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Divergent blood pressure response following high-intensity interval exercise: a signal of delayed recovery?

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Abstract

The objective of this commentary is to highlight potential factors influential to the adaptation of high-intensity exercise. Herein, we present a rationale supporting the contention that elevated systolic blood pressure, following a bout of high-intensity exercise, may be indicative of delayed/incomplete recovery. Relative to type I skeletal muscle fibers, the unique cellular/vascular characteristics of type II muscle fibers may necessitate longer recovery periods, especially when exposed to repeated high-intensity efforts (i.e., intervals). In addition to the noted race disparities in cardio-metabolic disease risk, including higher mean blood pressures, African Americans may have a larger percentage of type II muscle fibers, thus possibly contributing to noted differences in recovery following high-intensity exercise. Given that optimal recovery is needed to maximize physiological adaptation, high-intensity training programs should be individually-tailored and consistent with recovery profile(s). In most instances, even among those susceptible, the risk to non-functional over-reaching can be largely mitigated if sufficient recovery is integrated into training paradigms.

Keywords

Arterial elasticity; over-training; sympathetic tone; vasodilation

Introduction

Successful exercise training, characterized by noted improvement in cardio-metabolic health and/or sport-specific performance, is the resultant effects from the appropriate manipulation of frequency, intensity, and volume of training stimuli. Given that optimal exercise-induced adaptation necessitates matching progressive overload with suitable recovery, both under-training (i.e., insufficient exercise stimulus) and non-functional over-reaching (i.e.,

exceeding recovery capacity) can interfere with physiologic progression. Indeed, effective adaptation requires transient periods of over-reaching (i.e., functional over-reaching), as indicated by a recent joint position statement by the European College of Sport Science and American College of Sports Medicine (28). However, over-reaching becomes problematic should the individual fail to achieve expected performance gains, thus leading to stagnation (i.e., non-functional over-reaching). While slight nuances concerning the presentation of these stages may exist (Table 1), it is generally believed the time needed for the restoration of performance is the defining characteristic of exercise-induced overload. Over-training syndrome, on the other hand, is characterized by prolonged performance decrement with multiple maladaptive symptoms (e.g., systemic inflammation; depressed immune function) and the resultant effects of chronic non-functional over-reaching (28). From the novice to elite-athlete, training load should be monitored and adjusted in a manner consistent with individual responsiveness. Thus, identifying objective biomarkers suitable for tracking individual tolerance to a prescribed exercise training schema are needed to mitigate the risk of non-functional over-reaching, and ultimately, over-training syndrome. (28). To this end, the present commentary endeavors to identify potential physiologic factors that may influence successful adaptation to high-intensity exercise training.

Variability in Exercise Training Response

Habitual aerobic exercise training generally associates with increased maximal oxygen uptake ($\dot{V}O_{2max}$) and decreased risk factors of cardio-metabolic disease, including blood pressure. However, a key feature in exercise training is the variability surrounding successful adaptation, as some exhibit large changes and corresponding improvement while others are seemingly non-responsive to the training load. Though an official definition does not exist, the terms ‘responder’ and ‘non-responder,’ are generally meant to distinguish responders (those who demonstrate a measurable health or performance gain) from non-responders (those that do not). Previous work has shown that up to 20% of individuals who participate in a structured exercise training program qualify as ‘non-responders’ (35). However, the issue becomes increasingly complicated as an individual may be a non-responder for one outcome, but not for others. For example, individuals undergoing aerobic training may be unable to improve their $\dot{V}O_{2max}$, but on the other hand, demonstrate an increase in glucose control as evidenced by decreased HbA_{1c} (32). In the classical sense, some individuals may quickly adapt to aerobic training but fail to achieve any appreciable gains in muscle size/strength from resistance training (*or vice versa*). From a practical perspective, this is unsurprising, especially when one considers the differential cellular/vascular signaling pathways responsive to aerobic and resistance training (33).

As previously mentioned, training load can be altered by modifying exercise frequency, intensity, or volume. If performed correctly, recovery will keep pace with the training load to elicit healthy adaptation. In general, greater intensity (or more frequency/volume) is associated with increased adaptability to the training stimuli; however, there are exceptions which can lead to maladaptive outcomes. It is possible to prescribe too much overload thereby blunting potential improvements or even diminishing performance with excess high-intensity, high-frequency, or high-volume training (19, 20, 35). In addition, three common overuse injuries including stress fractures, tendonitis, and posterior tibialis syndrome have

been linked to over-training. This training-induced overload is often defined as non-functional over-reaching that can transition to over-training syndrome. Certainly, there are multiple factors that may influence individual responsiveness to an exercise training program including: age, genetics, environment, nutritional status, as well as, psychological stressors.

