

REVIEW

Blood flow restriction training and the high-performance athlete: science to application

 Christopher Pignanelli,¹ Danny Christiansen,² and Jamie F. Burr¹

¹Department of Human Health and Nutritional Sciences, University of Guelph, Guelph, Ontario, Canada and ²Department of Internal Medicine, University of Utah, Salt Lake City, Utah

Abstract

The manipulation of blood flow in conjunction with skeletal muscle contraction has greatly informed the physiological understanding of muscle fatigue, blood pressure reflexes, and metabolism in humans. Recent interest in using intentional blood flow restriction (BFR) has focused on elucidating how exercise during periods of reduced blood flow affects typical training adaptations. A large initial appeal for BFR training was driven by studies demonstrating rapid increases in muscle size, strength, and endurance capacity, even when notably low intensities and resistances, which would typically be incapable of stimulating change in healthy populations, were used. The incorporation of BFR exercise into the training of strength- and endurance-trained athletes has recently been shown to provide additive training effects that augment skeletal muscle and cardiovascular adaptations. Recent observations suggest BFR exercise alters acute physiological stressors such as local muscle oxygen availability and vascular shear stress, which may lead to adaptations that are not easily attained with conventional training. This review explores these concepts and summarizes both the evidence base and knowledge gaps regarding the application of BFR training for athletes.

BFR exercise; ischemic training; sport performance; vascular occlusion

INTRODUCTION

The intentional reduction of blood flow during exercise, commonly referred to as blood flow restriction (BFR) exercise, is typically accomplished using a tourniquet to reduce arterial blood flow and restrict or occlude venous outflow. Seminal work demonstrated strength and endurance adaptations following BFR training, using modes and intensities of exercise traditionally thought incapable of stimulating change in healthy populations (1, 2). Since these original studies, the addition of BFR exercise into regular training has been shown to improve adaptations in both strength- and endurance-trained athletes (3–6). In this review, we highlight the recent advances in BFR training within the context of improving the physical preparation of athletes. Our specific purpose is threefold: 1) examine the evidence for and against a potentiating effect of BFR on training adaptations in diverse, well-trained individuals; 2) discuss possible mechanisms underpinning BFR training; and 3) identify knowledge gaps and future research directions regarding the use of BFR training in athletes. The safety aspects related to BFR exercise are beyond the scope of this review, and we direct the readers to recent literature on this matter (7, 8).

THE INFLUENCE OF BFR EXERCISE IN WELL-TRAINED INDIVIDUALS

BFR-Training Adaptations in Strength-Trained and Team-Sport Athletes

A majority of investigations to date have considered the efficacy of BFR-resistance training to improve muscle size and strength in untrained participants. More recent work also suggests BFR-resistance exercise positively influences adaptations in trained populations, with the most compelling support for its use provided in national-level powerlifters (3). In a 6.5-wk periodized strength program, Bjørnsen et al. (3) added two, 1-wk blocks consisting of front squats at either 1) low loads (~30% 1-repetition maximum; 1-RM) with continuous BFR or 2) conventional high loads (~60%–85% 1-RM). Despite the advanced training status, the addition of only 10 BFR sessions increased quadriceps cross-sectional area (CSA) by 3%–8% and increased individual muscle fiber CSA (12%), number of myonuclei (18%), and capillary-to-muscle fiber contacts (12%) for type-I muscle fibers, whereas no overall changes in these variables occurred in the conventional training group. When assessing changes in strength, no statistical differences were found after training between groups for knee-extensor peak isokinetic torque or 1-RM front squats. However, the change in peak torque from

