



ACUTE EFFECTS OF CYCLING EXERCISE ON POST-EXERCISE BLOOD PRESSURE IN INDIVIDUALS WITH DOWN SYNDROME

original paper

doi: 10.1515/humo-2017-0036

MARIA EDILMA DA SILVA BEZERRA¹, LYSLEINE ALVES DE DEUS¹, THIAGO DOS SANTOS ROSA¹, EDSON EDUARDO LEAL DA SILVA², HERBERT GUSTAVO SIMÕES¹, ELAINE VIEIRA¹

¹ Postgraduate Program on Physical Education, Catholic University of Brasília, Brasília, DF, Brazil

² University Centre of the State of Pará, Belém, Brazil

ABSTRACT

Purpose. Studies have shown that even a single session of physical exercise lowers blood pressure after its completion. This phenomenon is called post-exercise hypotension (PEH) and has been considered as a non-pharmacological treatment to control blood pressure. However, there are no studies regarding the occurrence of PEH after acute exercise in individuals with Down syndrome (DS). This study aimed to analyse the occurrence of PEH in these subjects and the possible role of exercise intensity.

Methods. Ten individuals with DS, of both genders, participated in the study (age, 29 ± 7 years; body mass, 60.7 ± 9 kg; height, 1.48 ± 0.11 m; BMI, 27.6 ± 2.4 kg/m²). The volunteers randomly underwent 2 sessions of exercise on a stationary bike for 20 minutes and 1 control session. Heart rate, systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured after 15 minutes of resting, in the 20th minute of each exercise session or control, and in the 15th, 30th, and 45th minute of post-exercise recovery.

Results. Both moderate and intense exercise performed acutely increased SBP ($p < 0.001$, $p < 0.01$, respectively), with no effect on DBP in individuals with DS. Neither the moderate nor the intense exercise was enough to elicit PEH.

Conclusions. The results indicated that individuals with DS may not present PEH for the intensities, duration, and exercise mode as applied in the present investigation. While additional studies with different exercise strategies are needed, our findings contribute to the body of literature regarding the PEH responses in adults with DS.

Key words: post-exercise hypotension, Down syndrome, moderate exercise, intense exercise

Introduction

Down syndrome (DS) is considered the oldest genetic anomaly related to intellectual deficiency [1, 2]. It is characterized by congenital heart defects, autonomic deregulation, dysmorphic features, and abnormalities in lipid metabolism [3–5]. Abnormalities in lipid metabolism are frequently observed in patients with DS but coronary artery disease-related mortality is surprisingly low in this population [6].

Most individuals with DS who live in community settings have a sedentary life style and follow unhealthy diet patterns [7]. These two factors may represent some of the reasons for the higher prevalence of obesity in patients with DS compared with other individuals with intellectual disabilities [8–10]. In addition, DS patients exhibit reduced work capacity, which is attributable to an attenuated heart rate (HR) response to exercise as well as to sympathoexcitatory challenges [11, 12]. Studies

suggest that such attenuated HR and blood pressure (BP) responses in individuals with DS may be coupled with a lesser reduction in baroreflex sensitivity, which is related to a blunted sympathetic activation and to a parasympathetic withdrawal [13, 14]. On the other hand, individuals with DS have an elevated arterial stiffness [15] and risk of stroke [16] so that BP control through exercise interventions could be an interesting strategy to apply in this population.

BP is the pressure of circulating blood on the walls of blood vessels. BP control through exercise does occur both acutely and chronically [17]. Individuals with elevated BP generally benefit from the physiological changes resulting from physical exercise, acute or chronic. The acute effect of exercise on reducing the resting BP within the first post-exercise minutes and hours is called post-exercise hypotension (PEH) and may contribute to both hypertension prevention and its non-pharmacological treatment.