Genetic variance is one factor that presumably accounts for a considerable amount of the variance in adaptability to exercise training. Individuals from the same family tend to respond similarly, but those from different families can exhibit marked differences (2, 4). Baumert et al. (3) recently reported that the TRIM (MuRF-1) gene polymorphism is associated with indicators of exercise-induced muscle damage following eccentric exercise. Apparently, individuals with the African Americans (AA) homozygote were stronger and recovered more rapidly following a taxing bout of eccentric exercise compared to individuals with the GG homozygote. These observations suggest that underlying genetic influence may be a determinant in the responsiveness to exercise type (i.e., high-intensity interval). The links between exercise recovery and genetic polymorphisms are suggestive that some individuals may be inherently sensitive to the physiologic perturbations caused by strenuous, high-effort (i.e., ↑ frequency or ↑ intensity) exercise. Susceptible individuals may be more vulnerable to periods of overload, and if unchecked, may surpass their capability to recovery, becoming over-trained. However, it is important to note that this does not contraindicate the utility/benefits from high-intensity training, though it does specify, concessions may be needed on an individual-basis by modifying training frequency or volume to facilitate adequate recovery between exercise sessions.

Possible Indicators of Non-functional Over-reaching and Over-training Syndrome

Non-functional over-reaching, though multi-faceted, is the resultant effects from chronic perturbations placed on the autonomic, endocrinologic, and immunologic systems (5). While over-training syndrome is the ultimate expression of chronic non-functional over-reaching, the two often exhibit over-lapping symptoms such that it is difficult to determine (in the present) whether an individual is presenting non-functional over-reaching or over-training syndrome. Nevertheless, derangement of the regulatory systems can negatively affect both physiologic/psychologic function and may result in improper execution of movements, unintended weight loss, extreme muscle soreness, difficulty sleeping, apathy, and depression (17, 22). It is believed that the progression to over-training syndrome may be due or may work in concert with systemic inflammation, which in turn, elicits undesirable transient neuro-hormonal changes and central (5) fatigue. Thus, in the absence of proper rest periods, heightened inflammatory responses can be prolonged and contribute to greater pathologic stress. The psychological impact from persistent health/performance degradation linked with over-training syndrome can be very discomforting. The Profile of Mood States (POMS), a questionnaire that measures both general and specific moods, has revealed athletes can exhibit significant elevations in negative mood states (e.g., anger, confusion, and tension) that parallel a decrease in positive mood states (e.g., vigor) during periods of rigorous physical training (5, 17). Unfortunately, by the time these symptoms have manifested, individuals may have reached a point of non-functional over-reaching that requires weeks to

fully recover. As such, identifying suitable biomarkers to track individual tolerance to a prescribed exercise training schema are of great interest.

Resting Blood Pressure as a Recovery Signal?

Briefly, central and peripheral mechanisms govern activities of the circulatory system and heart rate wherein mechanically-/metabolically-sensitive receptors relay information to defend blood pressure homeostasis (15). Consistent with inputs from higher brain function and contracting skeletal muscle, heart rate and blood pressure rise in an intensity-dependent manner to ensure appropriate tissue perfusion during exercise (30). Since mean arterial blood pressure is a product of cardiac output and total peripheral resistance, habitual aerobic exercise training characteristically reduces blood pressure. While improvements are frequently detected at rest they can also be seen during exercise, due in part, to improved endothelial function and increased vascular compliance (9, 26). However, given the demanding nature of high-intensity exercise, blood pressure can transiently increase in response evoked (possibly) by neural (Group III/IV afferents) and local (oxidative stress) perturbations. Indeed, elevated resting blood pressure, over normal levels, has been shown to associate with a disruption of the autonomic nervous system (i.e., sympathetic over-activity), and as such, may be representative of delayed/incomplete recovery from high-intensity exercise (19, 20). McKeag et al. (27) have reported that at the start of preseason, 22 of 94 NCAA Division I football players had persistently elevated resting blood pressures. Serial blood pressure measures were used to determine that elevated blood pressures correlated with the high-intensity training. Through the actual season, blood pressure monitoring revealed, in some, that severe systolic hypertension (> 160 mm Hg) doubled by early season. To alleviate this irregularity, the intensity of training was lowered such that, all but two athletes returned to a normotensive state by the end of the season. Results from this study support the possibility that training-induced increases in resting blood pressure may be largely mitigated by truncating the volume of high-intensity training. Similar findings have also been observed in a small group of power athletes where resting blood pressures had increased from 120/70 mm Hg (at baseline) to 160/100 mm Hg after performing high-intensity exercise over 5 days per week for several weeks. Of note, resting blood pressures returned to normal (i.e., normotensive) upon determining that the athletes could only tolerate 2–3 days of high-intensity training per week (37).