baseline increased following BFR training (4%), whereas the peak torque did not increase with conventional training ($\sim 0\%$). By contrast, only the conventional training group statistically increased their front-squat 1-RM from baseline ($\sim 4\%$), with a nonsignificant $\sim 3\%$ increase with BFR. The lack of statistical significance between groups for each strength outcome could be due to low statistical power as a result of small effect and sample ($n = 8$ or $9/\text{group}$) sizes. However, there were positive correlations ($r = 0.63\text{--}0.79$) for changes in peak torque or 1-RM, with changes in indices of muscle size only following BFR training. Several studies suggest that compared with control training groups, the addition of BFR training can positively influence muscle adaptations such as strength or size in collegiate and semiprofessional-level athletes [reviewed in (9)] and has translated to improved task performance, which are thought to benefit sporting success, for example, jump power/height ($\sim 2\%\text{--}5\%$), change of direction speed ($\sim 9\%$), 5-m and 10-m sprint times ($\sim 3\%\text{--}16\%$), and repeat sprint ($\sim 0.6\%$) or shuttle-run ($\sim 5\%\text{--}20\%$) performances (10–14). These findings are not universal, as supplementary BFR-resistance training or sport-specific training with BFR did not improve muscle size (15) and single jump or sprint performances (13, 15) in soccer athletes (semi-professional or youth levels) compared with the training groups that did not perform BFR training. The above discrepancies may be due to endurance training being performed immediately after resistance exercise regardless of BFR (15), which could blunt general resistance-training adaptations (16). Further, the concurrent plyometric training with or without BFR (13) may explain why similar improvements in 40-m sprint and vertical jump performances were observed, such that BFR did not influence the primary determinants of these tasks any further. In summary, supplementary BFR training is able to potentiate some physical characteristics and sport-specific task performances of strength- and team-sport athletes, and importantly, seems not to be inferior compared with conventional training methods.

BFR-Training Adaptations in Endurance-Trained Athletes

It is well documented that short-term, intensified interval training can improve the physiological adaptations and performance in athletes (17). Although BFR exercise has typically been associated with resistance training, emerging evidence supports that BFR can improve the physiological response to interval training in athletes. In well-trained cyclists ($\bar{x} \dot{V}O_{2\text{max}} > 60 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$), 4 wk of supplementary sprint-interval training (SIT) with BFR during rest periods increased pulmonary maximal oxygen uptake ($\dot{V}O_{2\text{max}}$) by $\sim 5\%\text{--}6\%$ with a simultaneous trend toward a greater maximal aerobic power output ($\sim 3\%\text{--}4\%$) (5, 6). By contrast, no change occurred in control groups performing SIT without BFR. In these studies, BFR was applied during the first 2 min of each recovery period as opposed to the traditional application (i.e., during exercise), yet improvements in $\dot{V}O_{2\text{max}}$ were still apparent. Despite an increase in $\dot{V}O_{2\text{max}}$, this did not manifest as an improvement in 15-km time-trial performance from baseline (6), and critical power increased similarly compared with SIT without BFR ($\sim 3\%$) (5). It is possible that the selected time-trial test lacked the sensitivity to detect

small improvements with trained athletes. Alternatively, a shorter, higher-intensity performance test designed to elicit a greater fraction of aerobic power output may have revealed a performance benefit.

Supplementary BFR training also appears effective when performed during lower-intensity intervals. In a group of well-trained rowers ($\bar{x} \dot{V}O_{2\text{max}} \sim 63 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$), three weekly sessions of low-volume ($2 \times 10\text{-min}$ bouts), low-intensity (below first lactate threshold) rowing with BFR on the legs were added to 5 wk of regular training and compared with a control group that continued training without BFR (4). Unexpectedly, large improvements in $\dot{V}O_{2\text{max}}$ (9%) and maximum aerobic power output (15%) were observed only in the rowers who trained with BFR. No competition-performance outcomes were measured in the study. Thus, despite the large effect on aerobic variables, the impact on actual rowing performance remains unclear. These studies demonstrate that supramaximal- or low-intensity BFR training of a short duration (4–5 wk) can increase the aerobic power of endurance-trained athletes. However, translation of these benefits to competition-performance needs further evaluation.