Correspondence address: Elaine Vieira, Postgraduate Program on Physical Education, Universidade Católica de Brasília-UCB, Campus I - QS 07 – Lote 01 – EPCT – Águas Claras – Brasília – DF CEP: 71966-700 Brazil, e-mail: elaine.vieira@ucb.br

Received: May 9, 2017

Accepted for publication: September 27, 2017

Citation: Da Silva Bezerra ME, De Deus LA, Dos Santos Rosa T, Da Silva ELL, Simões HG, Vieira E. Acute effects of cycling exercise on post-exercise blood pressure in individuals with down syndrome. Hum Mov. 2017;18(4):61–59; doi: 10.1515/humo-2017-0036.

PEH has been observed in both normotensive and hypertensive individuals; it depends on individual characteristics, with a more pronounced effect observed in hypertensive subjects [18–20]. Mean arterial BP is a functional product of cardiac output and total peripheral resistance. Despite this, a number of studies failed to elucidate any definitive mechanism(s) underlying PEH, although there is evidence that both the central and peripheral mechanisms are responsible for PEH [19, 21–27].

Although the hypotensive effect of physical exercise has been widely demonstrated in several populations, including healthy and hypertensive individuals, this effect has not been extensively studied in DS patients. Since it was suggested that individuals with DS have impaired vascular function, autonomic deregulation, and arterial stiffness, knowledge of PEH mechanisms, magnitude, and duration may give insight into autonomic nervous system function and provide a potential tool to understand BP regulation in both exercise and resting states in this population. At present, this is the first report accounting for the role of acute moderate and intense exercise on PEH in individuals with DS.

Thus, the present study aimed to analyse the occurrence of PEH in individuals with DS after acute high intensity aerobic exercise (HIAE) and moderate intensity aerobic exercise (MIAE). We hypothesized that both HIAE and MIAE, applied acutely, might reduce BP and thus alleviate and/or prevent the development of cardiovascular diseases in individuals with DS. One of the most important practical applications of the present study is to find out whether acute moderate or high intensity exercise may be a good strategy to alleviate and/or prevent the development of cardiovascular diseases in DS patients.

Material and methods

Participants

This was a cross-sectional study with a sample composed of 10 individuals with DS (3 women and 7 men), aged 29 ± 7 years. The sample involved those who met the inclusion criteria and agreed to participate in the study. Individuals using medications that could affect HR and those with cardiac problems were excluded from the study.

After receiving explanations on the terms of the research, the participants and/or their parents provided their written informed consent prior to the start of the procedure. The study was approved by the Research Ethics Committee of the Catholic University of Brasilia, Brazil (approval number 1.185.862).

Anthropometric data of groups obtained before the exercise intervention are shown in Table 1. The participants had a mean body mass index (BMI) ($27.6 \pm 2.4 \text{ kg/m}^2$) corresponding to the overweight group according to World Health Organization. Systemic arte-

Table 1. Anthropometric data of the participants

| Variable | Mean \pm SD |
|--|-----------------|
| Age (years) | 29 ± 7 |
| Body mass (kg) | 60.7 ± 9 |
| Height (m) | 1.48 ± 0.11 |
| Body mass index (kg/m^2) | 27.6 ± 2.4 |
| Resting heart rate (bpm) | 72 ± 9 |
| Resting systolic blood pressure (mm Hg) | 99 ± 9 |
| Resting diastolic blood pressure (mm Hg) | 61 ± 5 |
| Resting mean blood pressure (mm Hg) | 73 ± 6 |
| Double product (mm Hg \cdot bpm) | 7223 ± 1459 |

Data are expressed as the mean \pm standard deviation.

rial pressure values of all subjects were below the ‘optimal’ reference values (120/80 mm Hg) for individuals with normal BP. No water intake was allowed during the experiments to avoid changes in BP. Since BP fluctuates over 24 hours following the circadian rhythm, all experiments were conducted at the same time of the day, i.e. at 2:00 pm.

Experimental sessions and equipment

The participants underwent a control session sitting on a cycle ergometer, in a stationary position for 20 minutes to mimic the exercise session; BP was monitored every 15 minutes post-exercise, in accordance with the protocol. During this time, both BP and HR were measured every 5 minutes to have a mean between the values acquired.