Chronically elevated resting blood pressures have been shown to associate with poor weight lifting performance (19). Eight lifters from a Middle Eastern National Olympic weight lifting team exhibited markedly elevated resting systolic blood pressures of $+20$ mm Hg (from ≈ 120 – 130 mm Hg, taken just prior to a training session) across a six-week training cycle leading up to an international competition (Figure 1). Just a single individual was able to duplicate the training lifts completed early in the training cycle. Importantly though, he was the only lifter that *did not* experience an elevation in systolic blood pressure. After a short rest following the event, blood pressures returned to normal, after which, the lifters trained for another international event to be held six weeks later. Preparations for this competition were modified such that any athlete presenting an increased resting blood pressure by 10 mm Hg, would perform a significantly reduced training session for the day. As shown in Figure 1, systolic blood pressures did not increase, which also coincided with

an exceptional team performance (e.g., 10 personal records and 8 national records were set). Interestingly, the most accomplished lifters appeared to be highly susceptible to non-functional over-reaching, as they experienced the largest and most frequent elevations in systolic blood pressure following high-intensity exercise bouts. In a separate study, a mixed-cohort (e.g., sprint cyclists and road cyclists) of competitive cyclists performed high-intensity/high-volume exercise 5x/week for 4 consecutive weeks (20). Each session included both cycling and resistance training. Cycling consisted of interval exercise on a cycle ergometer at a fixed ratio of 60 sec of work followed by 60 sec of rest until RPMs could not be maintained. If 15 intervals were completed, without a decrease in work rate, workload was increased during next training session. Immediately following each workout session on the cycle ergometer, participants performed resistance training which included 3 sets (e.g., set 1 for 15 repetitions, set 2 for 10 repetitions and set 3 for 5 repetitions) of nine total body exercises. Resistance was increased when participants completed the entirety of prescribed repetitions. Importantly, resting systolic blood pressure increased dramatically in the sprint cyclists who are known to have an increased percentage of type IIx fibers, which was not observed among the road cyclists who rely more so on well-developed type I oxidative muscle fibers. These results appear suggestive that perturbations in the autonomic system, manifested by an elevated resting blood pressure, may be an early sign indicative of delayed/inadequate recovery. In addition, those individuals with larger power outputs (associated with a greater density of type IIx muscle fibers) may be more vulnerable to non-functional over-reaching, as evidenced by an increased blood pressure.

Skeletal Muscle Fiber Type and Systolic Blood Pressure

Skeletal muscle fiber type is related to the potential for power output, as it is well-established that individuals with a large percentage of type II muscle fibers tend to be more powerful during ballistic movements. Within this context, our group contends that individuals with a proportionally higher percentage of type II muscle fibers may have an increased propensity for exercise-induced elevations in resting blood pressure. Consistent with this premise, muscle fiber type has been linked to resting blood pressure and endothelial dysfunction (14, 16, 23). Fisher et al. (14) previously showed that systolic blood pressure (in premenopausal women) was inversely associated with large artery elasticity, and additionally, large artery elasticity was inversely associated with the percentage of type IIx muscle fibers. It is noteworthy that others have also found the percentage of type IIx muscle fibers to be positively associated with systolic blood pressure *during* exercise (18). Collectively, these results raise the question of how skeletal muscle fiber type may be influencing blood pressure.