PHYSIOLOGICAL ADAPTATIONS FOLLOWING BFR TRAINING

The previous section suggest that supplementary BFR training using a variety of exercise modalities can improve the training response of diverse athlete types, compared with regular exercise training. But, how BFR does so is also important, as this can optimize its application within a training program to improve desired physical traits. In this section, we provide an overview of the recent discoveries about the skeletal muscle and cardiovascular physiological adaptations following BFR training, their relevance to physical performance, and the possible underlying mechanisms. Most of the data presented herein have been conducted in untrained or recreationally active individuals; thus, the readers should consider that some of these physiological adaptations may not translate in well-trained individuals. However, these data are still valuable, as they provide a foundation for hypothesis-generated studies in athletic populations to improve physical performance.

Muscle Strength and Structural Adaptations

Most strength and conditioning programs seek to help athletes improve their force-generating capacity to increase power output. Indeed, increasing the maximal ability to produce force against an external object, referred to as muscle strength, can influence the economy of submaximal movements and the potential for explosive movements, such as sprinting and jumping (18). Outside of the athlete-specific literature, a recent meta-analysis has indicated that low-load BFR-resistance training increases muscle strength (grouped across 1-RM, isometric, and isokinetic tests) similar to high-load resistance training in untrained and recreationally active individuals (19). By contrast, another meta-analysis suggested superior improvements in strength (grouped across 1-RM, isometric, and isokinetic tests) with high-load resistance training (20). This discrepancy is likely due to more robust inclusion criteria in the former analysis (19)

such as the inclusion of only between-subject designs to eliminate the influence of crossover training effects between limbs and inclusion of only a single strength outcome for each study to eliminate double counting of some studies.

Indeed, the direct relationship between muscle size and strength in humans remains a contentious issue due to the difficulty of demonstrating a causative relationship between improvements in strength and increases in muscle size (21). However, it can be appreciated that muscle composition or architecture at both the microscopic (subcellular hypertrophy) and macroscopic (physiological CSA) levels can influence the functional output and force-generating capacity (22). At the microscopic level, myofibrillar protein synthesis increases more (~10%) following a single session of low-load BFR-resistance exercise compared with a work-matched control in recreationally active (≤ 1 day/wk of resistance exercise) individuals (23), and with training, daily myofibrillar protein synthesis increased comparably between low-load BFR and high-load resistance training in exercise-naïve individuals (24), suggesting a possible increase in number of contractile units. In agreement, compared with a work-matched exercise control, short-term (≤ 3 wk) low-load BFR-resistance training elevates muscle volume (~7%–10%) (25). Furthermore, muscle volume increases (~4%–8%) when BFR is applied during low-intensity walk or bike training (1, 26), despite these modalities being considered suboptimal to increase muscle size. However, when low-load exercise is performed to volitional fatigue with or without BFR (i.e., the control group performs more work), muscle size increases similarly (~5%–12%) in exercise-naïve and resistance-trained individuals (27, 28). In comparison, similar changes in muscle size may occur after low-load BFR or high-load resistance training (19, 20), albeit in untrained individuals. Thus, it is notable that muscle size increased more with the addition of BFR-resistance training compared with conventional high-load training in well-trained powerlifters (3), a finding that did not occur in recreationally active individuals who alternated between low-load BFR and high-load resistance training for 6 wk (29). On this basis, it is tempting to speculate that BFR exercise could act as a potentiating stimulus in well-trained individuals already accustomed to conventional training methods. To our knowledge, no work has investigated whether training status influences the hypertrophic response to BFR exercise. Nonetheless, based on the evidence to date, less work at similar external intensities/loads are required with BFR training to elicit a strong hypertrophic training response, which is important to maximize the potential acquisition of muscle mass within specific training phases.

