^[13] to as long as 20 weeks^[8] and the training experiences of the individuals have ranged from untrained or limited training background^[8,10] to highly trained or elite athletes.^[2,4] The type and duration of the training protocols that have been used would provide a different training stimuli as well as the subsequent physiological adaptations.

Similarly, the protocols that have been used to develop strength have varied in the volume of training (repetitions), type of muscle action, training load, number of sets and duration of the programmes.^[2,4,7,8,10,13] Variations in the training protocols, especially in regard to intensity and volume, will elicit distinct neuromuscular adaptations. Higher loads and lower volume have been associated with enhanced motor recruitment and neural potentiation while higher volumes and lower loads have been associated with muscular hypertrophy.^[16,17]

Based on the variations in the design of the different studies, especially in the training protocols, it is difficult to make a general conclusion or interpret the findings. It is possible that the different training protocols used to elicit increases in aerobic power and strength may interact to produce different levels of interference based on the specificity of the physiological and neuromuscular adaptations.

The purposes of this paper are therefore:

- to review the effects of different training protocols used to develop aerobic power and the physiological adaptations

- to review the effects of different training protocols on the development of strength and the subsequent neuromuscular responses
- to identify the training protocols used to enhance aerobic power and strength that may produce maximum or minimum interference for adaptation
- to propose a model that may be used to study the interference phenomenon in a systematic and controlled manner.

1. Training Maximal Aerobic Power

Maximal aerobic power (MAP) is the maximal rate at which energy can be produced in a muscle primarily through oxidative metabolism.^[18] The most common measurement of MAP is maximal oxygen consumption ($\dot{V}O_{2max}$) expressed as an absolute (L/min) or relative value (ml/kg/min). $\dot{V}O_{2max}$ is enhanced by providing a stimulus that increases the ability of the body to transport and utilise oxygen. Transportation of oxygen is considered to be dependent upon the cardiopulmonary system, referred to as the central component, and the adaptations that occur at the muscle tissue level, referred to as the peripheral component.^[19] The effectiveness of the cardiopulmonary system to deliver oxygen to the muscle tissue is dependent upon pulmonary diffusion, cardiac output (\dot{Q}) and haemoglobin affinity.^[19,20] Glycogen stores in muscle, capillary density, mitochondrial volume and density, aerobic enzymes and myoglobin content all influence the utilisation of oxygen in the muscle or the peripheral component.^[21-23]

Maximal aerobic capacity is another component of aerobic fitness and refers to the maximal amount of work that can be performed using primarily oxidative metabolism. Anaerobic threshold (AT), expressed as lactate threshold (LT) or ventilatory threshold (VT) depending on the measurement technique, is considered to reflect aerobic capacity, especially when expressed as some form of power output or velocity of movement.^[18] AT is dependent upon the capability of the muscle to remove or tolerate lactic acid. Although related to $\dot{V}O_{2max}$, it is considered to have unique characteristics that

require a specific training protocol. The protocol to improve AT has normally consisted of continuous training for at least 20 minutes at AT velocity,^[24] which typically would be 75 to 85% $\dot{V}O_{2max}$, depending on the training status of the athlete. AT is considered a strong predictor of success in distance events, such as the 10 000m run and cross-country skiing.^[18] Most studies investigating the interference phenomenon have focused on developing MAP. Consequently, this article will limit the discussion to the effects of different training protocols and the development of MAP or $\dot{V}O_{2max}$.

A variety of training protocols have been found to increase $\dot{V}O_{2max}$, including different levels of training intensities.^[13,25-27] However, training at different intensity levels appears to produce different physiological adaptations or the primary locus of change. Training intensities designed to elicit improvements in $\dot{V}O_{2max}$ are typically expressed as a percentage of heart rate maximum (%HRmax) or maximal oxygen consumption (% $\dot{V}O_{2max}$). Craig et al.^[28] showed a 12% increase in MAP for well trained males from training at 75% HRmax ($\approx 65\%$ $\dot{V}O_{2max}$) whereas Cunningham et al.^[29] showed significant increases in MAP training at both 80 and 100% $\dot{V}O_{2max}$. From an extensive review of the literature Wenger and Bell^[27] concluded that greater improvements occurred in MAP as training intensity was closer to $\dot{V}O_{2max}$. Exercise above 80% $\dot{V}O_{2max}$ generally cannot be sustained for long periods of time.^[30] Consequently, exercise at this intensity is usually performed for 1 to 3 minute work intervals interspersed with similar rest time, to allow more work at higher intensity levels. This approach to training MAP is often referred to as aerobic interval training.^[19]

In addition to the magnitude of change in MAP in response to changes in training intensity, a shift in the location of the physiological adaptations seems to occur (fig. 1).