Exercise intensity was monitored by a Polar Sport Test HR monitor. The device consists of a wireless chest strap that sends data to a monitor worn on the wrist. Exercise intensity was prescribed on the basis of the percentage of HR reserve. The HR reserve value was obtained by measuring the resting HR during the 15 minutes of pre-session assessment, while the maximal HR was predicted by considering the age of participants, in accordance with Fernhall et al. [28]. Acute exercise sessions consisted of 20 minutes of cycling at moderate intensity (MIAE, 50–70% of the HR reserve) or high intensity (HIAE, 70–89% of the HR reserve). During the 20 minutes of exercise, HR was monitored so that it did not escape from the target intensity range. After exercise, the participants remained seated for up to 45 minutes. In this interval, systolic BP (SBP) and diastolic BP (DBP) were measured every 15 minutes, reaching 45 minutes of recovery. All training sessions were performed on different days and in a random order, at least 72 hours apart, at the same time of day, at 2:00 p.m., and in a closed and conditioned environment with the temperature of 20°C.

SBP, DBP, and mean BP (MBP) were measured with an automated oscillometric device (Microlife BP 3AC1-1) before, immediately after, and in the 15th, 30th, and

45th minutes of the recovery period from exercise session. Double product (DP) was determined by multiplying SBP by HR for each moment of the measurement. Training tests were performed on an ATHLETIC brand cycle ergometer (ADVANCED 330BV). Body mass was expressed in kilograms and measured on scales (Filizola), whereas height was expressed in meters and measured with a stadiometer.

Statistical analyses

The occurrence of PEH was verified with the use of repeated measures two-way ANOVA, followed by Bonferroni's post-hoc comparisons. Data are presented as means \pm standard deviations (*SD*).

Statistical significance was accepted at $p < 0.05$. Statistical analyses were performed with the SPSS 20.0 software (SPSS Inc., USA).

Ethical approval

The research related to human use has been complied with all the relevant national regulations, institutional policies and in accordance the tenets of the Helsinki Declaration, and has been approved by the authors' institutional review board or equivalent committee.

Results

The descriptive characteristics of the participants are shown in Table 1. Baseline levels of HR, SBP, DBP, MBP, and DP were similar among participants. In the control session, no statistical difference was observed for the values of HR, SBP, DBP, MBP, or DP during the first 20 minutes in comparison with the baseline levels. These variables remained at resting levels during the recovery period (Table 2).

At the resting state, the mean HR was similar for all participants before MIAE and HIAE sessions (Figure 1A). MIAE significantly increased HR ($p < 0.0001$) when compared with resting HR levels. HIAE also increased HR ($p < 0.0001$) when compared with resting HR levels.

However, the increase in HR was higher after HIAE than after MIAE ($p < 0.0001$; Figure 1A).

Resting levels of SBP and DPB were similar before MIAE and HIAE sessions for all participants. The 20-minute MIAE increased SBP ($p < 0.001$) but did not affect DBP levels (Figure 1B and C, respectively) in comparison with the resting HR. These parameters returned to resting values in the 15th minute of post-exercise recovery and remained at these levels up to the 45th minute post-exercise. SBP was increased by HIAE in comparison with the resting levels ($p < 0.01$) but this rise in SBP reached the same levels as SBP after MIAE (Figure 1B). DBP levels were not changed after HIAE sessions. During the 45 minutes of post-exercise recovery, the SBP and DBP remained at values comparable to the pre-exercise resting state.

DP was also similar before MIAE and HIAE sessions for all participants. After the MIAE sessions, DP levels increased ($p < 0.0001$; Fig 1D) and returned to baseline values during the recovery period. HIAE sessions increased DP ($p < 0.0001$; Fig 1D), which then returned to basal levels during the recovery period. The increase in DP levels were higher after HIAE than after MIAE ($p < 0.0001$). These results suggest that the decline in BP after MIAE and HIAE was not sufficient to elicit PEH in the DS participants.