Given that knee-extensor rate of force development has been shown to increase from baseline (~9%–20%) with BFR-resistance training (30), it is possible that some neural adaptations may occur to improve muscle power generating capacity. However, in contrast to the clear effects of BFR training to increase muscle size, at present, definitive conclusions regarding neural adaptations cannot be made. This is, in part, due to few studies directly investigating neural adaptations to BFR training, and most studies having used surface electromyography (EMG) to infer muscle excitability, which increases with low-load BFR-resistance training [reviewed in (31)]. Although interesting, we do not recommend relying solely on changes in surface EMG variables

when interpreting neuromuscular adaptations to training, as surface EMG does not elucidate if specific locomotor/neural activation patterns change, which could also impact force production (32). As such, future studies are required to clarify adaptations along the central and peripheral nervous systems following BFR training. It would also be of practical value to understand the impact of BFR exercise with concurrent training methods known to improve neuromuscular function (e.g., plyometrics or ballistic exercises) and to assess possible interaction effects of these strategies on the performance in high-power and sport-specific tasks.

Muscle Redox and Ionic Buffering

Muscle fatigue is multifactorial and task-dependent. However, fatigue encountered from a range of physical efforts, typically those of a short duration (≤ 10 min), has been consistently linked to myocellular redox stress (33) and ionic perturbations (34). Thus, optimizing the capacity to maintain redox and ionic homeostasis during exercise is of interest to maximize the physical performance of the athlete. A growing body of evidence supports that these components of muscle function are improved with training that either increases the immediate demand for ion transport, antioxidant activity, or both (33, 34). Although this area of research is in its infancy, cyclical BFR during interval running in aerobically trained individuals ($\bar{x} \dot{V}O_{2\max} \sim 57 \text{ mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$) increases markers associated with ion transport (e.g., *phospholemman* mRNA—a regulator of sodium-potassium pumps) and the accumulation of reactive oxygen species (*catalase/HSP70* mRNA content and HSP27 protein expression) (35). A similar cyclical BFR protocol has been used during 6 wk of bike-interval training in recreationally active individuals ($\bar{x} \dot{V}O_{2\max} \sim 50 \text{ mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$), which increased the work capacity of the knee extensors to a greater extent compared with a work-matched control (23% vs. 11%) (36, 37). These performance improvements coincide with fiber-type specific changes in muscle sodium-potassium pump subunit content (increased type-II α_1 and maintained type-I β_1 content), an increased content of phospholemman, and a higher antioxidant capacity; all adaptations that may contribute to improve potassium ion regulation (37), a proposed critical determinant of high-intensity exercise performance (38). Tentatively, similar adaptations may have contributed to the improved repeated sprint and run performances of athletes following BFR training (10–14). However, future work is required to corroborate these findings and to clarify if similar adaptations occur with different modes of BFR exercise.

Muscle Oxidative Capacity

A high oxidative capacity of skeletal muscle is fundamental to blood oxygen extraction, to maintain high aerobic work outputs, and to perform and recover from repeated high-intensity efforts. Classically, improvements in muscle oxidative capacity have been linked to greater mitochondrial content, but recent findings suggest that training can alter mitochondrial function independent of changes in content (39). Indeed, repeated, transient myocellular stresses during acute exercise (e.g., increased oxygen tension and altered energy/redox states) and the subsequent transcriptional and translational events after exercise form a basis for changing

mitochondrial physiology (40). When exercise is combined with BFR, the reduced blood-borne substrate availability (e.g., oxygen and extracellular fuel sources) increases the reliance on local substrates [e.g., glycogen, phosphocreatine (PCr)] for ATP production. These changes in metabolism could augment aforementioned stresses and signaling events and thereby potentiate mitochondrial adaptations. Accordingly, early (0–4 h) cellular responses associated with mitochondrial biogenesis such as phosphorylation of AMPK α or ACC (presumably a better indicator of change in AMPK activity than AMPK α phosphorylation in response to exercise) and/or gene expression (e.g., *PGC-1 α* mRNA content) are increased following low-load BFR-resistance exercise (41) and moderate-intensity, steady-state (42, 43) or interval (35) BFR-endurance exercise compared with work-matched controls. Notably, inconsistencies have been observed in the literature. BFR applied between supramaximal bike sprints (6) or during 15 min of low-intensity cycling (44) did not alter p38-MAPK and AMPK α phosphorylation or *PGC-1 α* mRNA expression. By contrast, compared with work-matched controls, p38-MAPK phosphorylation (45) and *PGC-1 α* protein abundance (46) increased 3 h after low-intensity BFR-treadmill walking. However, caution is warranted when assessing the outcomes from the latter two studies due to the small sample sizes ($n = 5$ or 6), the limited number of signaling proteins investigated, and lack of gene expression analyses (45, 46).