Depending on the intensity of training, the adaptation may occur in the central or peripheral component. At lower intensities, the physiological adaptations occur primarily in the central component.^[19,29] MacDougall and Sale^[19] have suggested

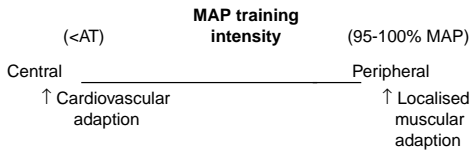


Fig. 1. Intensity continuum of maximal aerobic power (MAP) training and the primary location of adaptation. **AT** = anaerobic threshold; \uparrow = increased.

that maximal contractile forces of the heart occur at approximately 75% $\dot{V}O_{2max}$, and consequently the optimal training stimulus for enhancing the cardiopulmonary system would be at an intensity slightly below AT (70 to 80% $\dot{V}O_{2max}$). Cunningham et al.^[29] compared the effects of continuous (low intensity) and interval (high intensity) training in untrained female participants. The continuous group trained at 80% $\dot{V}O_{2max}$ for 20 minutes, 4 times per week whereas the interval group trained at 100% $\dot{V}O_{2max}$ with a 2 : 1 minute work to rest ratio. Total work was equated for both training groups. Results indicated that, although both groups showed significantly similar improvements in $\dot{V}O_{2max}$, the interval group demonstrated greater increases in the oxygen difference between the arterial and mixed venous blood ($a-\bar{v}O_2$ difference) than the continuous group, reflecting greater peripheral adaptation. The improvement elicited by the continuous group would have been more centrally mediated.

It has been proposed that peripheral adaptations are stimulated through the state of hypoxia experienced by the muscle during high intensity, aerobic interval training.^[19] Such physiological adaptations are similar to the changes elicited by exposure to high altitude. Terrados^[31] showed that simulated altitude training resulted in increased myoglobin content and oxidative enzyme activity similar to the peripheral changes resulting from high intensity aerobic training as described in reviews by Holloszy and Coyle,^[21] and Hoppeler and colleagues.^[23] Other adaptations include increases in muscle capillarisation, mitochondrial enzyme activity and myoglobin content.^[21,23]

More direct effects of training intensity and muscle hypoxia have been found through the use of near infrared spectroscopy (NIRS). NIRS is a noninvasive method of measuring total muscle oxygenation using the light absorption properties of haemoglobin and myoglobin. Several studies using NIRS have shown that the degree of muscle hypoxia is directly related to the increase in exercise intensity.^[32-34] Using an incremental exercise protocol Bhambhani and colleagues^[32] demonstrated that the level of oxygen saturation decreased as exercise intensity increased. In the first 2 minutes there was a quick increase in the level of saturation followed by a steady decline as power output increased. At approximately the VT the rate of desaturation slowed until $\dot{V}O_{2max}$ was attained and did not change much at near maximal intensities. However, the state of hypoxia was greatest at $\dot{V}O_{2max}$. Belardinelli et al.^[33] and a more recent study by Bhambhani and colleagues^[34] have also supported the relationship between hypoxia in muscle and exercise intensity.

Higher exercise intensities have also been associated with a shift in fibre-type recruitment and adaptation. Dudley et al.^[35] measured cytochrome C levels in mice after 8 weeks of training at various intensities. Cytochrome C is an important constituent in aerobic metabolism.^[36] At low training intensities (10 m/min) type I and IIa fibres were primarily recruited and showed increases in oxidative capacity. As intensity increased there was adaptation in type I and IIa fibres and an increase the oxidative capacity of IIb fibres. At 60 m/min only type IIb fibres showed any further increase in oxidative capacity.