It is well-known that type IIx muscle fibers exhibit marked phenotypic differences compared to type I muscle fibers, including lower capillary density, less mitochondria, and lower oxidative capacity (38). Type II muscle fibers (34), as well as, the percentage of fast-twitch muscle fibers (8) have been linked to greater oxidative stress following exercise training. Therefore, it is probable that the relationship between type IIx muscle fibers and resting systolic blood pressure may be mediated, at least in part, by the combined effects of increased resistance to flow (due to ↓capillary density) and increased potential for reactive oxygen species (ROS) production (due to ↓mitochondria and ↓oxidative capacity).

Accordingly, if muscle fiber type were related to systolic blood pressure it would also seem possible that exercise with significant intensity, to perturb the skeletal muscle to adapt, may also affect blood pressure responses. Indeed, we have found this to be the case in our recent unpublished observation, where the percentage of *type IIa* muscle fibers were negatively associated with the change in resting systolic blood pressure ≈ 22 hours after a high-intensity bout of cycle ergometry. It is important to note, the exercise session occurred following an 8-week period of aerobic exercise training which corresponded with a +28% increase in mitochondrial oxidative phosphorylation capacity (21). These data appear suggestive that aerobically-conditioned type IIa muscle fibers may augment the ability to recover from high-intensity efforts.

Racial Differences in Skeletal Muscle Fiber Type and Cardiovascular Disease Risk

Mortality due to cardiovascular disease continues to be a major public health problem, particularly among AAs who have a disproportionately higher cardiovascular disease mortality (29). Given the increased vascular resistance and increased potential for ROS production, having a higher percentage of type IIx fibers may influence the susceptibility for increased blood pressures. Indeed, several studies have shown that AAs tend to have a lower percentage of type I or a higher percentage of type IIx muscle fibers compared to European Americans (EAs) (1, 31, 36). However, one study by Duey et al. did not find statistically significant differences between AAs and EAs (10), although, AA men did have a 14% lower percentage of type I muscle fibers compared to EA men.

Though a modifiable factor, hypertension is felt to be a health risk that contributes not only to cardiovascular disease but also to the disparity between AAs and EA, as individuals of African descent are more likely to be hypertensive (7, 12, 25). Environmental factors may account for much hypertension prevalence among AAs, but physiological factors should also be considered. Certainly, the link between skeletal muscle fiber type and resting blood pressure, coupled with the tendency for AAs to have a lower percentage of type I muscle fibers, it is plausible that skeletal muscle characteristics may contribute to elevated risk for hypertension. Under these circumstances, aerobic exercise training would seem the optimal strategy to alleviate cardiovascular disease risk and promote cardiovascular health. Unfortunately, few clinical trials have compared the effects of exercise training between AAs and EAs with these purposes. It is unclear whether individuals endowed with a relatively high percentage of type II muscle fibers are *more or less* likely to improve cardio-metabolic health outcomes after exercise training. Though speculative, it is intriguing to consider that individuals with the highest percentage of type II muscle fibers, and thus, the lowest capillary density/mitochondrial content may benefit from aerobic training to a greater extent (in terms of cardio-metabolic health) than individuals with a relatively low percentage of type II muscle fibers.

Racial Divergence in Blood Pressure Following High-intensity Exercise

Previously our group has shown that exercise-trained AA women exhibit an increased systolic blood pressure response ≈ 22 hours after an unaccustomed bout of high-intensity

exercise (6). These findings were in stark contrast (AA +7 mm Hg vs. EA -3 mm Hg; $p = 0.04$) to the decreased systolic blood pressure observed among exercise-trained EA women who had performed the same relative exercise challenge (one hour of interval work at 84% peak $\dot{V}O_2$). It is important to note, among the total study sample ($n = 22$), the changes in systolic blood pressure were negatively associated with the changes in small artery elasticity (a reliable index of endothelial function), suggesting the elevation in resting systolic blood pressure among AA women may have been due, in part, to increased vascular resistance and/or disruption in vasodilation (possibly due to $\uparrow$ ROS). Consistent with this premise, Durand and Gutterman (11) have previously described how extreme, high-intensity exercise can instigate excessive ROS production leading to endothelial dysfunction and acute hypertension. As noted, laminar shear stress is a fundamental stimulus to trigger the release of nitric oxide (NO) and expression of endothelial NO synthase (24), yet there appears to be a point of diminishing returns wherein high-intensity exercise can become maladaptive. Interestingly, AA women have been shown to exhibit greater myeloperoxidase (marker of oxidative stress) compared to EA women, before and after weight loss (13). In this context, when coupled with an unaccustomed bout of high-intensity exercise, the underlying difference in oxidative stress may have shifted the balance of ROS/NO toward ROS generation ($\uparrow$ transient endothelial dysfunction/increased vascular resistance) that may have contributed to the elevated systolic blood pressure we previously noted in AA women. Our group have also found that changes in circulating insulin, adjusted for the percent of type IIa muscle fibers, were negatively associated with changes in systolic blood pressure following a bout of high-intensity interval cycling (partial $r = -0.66$, $p = 0.02$, unpublished results). Our interpretation of these findings posits that exercise-conditioned (i.e., non-naïve) type IIa muscle fibers may facilitate a more rapid recovery from heightened physiological strain imposed by high-intensity exercise. Since all participants in this study had performed at least 8 weeks of supervised, aerobic training (at the time of the testing), it is unclear whether untrained type IIa muscle fibers would mitigate the potential rise in blood pressure following an effort of high-intensity exercise.