In line with the acute responses, 6 wk of low-load BFR-resistance training increased muscle mitochondrial protein synthesis and respiratory capacity similar to the changes observed after high-load resistance training (47). However, when low-load resistance training is performed to volitional fatigue, the addition of BFR does not further augment the mitochondrial respiratory capacity (28). Further, markers commonly used as a proxy for mitochondrial content (COXIV protein abundance and citrate synthase activity) did not change in these studies (28, 47). In regard to endurance-

type exercise, 4 wk of moderate-intensity bike training (45 min/session) with BFR increased citrate synthase activity more (~20%) compared with the work-matched control (48). Similarly, muscle COXIV abundance increased from baseline (~20%) following low-intensity bike training (30 min/session) with BFR (49). Favorable effects of BFR-interval training on muscle oxidative capacity have also been demonstrated with 4–6 wk of bike-interval training with cyclical BFR, which improved muscle diffusional O₂ conductance (50) and oxygen kinetics (51). However, in the former study, COXIV abundance did not significantly increase (50). A lack of an increase in markers for muscle oxidative capacity (COXIV, COXII, and citrate synthase protein abundance) has also been observed after 4 wk of supramaximal bike sprint training with BFR during the rest intervals despite improvements in $\dot{V}O_{2\max}$ (5).

In summary, despite the consistent findings of a greater work capacity and/or $\dot{V}O_{2\max}$ with BFR training, increases in muscle oxidative capacity seem dependent on the type of BFR training performed, with greatest benefits achieved from endurance- versus resistance-type BFR training. However, few BFR studies have examined functional variables of mitochondrial respiration. Notably, most studies have explored mitochondrially located proteins to estimate content, which may not necessarily parallel structural and functional changes in the mitochondrial network following training (52). Based on few published studies, the potential to increase muscle oxidative capacity in athletes with BFR training may occur via different mechanisms. Specifically, interval-type BFR training improves the skeletal muscle's mitochondrial function, independent of robust increases in markers of mitochondrial content, whereas continuous, steady-state BFR training may increase mitochondrial content. Future work is required to clarify the interaction of BFR with key training variables (duration, frequency, and intensity) on muscle oxidative capacity.