In summary, the type of physiological adaptation to aerobic training is dependent on the training intensity. Lower intensity training is associated with changes in the cardiopulmonary mechanisms such as pulmonary diffusion, \dot{Q} and haemoglobin. As training intensity increases the location of adaptation appears to shift to the peripheral components with changes in muscle capillarisation, oxidative enzyme activity, mitochondrial volume and density, and myoglobin. Preferential recruitment of

type IIb fibres also occurs at higher levels of intensity with corresponding increases in oxidative capacity. In developing an understanding and insight into the possible interference in simultaneously training strength and aerobic power it would seem important to identify and isolate the specific training protocol used to elicit improvement in aerobic performance.

1.1 Training Muscular Strength

Improvements in muscular strength, as measured by the force produced during a maximal voluntary contraction (MVC), occur as a result of an increase in muscle cross-sectional area (CSA) and the ability to effectively activate motor units.^[16] The increase in CSA of muscle is considered to occur as a result of protein synthesis, primarily actin and myosin in the myofilaments, which produces a greater number of contractile units.^[37] Enhanced motor unit activation (MUA) results from a greater number of fibres being recruited, increased firing frequency, decreased co-contraction of antagonists, better synchronisation of MUA and inhibition of reflexive mechanisms (such as the golgi tendon organ) that normally govern the amount of force that can be generated.^[16,38] Increases in muscle CSA typically result in an increased body mass and are important in developing absolute strength. Programmes designed to enhance MUA are usually aimed at improving relative strength, or strength relative to body mass.^[17] Increases in MVC have been produced through a variety of training protocols, including the training variables of intensity (load or resistance) and volume (number of repetitions). Intensity in strength training is usually expressed as a percentage of the maximum weight an individual can lift and is referred to as 1-repetition maximum (1RM). It is also expressed as the number of repetitions that produce muscle failure such as 3RM. There is obviously an inverse relationship between the weight that is lifted and the number of repetitions that produce muscle failure.

It has been suggested that protein synthesis is stimulated by stressing the muscle energy systems to produce a significant displacement from rest.^[39]

A variety of training loads and subsequent repetitions have been found to increase the CSA of muscle. Muscle hypertrophy has been shown to occur in individuals training with loads of 6RM or greater;^[40,41] however, the greatest increases in CSA have been found to occur with 8 to 12RM loads.^[39,42-45] Although lighter RM loads (12 to 15RM) have been found to increase CSA,^[46-48] Sale and MacDougall,^[47] and Arnett^[48] have suggested that the hypertrophic response decreases as the RM load becomes lighter and the number of repetitions extends beyond 15 (fig. 2).

Consequently most practitioners recommend training at 8 to 12RM loads to induce muscle hypertrophy.^[49,50] In addition, muscle hypertrophy is also optimised when there is sufficient training volume and there are multiple exercises per muscle group.^[51] Time under tension, as reflected by the tempo of the eccentric and concentric actions, is also considered an important factor in enhancing the CSA of muscle.^[40] An 8 to 10RM loading protocol has also been found to produce the highest circulating levels of growth hormone (GH), which has been associated with protein synthesis.^[52]

Training at higher loads (4 to 6RM) has resulted in increased MVC in the absence of significant muscle hypertrophy.^[53] Such increases in force generation are attributed to neural adaptations that include increased MUA, faster firing frequency of motor units, improved synchronisation and decreased co-contraction of antagonists.^[16] Kraemer et al.^[54] have suggested that as the training stimulus promotes an increase in CSA, the contributions from the neural mechanisms to force production diminish. However, if the training stimulus is of insufficient volume to stimulate hypertrophy (sug-

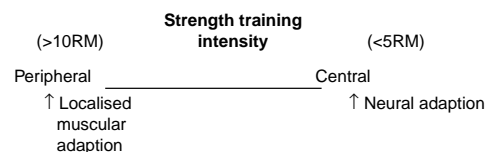


Fig. 2. Intensity continuum and primary location of adaptation for strength training. RM = repetition maximum; ↑ = increased.

gested to be >90% 1RM), greater neural adaptations occur.