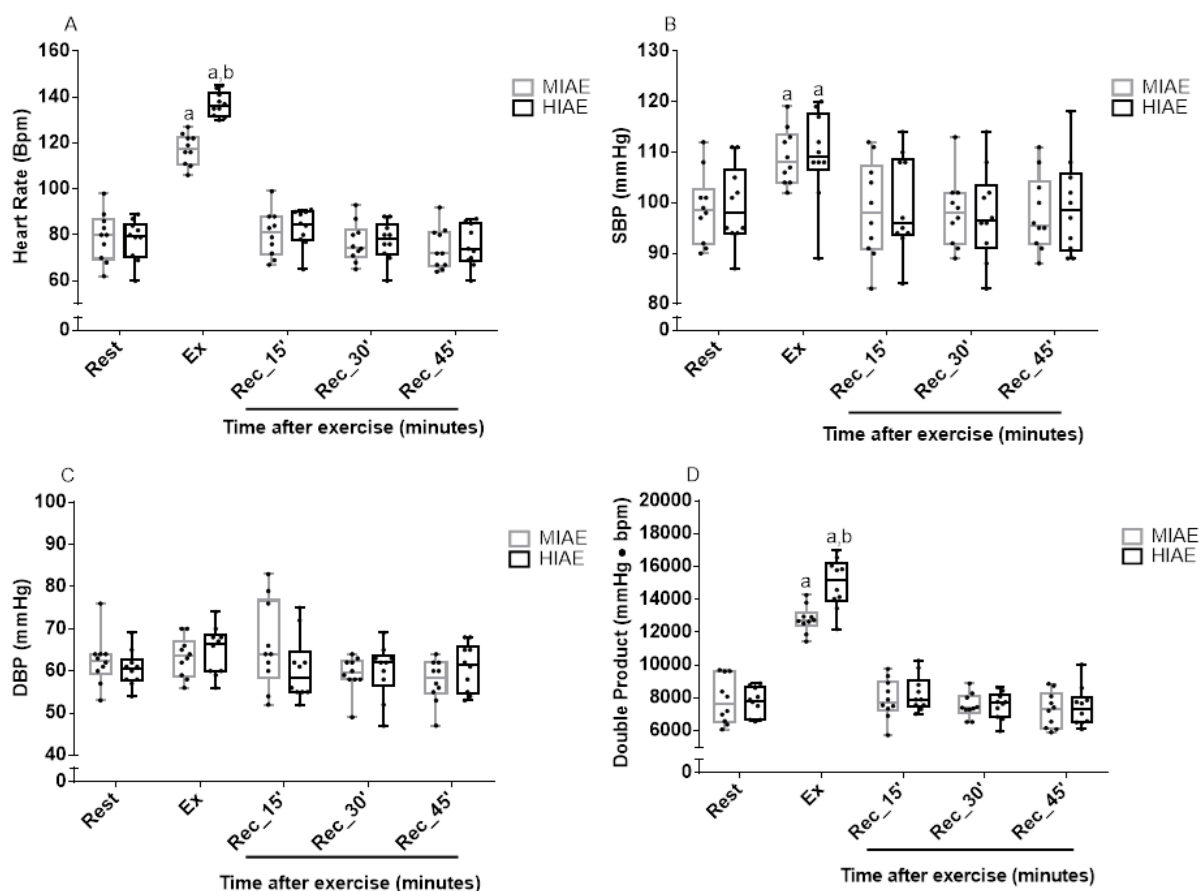
Discussion

The present study investigated the acute effects of both MIAE and HIAE on post-exercise BP responses in overweight adults with DS. Our results suggest that neither moderate nor intense exercise modalities were sufficient to elicit PEH in participants with DS. This is the first report accounting for the effects of acute exercise in overweight DS adults. The effect of physical exercise on BP in individuals with DS has not been extensively studied. Our data show that both moderate and intense acute exercises provoke an increase of HR, SBP, DBP, and DP in comparison with resting levels in participants with DS. Interestingly, BP returned to rest-