Conclusion

Elevated systolic blood pressure following a high-intensity exercise bout may be indicative of delayed/incomplete recovery. As shown in Figure 2, we have put-forth a theoretical framework postulating that among individuals with a higher percentage of type II muscle fibers (especially in the untrained state) may require longer periods to recover when exposed to repeated high-intensity efforts. It is also possible that AAs may have a higher percentage type II muscle fiber which could contribute to an increased risk of hypertension. Though exercise training normally reduces blood pressure, independent of racial ancestry, systolic blood pressure ≈ 22 hours following an unaccustomed high-intensity exercise bout has been shown to significantly increase systolic blood pressure in AAs but not EAs. The observed increase in systolic blood pressure among AAs may be a signal for delayed recovery, and thus a degree of over-reaching. As many tend to believe more is always better, there is a point of diminishing returns, as too how much high-intensity training may lead to impaired performance and risk of injury. However, in an attempt to maximize training adaptation, coaches/trainers should incorporate a standardized system to consistently monitor resting

blood pressure among athletes. Adequate manipulation of exercise training frequency, intensity, and volume may serve to prevent elongated periods wherein the training stimulus exceeds recovery capacity (i.e., non-functional over-reaching). Ultimately, even among those susceptible, the risk to non-functional over-reaching/over-training syndrome can be moderated if sufficient recovery is integrated into training paradigms. Future research is needed to delineate the mechanistic origin(s) that may be contributing to the exercise-induced elevation in blood pressure and determine whether this phenomenon is accurately predictive of non-functional over-reaching/over-training syndrome. Additionally, research should determine which variants in exercise prescription (e.g., intensity, volume or frequency) are more important to mitigate the risk of non-functional over-reaching/over-training syndrome.

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Reference List

1. Ama PFM, Simoneau JA, Boulay MR, Serresse O, Theriault G, and Bouchard C. Skeletal muscle characteristics in sedentary Black and Caucasian males. *J. Appl. Physiol* 61:1758–1761, 1986. [PubMed: 2946652]
2. An P, Teran-Garcia M, Rice T, Rankinen T, Weisnagel SJ, Bergman RN, Boston RC, Mandel S, Stefanoyksi D, Leon AS, Skinner JSR, and Bouchard C. Genome-wide linkage scans for prediabetes phenotypes in response to 20 weeks of endurance exercise training in non-diabetic whites and blacks: the HERITAGE Family Study. *Diabetologia* 48:1142–1149, 2005. [PubMed: 15868134]
3. Baumert P, Lake M, Drust B, Stewart CE, and Erskine RM. TRIM63 (MuRF-1) Gene Polymorphism is associated with biomarkers of exercise-induced muscle damage. *Physiol Genomics* Epub ahead of print, 2017.
4. Boule NG, Wisnagel SJ, Lakka TA, Tremblay A, Bergman RN, Rankinen T, Leon AS, Skinner JS, Wilmore JH, Rao DC, and Bouchard C. Effects of exercise training on glucose homeostasis: the HERITAGE Family Study. *Diabet Care* 28:108–114, 2005.
5. Carfagno DG and Hendrix JC. Overtraining syndrome in the athlete: current clinical practice. *Curr Sports Med Rep* 13:45–51, 2014. [PubMed: 24412891]
6. Carter SJ, Goldsby TU, Fisher G, Plaisance EP, Gower BA, Glasser SP, and Hunter GR. Systolic blood pressure response after high-intensity interval exercise is independently related to decreased small arterial elasticity in normotensive African American women. *Appl Physiol Nutr Metab* 41:1–7, 2016. [PubMed: 26642915]
7. Ceaser THG Black and White Race differences in aerobic capacity, muscle fiber type, and their influence on metabolic processes. *Sports Med.* 45:615–623, 2015. [PubMed: 25739558]
8. Chang CC, Yang MH, Tung HC, Tsai YL, Huang JP, Yen TH, and Hung LM. Resveratrol exhibits differential protective effects on fast- and slow-twitch muscles in streptozotocin-induced diabetic rats. *J Diabetes* 6:60–67, 2014. [PubMed: 23786522]
9. Cornelissen VA and Smart NA. Exercise training for blood pressure: a systematic review and meta-analysis. *J Am Heart Assoc* 112004473:1–9, 2013.
10. Duey WJ, Basset DR, Torok DJ, Howley ET, Bond V, and Trudell R. Skeletal muscle fiber type and capillary density in college-aged blacks and whites. *Ann. Hum. Biol* 24:323–331, 1997. [PubMed: 9239438]
11. Durand MJ and Gutterman DD. Exercise and vascular function: how much is too much? *Can J Physiol Pharmacol* 92:551–557, 2014. [PubMed: 24873760]