Schmidbleicher and Buehle^[53] showed that although both low and high intensity strength training resulted in similar increases in MVC (21 and 18%, respectively) the contribution from neural adaptation and muscle hypertrophy were different between the 2 loading intensities. High intensity training resulted in a greater rate of force development, considered to reflect neural adaptation (34% compared with 4%), whereas low intensity training resulted in a greater increase in muscle size (7% compared with 3%). Hakkinen et al.^[55] also showed that high intensity training resulted in greater neural adaptation as reflected by increased electromyogram activity.

In summary, muscular strength can be increased by changes in the CSA of muscle from protein synthesis and neural adaptations that enhance MUA. Variations in training intensity and protocol appear to elicit different neuromuscular adaptations. High loads (3 to 6RM) and lower volume are associated with an increase in force generation without an increase in muscle size, and are related to neural adaptations. Muscle hypertrophy is produced with lower loads (8 to 12RM) and higher volume resistance training and occurs through increased protein synthesis in the muscle fibre. Consequently, different training protocols would appear to increase strength through different physiological adaptations. In examining the potential interference in simultaneously training strength and aerobic power it would seem important to identify the training protocol used to enhance strength.

2. A Model for the 'Interference Phenomenon'

It is apparent that there has been no systematic approach to investigating the interference phenomenon, with particular reference to the components of strength and aerobic power. It has been suggested that individual laboratories focus on a particular training model and perform a series of investigations using their model.^[5] Based on the physiological adaptations that result from the different training protocols used to enhance strength and aerobic power it is possible to propose a model that may provide a way in which to systematically study the question of an 'interference phenomenon'.

Figure 3 presents a model which should allow the development of hypotheses that predict the training protocols which will minimise or maximise the interference effect when simultaneously training for strength and aerobic power. It may also be possible to construct other models that allow a systematic study of interference effects between other physiological attributes. Researchers should understand the need to clearly define the training variables and deliberately select the training protocols in relation to each other.

The proposed model for the study of simultaneously training for strength and aerobic power has focused primarily on the manipulation of training intensity, with some inference that there is an inverse relationship between the intensity and volume of training. Normally, as the training intensity (resistance and % VO_{2max} in the context of strength and aerobic power, respectively) increases, the vol-

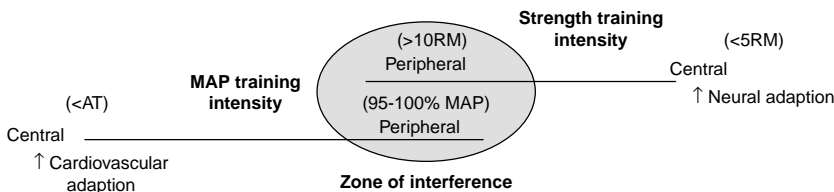


Fig. 3. Intensity continuums and primary location of adaptations for both maximal aerobic power (MAP) and strength training, and the possible overlap when the 2 modes of training are combined. AT = anaerobic threshold; RM = repetition maximum; ↑ = increased.

ume (sets and repetitions) would decrease. From the model it would be hypothesised that interference would be maximised when athletes use high intensity interval training to improve aerobic power and an 8 to 12RM multiple set resistance training protocol to increase strength. The strength training protocol would be attempting to enhance protein synthesis in the muscle and stress the anaerobic energy system with corresponding increases in muscle lactate. Aerobic interval training would create hypoxia in the muscle, requiring the muscle to increase its oxidative capability. In this situation the muscle would be required to adapt in distinctly different physiological and anatomical ways, which may reduce the adaptation of one of the systems.

If aerobic interval training was combined with high intensity (3 to 6RM) resistance training, the model would predict less interference because the training stimulus for increases in strength would stress the neural system and not place metabolic demands on the muscle. Presumably the muscle could increase its oxidative capability without affecting neural adaptation such as increased firing frequency, more efficient synchronisation of motor units, decreased inhibition and co-contraction of antagonist muscles.