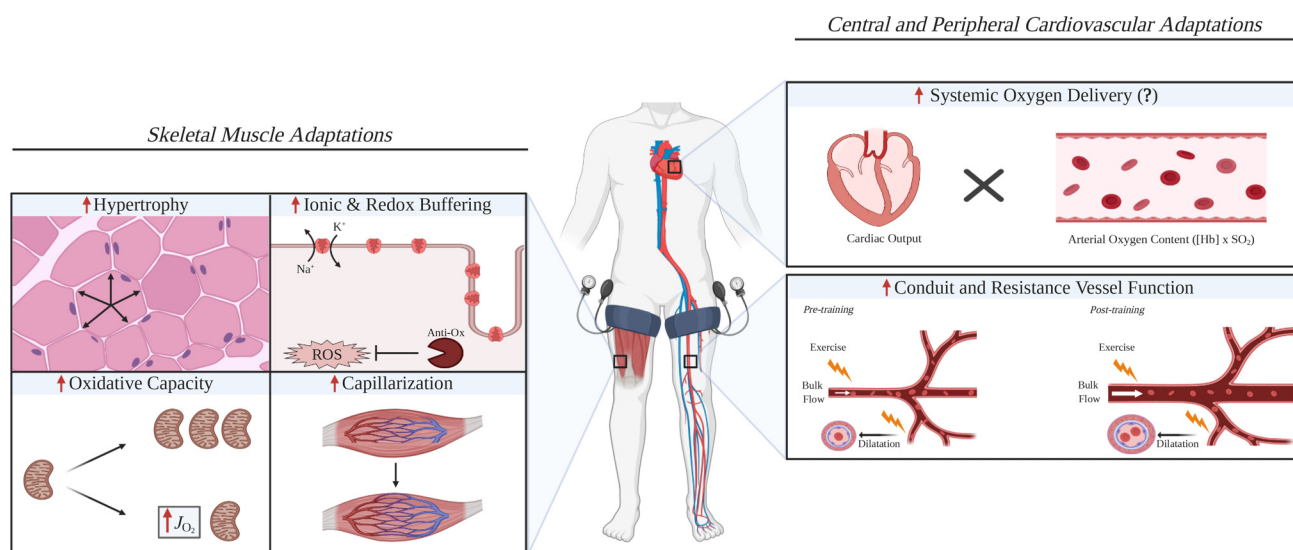


Figure 1. A schematic diagram illustrating possible skeletal muscle and cardiovascular adaptations that are improved with blood flow restriction training compared with work-matched training. Each inset was derived based on available literature with the exception of systemic oxygen delivery, which has not been investigated yet as indicated by (?). It remains to be determined how the interaction of blood flow restriction and principle exercise training variables (intensity, duration, and frequency) influence each distinct adaptation. Anti-Ox; antioxidant, J_{O_2} ; oxygen consumption, ROS; reactive oxygen species.

Cardiovascular Adaptations

The cardiorespiratory system is essential in the delivery of substrates, removal of by-products, and whole body regulatory processes (e.g., acid-base balance and thermoregulation). Thus, along with sustaining or improving intramuscular physiology with training, athletes must also maintain a well-developed cardiorespiratory system. Fundamentally, the cardiovascular system from the heart to the microvasculature strongly governs whole body $\dot{V}O_{2\max}$, which is determined by the product of maximal cardiac output ($\dot{Q}_{\max}$) and the arterial to mixed-venous oxygen ($a-\bar{v}O_2$) difference. What factor(s) limit $\dot{V}O_{2\max}$ is dependent on the interaction of systemic oxygen delivery (product of $\dot{Q}_{\max}$ and arterial oxygen content; CaO_2) with oxygen extraction ($a-\bar{v}O_2$ difference/ CaO_2), a relationship that is influenced by training status (53) and disease/health state (54). Although these factors are multivariable and not mutually exclusive [see refs (53, 54) for more information], recent evidence suggests that oxygen delivery to the active muscles may limit $\dot{V}O_{2\max}$ in endurance-trained individuals as opposed to intrinsic skeletal muscle mitochondrial respiratory capacity (55). On the contrary, mitochondrial respiratory capacity may play a contributing role in $\dot{V}O_{2\max}$ limitations in untrained individuals (55).

Different $\dot{V}O_{2\max}$ limitations in endurance-trained versus untrained subjects are intriguing because improvements in $\dot{V}O_{2\max}$ with BFR training have been observed regardless of exercise mode or intensity in both the untrained (26, 49, 56) and trained states (4–6). Local adaptations along the vascular tree, such as an increase in conduit artery blood flow and muscle capillary density, and those within skeletal muscle such as an elevated oxidative capacity probably contribute to increase $\dot{V}O_{2\max}$ with BFR training (48, 50). Nevertheless, given the strong relationship between $\dot{Q}_{\max}$ and $\dot{V}O_{2\max}$ (53), central cardiovascular improvements may also contribute to increase $\dot{V}O_{2\max}$ in some instances [e.g., endurance-trained athletes (4–6)]. The lack of an increase in muscle capillarity or mitochondrial protein content in endurance-trained athletes, despite robust increases in $\dot{V}O_{2\max}$ with SIT + BFR (5), supports the notion of central cardiovascular improvements. To date, no studies have directly investigated changes in cardiac structure and function following BFR training. Theoretically, increases in cardiac demand (attenuated stroke volume and increased double product) with BFR exercise (57) might expedite cardiac structural adaptations such as an increase in left ventricular size, which could elevate stroke volume and therefore $\dot{Q}_{\max}$. Although this proposition is enticing, structural changes within the heart take months to years of training (58), thus, casting doubt as a primary mechanism with short-term BFR training. On a speculative note, hematological variables such as plasma volume and hemoglobin mass can increase at the onset of short-term training to elevate stroke volume and systemic oxygen delivery (58), but these possibilities also remain to be investigated with BFR training.