Table 2. Heart rate and blood pressure values in the control session

| Variable | Control session 20' | Recovery session | | |
|------------------------------------|------------------------|------------------|-----------------|-----------------|
| | | 15' | 30' | 45' |
| Heart rate (bpm) | 73 \pm 11 | 73 \pm 10 | 72 \pm 8 | 70 \pm 8 |
| Systolic blood pressure (mm Hg) | 97 \pm 9 | 97 \pm 7 | 98 \pm 9 | 99 \pm 8 |
| Diastolic blood pressure (mm Hg) | 60 \pm 7 | 62 \pm 8 | 60 \pm 8 | 60 \pm 6 |
| Mean blood pressure (mm Hg) | 73 \pm 7 | 74 \pm 7 | 73 \pm 7 | 73 \pm 6 |
| Double product (mm Hg \cdot bpm) | 7160 \pm 1483 | 7203 \pm 1469 | 7108 \pm 1395 | 7013 \pm 1306 |

The participants were instructed to sit on the cycle ergometer in a stationary position for 20 minutes (20', control session), which represents the time of each exercise session. They remained seated on the cycle ergometer in a stationary position also during the recovery periods of 15 minutes (15'), 30 minutes (30'), and 45 minutes (45'). Heart rate and blood pressure were measured after 20' and every 15 minutes at the recovery session after the control session. Data are expressed as means \pm standard deviations.



Heart rate: $F(4, 36) = 483.7, p < 0.0001$; $F(1, 9) = 7.205, p = 0.0250$; $F(4, 36) = 39.31, p < 0.0001$. SBP: $F(4, 36) = 13.12, p < 0.0001$; $F(1, 9) = 0.1872, p = 0.6755$; $F(4, 36) = 0.09031, p = 0.9849$. DBP: $F(4, 36) = 5.819, p = 0.0010$; $F(1, 9) = 0.03512, p = 0.8555$; $F(4, 36) = 2.343, p = 0.0733$. Double product: $F(4, 36) = 370.1, p < 0.0001$; $F(1, 9) = 9.189, p = 0.0142$; $F(4, 36) = 10.17, p < 0.0001$.

SBP – systolic blood pressure, DBP – diastolic blood pressure, Rest – resting, Ex – exercise, Rec – recovery period, MIAE – moderate intensity aerobic exercise, HIAE – high intensity aerobic exercise, a – significant difference compared with resting state, b – difference between groups within the same period of time

Figure 1. Heart rate (A), systolic blood pressure (B), diastolic blood pressure (C), and double product (D) in moderate and high intensity aerobic exercise among individuals with Down syndrome. Values are expressed as means \pm standard deviation. The F and *p* values are the following: effect of time, effect between groups, and interaction time versus group, respectively

ing levels after its reduction by MIAE and HIAE and remained at these levels up to 45 minutes post-exercise. In another study, both aerobic and resistance training reduced SBP, DBP, and MBP levels after 12 weeks of training in young individuals with DS but PEH was not analysed [29]. In addition, whereas no differences in SBP or DBP in the 3rd minute after maximal exercise were found in individuals with DS, the arterial stiffness responses to maximal exercise in persons with DS were blunted, suggesting an impaired vascular function [15]. Another study, employing a handgrip test in individuals with DS, revealed an attenuated HR and SBP response that could be explained by a blunted parasympathetic withdrawal, suggesting reduced baroreflex control of HR and SBP in individuals with DS [4].

Knowledge of PEH is potentially useful in designing strategies against hypertension as well as allowing a further understanding of BP regulation in both health and disease. The PEH responses in individuals with DS are more related with BP regulation since most individuals

with DS do not exhibit hypertension [30] or exhibit low BP [31].