Continuous aerobic training would be predicted to have minimal interference on strength development using either high load or medium load strength training protocols. The physiological adaptations associated with continuous training would be centrally mediated, involving increased \dot{Q} , haemoglobin and greater pulmonary diffusion. Consequently, it should not interfere with either neural adaptation or muscle hypertrophy since the location of physiological adaptation and metabolic response would seem to be different.

3. Testing the Model

An initial step would be to test the model against the current literature that has investigated the concurrent training of strength and aerobic power. Unfortunately, many studies do not clearly describe the training protocols, especially in regard to training intensity and volume. In addition, the exercise

modes, training duration and the training experience of participants have varied between studies and some studies have used a modified 'periodised' strategy and have changed the training protocol during the training period. Such variations in study design make it difficult to apply all investigations to the model. For this reason only studies with relatively detailed training protocols and consistent loading parameters have been included in the assessment of this model.

Support for the model is provided by McCarthy et al.^[11] who had untrained individuals train for 10 weeks with a 5 to 7RM protocol for 3 sets, 3 times a week to increase strength. Aerobic training consisted of 45 minutes continuous cycling at 75% heart rate reserve for 3 days per week. Both strength and aerobic training were not performed at intensities that would maximise interference according to the model. Neither $\dot{V}O_{2\max}$ nor strength were compromised compared with when strength and aerobic power were separately trained. $\dot{V}O_{2\max}$ increased significantly in both the separate aerobic-trained and concurrent-trained groups (18 and 16%, respectively). Strength significantly increased for both the separate-strength- and concurrent-trained groups (23 and 22% for 1RM squat, respectively, and 18% for 1RM bench press for both groups).

The physiological responses of 19 active women to 11 weeks of concurrent strength and endurance training were compared with strength training only.^[56] In addition, half the concurrent training group completed their endurance training before strength training and the other half of the group did strength training before endurance training. Manipulation of the sequence of training attempted to control for possible acute fatigue effects. Endurance training consisted of rowing at 70% $\dot{V}O_{2\max}$ performed 3 times per week. Strength training consisted of 45 minutes of 5 to 6 lower body exercises progressing from 2 to 4 sets of 10 repetitions over the training period. As the model would predict, there were no negative effects on strength training for women concurrently training strength and endurance. Furthermore, the sequence of training did

not affect strength development but may have limited MAP.

The findings of Nelson and colleagues,^[8] who trained untrained individuals for 20 weeks, provide partial support for the model. Strength training consisted of 6 maximal isokinetic leg extensions at 30° per second for 3 sets, 4 times per week, which would be considered neural training in the context of the proposed model. Aerobic training consisted of cycling at increasing intensities and duration for 6 weeks and the remainder of the training period at 85% HR_{max} for 60 minutes ($\approx 75\% \dot{V}O_{2max}$) which would be considered continuous training. As expected from the model, strength gains in the concurrent group were not compromised compared with the gains observed in the group who only strength trained. However, after 11 weeks the group performing only aerobic training continued to show significant increases in $\dot{V}O_{2max}$ while the concurrent training group did not change. It is possible that the concurrent group had optimised the training effect from continuous, low intensity training and needed a different, more intense training stimulus.

The proposed model is not supported by the results from Dudley and Djamil.^[13] Applying the protocols used in their study, the model would predict compromised training effects but in fact both concurrent and strength training groups demonstrated significant increases in strength at 0, 0.84 and 1.68 rads/sec. Unfortunately, no direct statistical comparison between the groups was reported. According to the model the strength protocol of 2 sets of 26 to 28 contractions on an isokinetic dynamometer at 4.19 rads/sec and an aerobic interval training programme of 5 × 5 minute intervals at $\dot{V}O_{2max}$ on a cycle ergometer should have produced some interference or compromise in the training effect. However, although not discussed, the concurrently trained individuals had actual improvements in peak torque that were 6% less at all 3 velocities than the individuals who only trained strength. In addition, isokinetic exercise does not eccentrically load muscle, which is considered to be an important stimulus for muscle hypertrophy.^[39] Therefore, the muscle

may not have been optimally stressed compared with dynamic constant exercise resistance (DCER).^[17] Additionally, change in muscle hypertrophy was not reported by Dudley and Djamil.^[13] Training at a relatively high velocity, which would not place the muscle under optimal tension to elicit muscle hypertrophy, also confounds the results from the study in the context of the model.