Although $\dot{Q}_{\max}$ and CaO_2 dictate the systemic oxygen delivery capacity, the peripheral vascular bed of the exercising muscle plays an important role in the regulation of blood flow and substrate delivery/uptake (54). Compared with regular exercise, BFR exercise provides a unique pattern of blood flow (37). During BFR exercise, muscle oxygen supply

is reduced, which stimulates the release of local vasodilatory substances from the vasculature and contracting muscles. As a result, upon tourniquet/cuff release, a large increase in blood flow transiently elevates vascular shear stress (37). Both the reduced muscle oxygen supply during exercise and elevated vascular shear stress after exercise supports that BFR training may promote adaptations in skeletal muscle blood perfusion along the peripheral vascular tree, as has been reported (37, 48). Accordingly, BFR-endurance and BFR-resistance exercise acutely increases the mRNA content for regulators of angiogenesis (41, 59), and over several weeks, may expand the capillary network (~20%–40%) with training compared with work-matched controls (48, 60). However, when resistance training is performed to volitional fatigue, microvascular expansion increased similarly with and without BFR (~14%–18%) (28). Together, these findings highlight that the addition of BFR to endurance- or resistance-exercise magnifies the short-term angiogenic response for a given amount of exercise. Moreover, BFR-resistance training positively influences resistance and conduit vascular function and structure (61, 62), although impairments in vascular function have been observed with this type of BFR training (63). This discrepancy could be due to an interaction

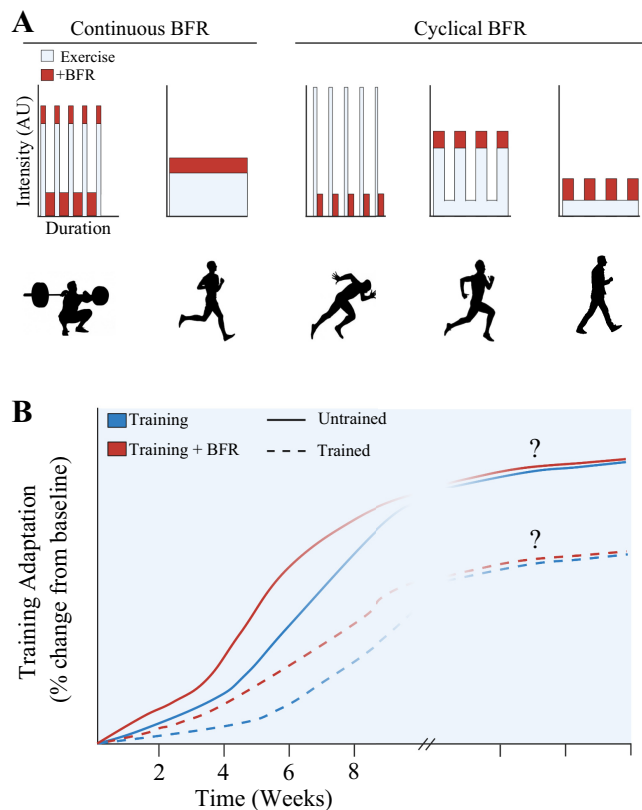


Figure 2. A: summary of the applications of blood flow restriction (BFR; red) with different exercise (gray) intensity, duration, and frequency. Exercise modes (walking/running, cycling, rowing) can be interchangeable. B: a hypothetical illustration of training adaptations without (blue) or with (red) BFR in untrained (solid lines) and trained (dashed lines) states. Note that the addition of BFR may accelerate the adaptations at the onset of training in both scenarios, but the magnitude and timeline of change may be different. The fading of each line and the question marks indicate that less is known about the temporal response to training with and without BFR for prolonged periods (>8 wk).