Although we do not know the reason for an attenuated PEH response in our study, we can speculate that the lack of PEH observed in the participants could be due to obesity-related factors, impaired vascular function, or autonomic deregulation. It has been shown that individuals with DS exhibit blunted arterial stiffness responses to maximal exercise, which potentially reflects a diminished vascular reserve. In addition, obesity and particularly VO_2 peak were related with the impaired vascular function observed in these individuals [15]. Regarding this, a study showed that HR and BP responses to exercise in individuals with DS were attributed to an autonomic dysfunction and associated with obesity. It has also been shown that DS subjects display attenuated HR and SBP responses to handgrip exercise. This was explained by blunted parasympathetic withdrawal and alterations in both parasympathetic (vagal) and sympathetic responses, suggesting reduced

baroreflex control of HR and SBP in individuals with DS [4]. In addition, the risk of major cardiovascular events in people with DS was studied in the Australian state of Victoria in years 1993–2010 [16]. The research proved a higher prevalence of congenital heart disease, cardiac arrhythmia, dementia, pulmonary hypertension, diabetes, and sleep apnoea in the DS group compared with the group without DS. The mentioned study also concluded that DS was associated with a higher risk of stroke in all ages. Thus, DS individuals would benefit from PEH because of more cardiovascular risk factors and a higher risk of obesity. In this context, many studies found positive results on cardiopulmonary capacity and general physical fitness in these individuals [32–34].

One of the limitations of the present study was the low number of participants and the use of both genders. It would be interesting to check whether there is a gender-related difference in the effect of acute exercise on BP in this population or not. Another limitation is the impossibility of blood collection in this population, which could provide some information about the potential mechanism. The time of the day at which the experiments were conducted could be another factor affecting the results as the systolic hypotensive effect is greater after morning exercise than after evening exercise when circadian variations are considered [35]. Future studies are needed to evaluate the occurrence of PEH in different times of the day in DS individuals.

Thus, our results will certainly contribute to a better understanding of the different effects of physical exercise on BP regulation in adult individuals with DS.

Conclusions

In conclusion, our results provide new knowledge regarding the effect of acute exercise on PEH in overweight adults with DS. The lack of PEH for the intensities, duration, and exercise modes applied in the present study suggests that additional studies with different exercise modes, intensities, and durations are needed. Future research directions also include further exploration of the potential benefits of physical exercise on the regulation of vascular function and targeted interventions in individuals with DS.

Disclosure statement

No author has any financial interest or received any financial benefit from this research.

Conflict of interest

Authors state no conflict of interest.

Acknowledgements

The authors thank the Brazilian Federal Foundation, the National Council for Scientific and Technological Development, and the Science without Borders programme. We thank Gerardo Gabriel Mirizio for the English revision of the manuscript.