Craig and colleagues^[28] used a strength programme of 6 to 8 repetitions, for 3 sets, 3 times per week, which would be expected to produce neural and hypertrophic adaptations. Aerobic training consisted of continuous running for 35 minutes at 75% HR_{max} which should produce adaptations in the cardiopulmonary system. Based on the model, a moderate interference effect would be expected. Although Craig and colleagues concluded from their findings that concurrent training inhibited optimal strength improvement, the percentage change in leg press was similar for the strength-only- and concurrently trained individuals, being 5.8 and 4.6%, respectively. In addition, the strength training for the individuals who trained both strength and aerobic power was performed 5 minutes after aerobic training. The authors suggested that a lack of rest, and residual fatigue may well have impacted on the quality of strength training and subsequent training effect.

In a recent study,^[57] 45 males and females were randomly assigned to one of 4 groups; strength training only (S), endurance training only (E), concurrent strength and endurance training (SE), or a control group. Strength intensity was increased by 4% every 3 weeks (with a mean of 72 to 84% 1RM). Sets and repetitions ranged from 4 to 12, and 2 to 6, respectively. Endurance training consisted of 2 training sessions per week, of continuous training that started at 30 minutes and progressed to 42 minutes with intensity set at the VT; and 1 aerobic interval training session per week at a rest-to-work ratio of 3 minutes of exercise and 3 minutes active rest. Intensity was equivalent to 90% $\dot{V}O_{2max}$ and began with 4 sets and built to 7 sets. Both E and SE groups improved $\dot{V}O_{2max}$. Leg press and knee extension (1RM) strength increased in groups S and

SE but gains in knee extension were greater for the S group compared with all other groups. The authors concluded their findings indicated some interference with the development of strength that may be specific to particular movement patterns. The hybrid nature of the strength and aerobic training protocols may have contributed to the ambiguous findings and make it difficult to interpret in the context of the proposed model.

4. Conclusion

It has been suggested that simultaneously training for strength and aerobic power compromises the development of strength compared with training strength by itself. The development of aerobic power during concurrent training appears relatively unaffected. The compromised training has been referred to as the interference phenomenon. According to Kraemer and Nindl,^[6] the phenomenon is the result of overtraining. However, the equivocal findings in the literature suggest their conclusion is perhaps an oversimplification. The lack of systematic application of the training variables has made it difficult to make any definitive conclusions. A model has been proposed, based on the physiological adaptations to specific training protocols, which should provide greater understanding of the interference phenomenon. The model predicts the training protocols that will likely minimise or maximise the level of interference.