effect of exercise intensities [30%–40% 1-RM (61, 62) vs. 60% 1-RM (63)] with BFR duration [<10 min (61, 62) vs. 20 min (63)].

Seeing how vascular structure and/or function are improved with cyclical BFR [i.e., ischemic preconditioning (64)] and exercise training (65), the combined application (cyclical BFR exercise) could produce an additive or synergistic vascular effect. However, limited experimental evidence currently exists in support. In response to 6 wk of BFR bike-interval training, knee-extensor oxygen delivery at a high intensity (90% of incremental peak power output) increased by 13% from baseline, whereas the free-flow condition did not improve with training (50). Because of the within-subject design, this observation supports an expanded oxygen transport capacity with BFR-interval training due to peripheral mechanisms. This is clearly relevant for sport performance, as an improved oxygen delivery for the same absolute workload can facilitate a better matching of oxygen delivery to the metabolic demands in the exercising muscles, thus delaying fatigue, which could permit athletes to perform at higher absolute workloads/speeds. Although promising, more mechanistic work is required to evaluate the vascular effects of this BFR model such as changes in capillary supply and/or improved control of blood flow (e.g., functional sympatholysis and capillary recruitment) and to explore if other BFR-exercise modes have similar potential to improve oxygen delivery during exercise.

CONCLUSION AND PERSPECTIVES

There is accumulating evidence supporting that the addition of BFR exercise to an existing training program can improve the physical characteristics of diverse athletic populations, with some adaptations coinciding with improvements in sport-specific tasks or performance outcomes. Research has started to unravel how the addition of BFR may mechanistically improve strength and endurance performance, but more work is required to translate laboratory findings to real-world applications.

Because the manipulation of muscle perfusion is the overarching consequence of BFR, it is perhaps not surprising that BFR combined with various exercise modes and intensities evokes a broad spectrum of training adaptations (Fig. 1). Indeed, the BFR literature is a good illustration of how classically defined “endurance” and “resistance” training adaptations are not bimodal but rather happen simultaneously along a continuum that appears accentuated with BFR. However, the combination of different exercise modes and BFR protocols throughout the literature makes it challenging to define the physiological mechanisms that underpin improvements in physical performance with BFR training. Further characterization of how the skeletal muscle, neural, and cardiovascular systems adapt to the acute physiological perturbations imposed by BFR is required, with attention to the key exercise variables (duration, frequency, and intensity). Real-world applicability of BFR training for the elite athlete would also benefit from consideration of how BFR-stimulated training adaptations best fit within a typical periodized training program.

Despite the appeal of BFR exercise as an adjunct training modality to facilitate short-term adaptations in well-trained populations, the most effective method of implementation

remains uncertain (Fig. 2). Another current limitation to the understanding of BFR training for athletes is that the majority of mechanistic insight has been gleaned from recreationally active or untrained individuals. Since training status influences the response to exercise, there is a need to characterize the athlete response to BFR exercise. Nonetheless, the recent advances in understanding how BFR influences the physiological adaptations to training and the insights gained offers promise of improved targeted application of BFR exercise as an adjunct modality for optimizing the physical performance of athletes.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

C.P., D.C., and J.F.B. conceived and designed research; C.P. prepared figures and drafted manuscript; D.C. and J.F.B. edited and revised manuscript; C.P., D.C., and J.F.B. approved final version of manuscript.

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