References

1. Kasari C, Freeman SF. Task-related social behavior in children with Down syndrome. *Am J Ment Retard.* 2001; 106(3):253–264; doi:10.1352/0895-8017(2001)106<0253:TR SBIC>2.0.CO;2.
2. Kasari C, Freeman SF, Hughes MA. Emotion recognition by children with Down syndrome. *Am J Ment Retard.* 2001;106(1):59–72; doi: 10.1352/0895-8017(2001)106<0059:ERBCWD>2.0.CO;2.
3. Dörner K, Gaethke AS, Tolksdorf M, Schumann KP, Gustmann H. Cholesterol fractions and triglycerides in children and adults with Down's syndrome. *Clin Chim Acta.* 1984;142(3):307–311; doi: 10.1016/0009-8981(84) 90267-5.
4. Figueroa A, Collier SR, Baynard T, Giannopoulou I, Goulopoulou S, Fernhall B. Impaired vagal modulation of heart rate in individuals with Down syndrome. *Clin Auton Res.* 2005;15(1):45–50; doi: 10.1007/s10286-005-0235-1.
5. Vis JC, Duffels MG, Winter MM, Weijerman ME, Cobben JM, Huisman SA, et al. Down syndrome: a cardiovascular perspective. *J Intellect Disabil Res.* 2009;53(5):419–425; doi: 10.1111/j.1365-2788.2009.01158.x.
6. Baird PA, Sadovnick AD. Causes of death to age 30 in Down syndrome. *Am J Hum Genet.* 1988;43(3):239–248.
7. Van den Akker M, Maaskant MA, van der Meijden RJ. Cardiac diseases in people with intellectual disability. *J Intellect Disabil Res.* 2006;50(Pt 7):515–522; doi:10.1111/j.1365-2788.2006.00797.x.
8. Bell AJ, Bhate MS. Prevalence of overweight and obesity in Down's syndrome and other mentally handicapped adults living in the community. *J Intellect Disabil Res.* 1992;36(Pt 4):359–364; doi: 10.1111/j.1365-2788.1992.tb00534.x.
9. Melville CA, Cooper SA, McGrother CW, Thorp CF, Collacott R. Obesity in adults with Down syndrome: a case-control study. *J Intellect Disabil Res.* 2005;49(Pt 2): 125–133; doi: 10.1111/j.1365-2788.2004.00616.x.
10. Prasher VP. Overweight and obesity amongst Down's syndrome adults. *J Intellect Disabil Res.* 1995;39(Pt 5): 437–441; doi: 10.1111/j.1365-2788.1995.tb00548.x.
11. Baynard T, Pitetti KH, Guerra M, Unnithan VB, Fernhall B. Age-related changes in aerobic capacity in individuals with mental retardation: a 20-yr review. *Med Sci Sports Exerc.* 2008;40(11):1984–1989; doi: 10.1249/MSS.0b013e31817f19a1.
12. Bunsawat K, Goulopoulou S, Collier SR, Figueroa A, Pitetti KH, Baynard T. Normal HR with tilt, yet autonomic dysfunction in persons with Down syndrome. *Med Sci Sports Exerc.* 2015;47(2):250–256; doi: 10.1249/MSS.0000000000000411.
13. Fernhall B, Mendonca GV, Baynard T. Reduced work capacity in individuals with Down syndrome: a consequence of autonomic dysfunction? *Exerc Sport Sci Rev.* 2013;41(3):138–147;doi:10.1097/JES.0b013e318292f408.
14. Fernhall B, Otterstetter M. Attenuated responses to sympathoexcitation in individuals with Down syndrome. *J Appl Physiol.* 2003;94(6):2158–2165; doi: 10.1152/jap-physiol.00959.2002.
15. Hu M, Yan H, Ranadive SM, Agiovlasitis S, Fahs CA, Atiq M, et al. Arterial stiffness response to exercise in persons with and without Down syndrome. *Res Dev Disabil.* 2013;34(10):3139–3147;doi:10.1016/j.ridd.2013.06.041.