References

1. Fry R, Morton A, Keast D. Periodization of training stress: a review. *Can J Sport Sci* 1992; 17 (3): 234-40
2. Bell G, Syrotuik D, Socha T, et al. Effects of strength training or concurrent strength and endurance training on strength, testosterone, and cortisol. *J Strength Cond Res* 1997; 11 (1): 57-64
3. Chromiak J, Mulvaney D. A review: the effects of combined strength and endurance training on strength development. *J Appl Sport Sci Res* 1990; 4 (2): 55-60
4. Kraemer W, Patton J, Gordon S, et al. Compatibility of high-intensity strength and endurance training on hormonal and skeletal muscle adaptations. *J Appl Physiol* 1995; 78 (3): 976-89
5. Leveritt M, Abernethy P, Barry B, et al. Concurrent strength and endurance training: a review. *Sports Med* 1999; 28 (6): 413-27
6. Kraemer W, Nindl B. Factors involved with overtraining for strength and power. In: Kreider R, Fry A, O'Toole M, editors. *Overtraining in sport*. Champaign (IL): Human Kinetics, 1998: 69-86
7. Hickson R. Interference of strength development by simultaneously training for strength and endurance. *Eur J Appl Physiol* 1980; 45: 255-63
8. Nelson A, Arnall D, Loy S, et al. Consequences of combining strength and endurance training regimens. *Phys Ther* 1990; 70 (5): 287-94
9. Hennessey L, Watson A. The interference effects of training for strength and endurance simultaneously. *J Strength Cond Res* 1994; 8 (1): 12-9
10. Hunter G, Demment R, Miller D. Development of strength and maximum oxygen uptake during simultaneous training for strength and endurance. *J Sports Med Phys Fitness* 1987; 27 (3): 269-75
11. McCarthy J, Agre J, Graf B, et al. Compatibility of adaptive responses with combining strength and endurance training. *Med Sci Sports Exerc* 1995; 27 (3): 429-36
12. Abernethy P, Quigley B. Concurrent strength and endurance training of the elbow flexors. *J Strength Cond Res* 1993; 7 (4): 234-40
13. Dudley G, Djamil R. Incompatibility of endurance and strength training modes of exercise. *J Appl Physiol* 1985; 59 (4): 1446-51
14. Sale D, MacDougall J, Jacobs I, et al. Interaction between concurrent strength and endurance training. *J Appl Physiol* 1990; 68 (1): 260-70
15. Bell G, Pedersen SR, Wessel J, et al. Physiological adaptations to concurrent endurance training and low velocity resistance training. *Int J Sports Med* 1991; 12 (4): 384-90
16. Sale D. Neural adaptation to strength training. In: Komi P, editor. *Strength and power in sport*. Oxford: Blackwell Scientific Publications, 1992: 249-65
17. Fleck S, Kraemer W. *Designing resistance training programs*. 2nd ed. Champaign (IL): Human Kinetics, 1997
18. Thoden J. Testing aerobic power. In: MacDougall J, Wenger H, Green H, editors. *Physiological testing of the high-performance athlete*. 2nd ed. Champaign (IL): Human Kinetics, 1991: 107-73
19. MacDougall D, Sale D. Continuous vs interval training: a review for the athlete and the coach. *Can J Appl Sport Sci* 1981; 6 (2): 93-7
20. Sutton J. $\dot{V}O_{2max}$: new concepts on an old theme. *Med Sci Sports Exerc* 1992; 24 (1): 26-9
21. Holloszy J, Coyle E. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol* 1984; 56 (4): 831-8
22. Brodal P, Ingjer F, Hermansen L. Capillary supply of skeletal muscle fibers in untrained and endurance-trained men. *Am J Physiol* 1977; 232 (6): H705-12
23. Hoppeler H, Howald H, Conely K, et al. Endurance training in humans: aerobic capacity and structure of skeletal muscle. *J Appl Physiol* 1985; 59 (2): 320-7
24. Hoffman R. Effects of training at the ventilatory threshold and performance in trained distance runners. *J Strength Cond Res* 1999; 13 (2): 118-23
25. Fox E, Bartels R, Billings C, et al. Frequency and duration of interval training programs and changes in aerobic power. *J Appl Physiol* 1975; 38 (3): 481-94
26. Eddy D, Sparks K, Adelizi D. The effects of continuous and interval training in women and men. *Eur J Appl Physiol* 1977; 37: 83-92
27. Wenger H, Bell G. The interactions of intensity, frequency and duration of exercise training in altering cardiorespiratory fitness. *Sports Med* 1986; 3: 346-56
28. Craig B, Lucas J, Pohlman R, et al. The effects of running, weightlifting and a combination of both on growth hormone release. *J Appl Sport Sci Res* 1991; 5 (4): 198-203