12. Egan BM, Zhao Y, and Axon RN. US trends in prevalence, awareness, treatment, and control of hypertension, 1988–2008. *JAMA* 303:2043–2050, 2010. [PubMed: 20501926]
13. Fenster C, Darley-Usmar VM, Landar AL, Gower BA, Weinsier RL, Hunter GR, and Patel RP. Weight loss and race modulate nitric oxide metabolism in overweight women. *Free Radical Bio & Med* 37:695–702, 2004. [PubMed: 15288126]
14. Fisher G, Windham ST, Griffin P, Warren JL, Gower BA, and Hunter GR. Associations of human skeletal muscle fiber and insulin sensitivity, blood lipids, and vascular hemodynamics in a cohort of premenopausal women. *Eur J Appl Physiol* 117:1413–1422, 2017. [PubMed: 28497385]
15. Fisher JP, Young CN, and Fadel PJ. Autonomic adjustments to exercise in humans. *Compr Physiol* 5:475–512, 2015. [PubMed: 25880502]
16. Frisk-Holmberg M, Essen B, Fredrikson M, Strom G, and Wibell L. Muscle fiber composition in relation to blood pressure response to isometric exercise in normotensive and hypertensive subjects. *Acta Med Scand* 213:21–26, 1983.
17. Henschen KP Prevention and treatment of athletic staleness and burnout. *Sci Period Res Technol Sport* 10:1–8, 1990.
18. Houmard JA, Weidner ML, Koves TR, Hickner RC, and Cortright RL. Association between muscle fiber composition and blood pressure levels during exercise in men. *Am J Hypertens* 13:587–592, 2000.
19. Hunter GR Overtraining and systolic blood pressure. *Int Olymp Lift* 7:30–31, 1980.
20. Hunter GR and McCarthy JP. Pressor responses associated with high-intensity anaerobic training. *Phys Sports Med* 11:151–154, 1983.
21. Hunter GRMDR, Carter SJ, Gower BA, Bamman MM, Hornbuckle LM, Plaisance EP, and Fisher G. Potential causes of elevated REE after high-intensity exercise. *Med Sci Sports Exerc* 49:2414–2421, 2017. [PubMed: 28737531]
22. Johnson MB and Thiese SM. A review of overtraining syndrome-recognizing the signs and symptoms. *J Ath Train* 27:352–354, 1992.
23. Juhlin-Danfelt A, Frisk-Holmberg M, Karlson J, and Tesch P. Central and peripheral circulation in relation to muscle-fibre composition in normo- and hyper-tensive man. *Clin Sci* 56:335–340, 1979. [PubMed: 477218]
24. Laughlin MH, Newcomer SC, and Bender SB. Importance of hemodynamic forces as signals for exercise-induced changes in endothelial cell phenotype. *J Appl Physiol* 104:588–600, 2008. [PubMed: 18063803]
25. Liu X, Tsilimingras D, and Paul TK. Prevalence and changes of untreated isolated systolic hypertension among non-Hispanic black adults in the United States. *Hypertens Res* 37:685–691, 2017.
26. McArdle WD, Katch FI, and Katch VL. *Exercise Physiology*. Philadelphia: Lippincott Williams & Wilkins, 2007.
27. McKeag D, Hough D, and Thompson C. Increased incidence of elevated blood pressure in an intercollegiate football team: a screening and follow-up. *Med Sci Sports Exerc* 10:39, 1978.
28. Meeusen R, Duclos M, Fry A, Gleeson M, Nieman D, Raglin J, Rietjens G, Steinacker J, and Urhausen A Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Med Sci Sports Exerc* 45:186–205, 2013. [PubMed: 23247672]
29. Mensah GA and Brown DW. An overview of cardiovascular disease burden in the United States. *Health Aff (Millwood)* 26:38–48, 2007. [PubMed: 17211012]
30. Mitchell JH, Kaumman MP, and Iwaoto GA. The exercise pressor reflex: its cardiovascular effects, afferent mechanisms, and central pathways. *Annu Rev Physiol* 45:229–242, 1983. [PubMed: 6342515]
31. Nielsen J and Christensen DL. Glucose intolerance in the West African Diaspora: a skeletal muscle fibre type distribution hypothesis. *Acta Physiol (Oxf)* 202:605–616, 2011. [PubMed: 21382179]
32. Pandey A, Swift DL, McGuire DK, Ayers CR, Neeland IJ, Blair SN, Johannsen N, Earnest CP, Berry JD, and Church TS. Metabolic effects of exercise training among fitness-nonresponsive patients with type 2 diabetes: the HART-D study. *Diabet Care* 38:1494–1501, 2015.