16. Sobey CG, Judkins CP, Sundararajan V, Phan TG, Drummond GR, Srikanth VK. Risk of major cardiovascular events in people with Down syndrome. *PLoS One*. 2015;10(9):e0137093; doi: 10.1371/journal.pone.0137093.
17. Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation*. 2008;117(25):3171–3180; doi: 10.1161/CIRCULATIONAHA.107.730366.
18. Arazi H, Asadi A, Rahimzadeh M, Moradkhani AH. Post-plyometric exercise hypotension and heart rate in normotensive individuals: influence of exercise intensity. *Asian J Sports Med*. 2013;4(4):235–240; doi: 10.5812/asjms.34240.
19. Forjaz CL, Tinucci T, Ortega KC, Santaella DF, Mion D Jr, Negrão CE. Factors affecting post-exercise hypotension in normotensive and hypertensive humans. *Blood Press Monit*. 2000;5(5–6):255–262; doi: 10.1097/00126097-200010000-00002.
20. Simões GC, Moreira SR, Kushnick MR, Simões HG, Campbell CS. Postresistance exercise blood pressure reduction is influenced by exercise intensity in type-2 diabetic and nondiabetic individuals. *J Strength Cond Res*. 2010;24(5):1277–1284; doi: 10.1519/JSC.0b013e3181d67488.
21. Chen CY, Bonham AC. Postexercise hypotension: central mechanisms. *Exerc Sport Sci Rev*. 2010;38(3):122–127; doi: 10.1097/JES.0b013e3181e372b5.
22. Floras JS, Wesche J. Haemodynamic contributions to post-exercise hypotension in young adults with hypertension and rapid resting heart rates. *J Hum Hypertens*. 1992;6(4):265–269.
23. Forjaz CL, Cardoso CG Jr, Rezk CC, Santaella DF, Tinucci T. Postexercise hypotension and hemodynamics: the role of exercise intensity. *J Sports Med Phys Fitness*. 2004;44(1):54–62.
24. Hamer M, Boutcher SH. Impact of moderate overweight and body composition on postexercise hemodynamic responses in healthy men. *J Hum Hypertens*. 2006;20(8):612–617; doi: 10.1038/sj.jhh.1002035.
25. Legramante JM, Galante A, Massaro M, Attanasio A, Raimondi G, Pigozzi F, et al. Hemodynamic and autonomic correlates of postexercise hypotension in patients with mild hypertension. *Am J Physiol Regul Integr Comp Physiol*. 2002;282(4):R1037–R1043; doi: 10.1152/ajp-regu.00603.2001.
26. Lockwood JM, Pricher MP, Wilkins BW, Holowatz LA, Halliwill JR. Postexercise hypotension is not explained by a prostaglandin-dependent peripheral vasodilation. *J Appl Physiol*. 2005;98(2):447–453; doi: 10.1152/jap-physiol.00787.2004.
27. Lockwood JM, Wilkins BW, Halliwill JR. H1 receptor-mediated vasodilatation contributes to postexercise hypotension. *J Physiol*. 2005;563(Pt 2):633–642; doi: 10.1113/jphysiol.2004.080325.
28. Fernhall B, McCubbin JA, Pitetti KH, Rintala P, Rimmer JH, Millar AL, et al. Prediction of maximal heart rate in individuals with mental retardation. *Med Sci Sports Exerc*. 2001;33(10):1655–1660; doi: 10.1097/00005768-200110000-00007.
29. Seron BB, Goessler KF, Modesto EL, Almeida EW, Greguol M. Blood pressure and hemodynamic adaptations after a training program in young individuals with Down syndrome. *Arq Bras Cardiol*. 2015;104(6):487–491; doi: 10.5935/abc.20150033.
30. Pucci F, Machado G, Solera E, Cenovicz F, Arruda C, Braga C, et al. Blood pressure levels and body mass index in Brazilian adults with Down syndrome. *Sao Paulo Med J*. 2016;134(4):330–334; doi: 10.1590/1516-3180.2016.0057180316.
31. Morrison RA, McGrath A, Davidson G, Brown JJ, Murray GD, Lever AF. Low blood pressure in Down's syndrome. A link with Alzheimer's disease? *Hypertension*. 1996;28(4):569–575; doi: 10.1161/01.HYP.28.4.569.
32. Lin HC, Wuang YP. Strength and agility training in adolescents with Down syndrome: a randomized controlled trial. *Res Dev Disabil*. 2012;33(6):2236–2244; doi: 10.1016/j.ridd.2012.06.017.
33. Ordonez FJ, Rosety M, Rosety-Rodriguez M. Influence of 12-week exercise training on fat mass percentage in adolescents with Down syndrome. *Med Sci Monit*. 2006;12(10):CR416–CR419.
34. Shields N, Taylor NF, Wee E, Wollersheim D, O'Shea SD, Fernhall B. A community-based strength training programme increases muscle strength and physical activity in young people with Down syndrome: a randomised controlled trial. *Res Dev Disabil*. 2013;34(12):4385–4394; doi: 10.1016/j.ridd.2013.09.022.
35. De Brito LC, Rezende RA, da Silva Junior ND, Tinucci T, Casarini DE, Cipolla-Neto J, et al. Post-exercise hypotension and its mechanisms differ after morning and evening exercise: a randomized crossover study. *PLoS One*. 2015;10(7):e0132458; doi: 10.1371/journal.pone.0132458.