29. Cunningham D, McCrimmon D, Vlach L. Cardiovascular response to interval and continuous training in women. *Eur J Appl Physiol* 1979; 41: 187-97
30. Åstrand P-O, Rodahl K. Textbook of work physiology: physiological bases of exercise. 3rd ed. New York: McGraw-Hill, 1986
31. Terrados N. Altitude training and muscle metabolism. *Int J Sports Med* 1992; 13 (Suppl. 1): S206-9
32. Bhambhani Y, Buckley S, Susaki T. Detection of ventilatory threshold using near infrared spectroscopy in men and women. *Med Sci Sports Exerc* 1997; 29 (3): 402-9
33. Belardinelli R, Varstow TJ, Porszasz J, et al. Changes in skeletal muscle oxygenation during incremental exercise measured with near infrared spectroscopy. *Eur J Appl Physiol* 1995; 70: 487-92
34. Bhambhani Y, Buckley S, Susaki T. Muscle oxygenation trends during constant work rate cycle exercise in men and women. *Med Sci Sport Exerc* 1999; 31 (1): 90-8
35. Dudley G, Abraham W, Terjung R. Influence of exercise intensity and duration on biochemical adaptations in skeletal muscle. *J Appl Physiol* 1982; 53 (4): 844-50
36. Lehninger A, Nelson D, Cox M. Principles of biochemistry. 2nd ed. New York: Worth, 1982
37. Goldspink G. Cellular and molecular aspects of adaptation in skeletal muscle. In: Komi P, editor. *Strength and power in sport*. Oxford: Blackwell Scientific Publications, 1992: 249-65
38. Wilson G. Disinhibition of the neural system: uses in programming, training, and competition. *Strength Cond Coach* 1995; 3 (3): 3-5
39. Houston M. Gaining weight: the scientific basis of increasing skeletal muscle mass. *Can J Appl Physiol* 1999; 24 (4): 305-16
40. Schott J, McCully K, Rutherford O. The role of metabolites in strength training. *Eur J Appl Physiol* 1995; 71: 337-41
41. Davies J, Parker P, Rutherford O, et al. Changes in strength and CSA of the elbow flexors as a result of isometric strength training. *Eur J Appl Physiol* 1988; 57: 667-70
42. Narici M, Keyser B. Hypertrophic response of human skeletal muscle to strength training in hypoxia and normoxia. *Eur J Appl Physiol* 1995; 70: 213-9
43. Narici M, Roi G, Landoni L, et al. Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur J Appl Physiol* 1989; 59: 310-9
44. McCall G, Byrnes W, Dickinson A, et al. Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. *J Appl Physiol* 1996; 81 (5): 2004-12
45. Kawakami Y, Abe T, Kune S, et al. Training-induced changes in muscle architecture and specific tension. *Eur J Appl Physiol* 1995; 72: 37-43
46. Jackson C, Dickenson A, Rinel S. Skeletal muscle fiber area alterations in two opposing modes resistance exercise training in the same individual. *Eur J Appl Physiol* 1990; 61: 37-41
47. Sale D, MacDougall J. Specificity in strength training: a review for coaches. *Can J Appl Sports Sci* 1991; 6 (2): 87-92
48. Arnett M. A review of concurrent strength and endurance training. *Sports Sci Periodical Res Technol Sport* 1993; 13 (2): 1-6
49. Poliquin C. Training for improving relative strength. *Sci Period Res Technol Sport (SPORTS)* 1991; 11 (7): 1-9
50. Schmidtbleicher D. Strength training. Part 1: classification of methods. *Sci Period Res Technol Sport (SPORTS)* 1985 Aug: 1-12
51. Baker D, Wilson G, Carolyn R. Periodization: the effect on strength of manipulating volume and intensity. *J Strength Cond Res* 1994; 8 (4): 235-42
52. Kraemer W, Marchitelli J, Gordon L, et al. Hormonal and growth factor responses to heavy resistance exercise protocols. *J Appl Physiol* 1990; 69: 1442-50
53. Schmidtbleicher D, Buerle M. Neuronal adaptations and increases of cross-sectional area studying different strength training methods. *Biomechanics* 1987; 6B: 615-20
54. Kraemer W, Fleck S, Evans W. Strength and power training: physiological mechanisms of adaptation. In: Holloszy J, editor. *Exercise and sports science reviews*. Baltimore: Williams and Wilkens, 1996: 363-93
55. Hakkinen K, Komi P, Alen M, et al. EMG, muscle fiber and force production characteristics during a 1 year period in elite weight-lifters. *Eur J Appl Physiol* 1987; 556: 419-27
56. Gravelle B, Blessing D. Physiological adaptation in women concurrently training for strength and endurance. *J Strength Cond Res* 2000; 14 (1): 5-13
57. Bell G, Syrotuik D, Martin T, et al. Effect of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations in humans. *Eur J Appl Physiol* 2000; 81 (5): 418-27

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