33. Pesta DH, Goncalves RLS, Madiraju AK, Strasser B, and Sparks LM. Resistance training to improve type 2 diabetes working toward a prescription for the future. *Nutr Metab* 14:24, 2017.
34. Quindry J, Miller L, McGinnis G, Irwin M, Dumke C, Magal M, Triplett NT, McBride J, and Urbiztondo Z. Muscle-fiber type and blood oxidative stress after eccentric exercise. *Int J Sport Nutr Exerc Metab* 21:462–470, 2011. [PubMed: 22089306]
35. Sparks LM Exercise training response heterogeneity: physiological and molecular insights. *Diabetologia* 60:2329–2336, 2017. [PubMed: 29032385]
36. Tanner CJ, Barakat HA, Dohn GL, Pories WJ, MacDonald KG, Cunningham PR, Swanson MS, and Houmard JR. Muscle fiber type is associated with obesity and weight loss. *Am. J. Physiol* 282:E1191–E1196, 2002.
37. Van Huss WD Specific responses to heavy exercise stress. *Osteopath Ann* 5:53–66, 1977.
38. Zierath JR and Hawley JA. Skeletal muscle fiber type: influence on contractile and metabolic properties. *PLoS Biol* 2:e348, 2004. [PubMed: 15486583]

Figure 1:

(A) Mean systolic blood pressure across 20 days of high-intensity Olympic weight lifting among 8 national team members of a Middle Eastern country in preparation for an international competition. Lifters were following a periodized training program with no modifications made based on blood pressure. Mean blood pressures increased by 11 mm Hg across the 20 days. No lifter was able to achieve any of the competition goals established for the competition. (B) Mean systolic blood pressure across 28 days of high-intensity Olympic weight lifting among the same 8 national team members shown in Figure (A). A similar periodized training program was followed with the exception – if systolic blood pressure was increased +10 mm Hg prior to training (for that day), lifting intensity was decreased to no more than 50% of maximum. 10 personal records and 8 national records were achieved at the subsequent competition.

Figure 2:

Proposed model linking skeletal muscle fiber type and increased blood pressure following high-intensity exercise.

Table 1.

Continuum of exercise-induced overload that may lead to over-training syndrome.

TRAINING	PERFORMANCE	MARKERS	RECOVERY
FUNCTIONAL OVER-REACHING	Temporary reduction	Subclinical damage (e.g., muscle soreness/tightness)	Typically last several days up to weeks
NON-FUNCTIONAL OVER-REACHING	Stagnation, and in some cases a decrease	Abnormal signs/symptoms (e.g., ↓ vigor)	Can last from weeks to months
OVER-TRAINING SYNDROME	Measureable decrement	Chronic illness or injury	Recovery may take months, even years