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Effects of Exercise on Hemorheological Parameters of Young Nigerian Smokers

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Aim: Regular physical exercise is associated with reduced risk of cardiovascular diseases. In this study, the hypothesis that acute submaximal exercise has similar effects on rheological parameters of smokers and non-smokers was tested.

Materials and Methods: Thirty-three male university undergraduates comprised of 18 smokers and 15 non-smokers were studied. All the subjects underwent submaximal exercise on cycloergometer for 30 minutes. Blood for hemorheological parameters was collected 30 minutes before and after exercise. Samples were analyzed by standard methods.

Results: A total of 33 undergraduates were studied. The hematocrit, plasma viscosity and plasma fibrinogen were significantly higher in smokers than non-smokers ($P < 0.01$). Post- exercise plasma viscosity was significantly higher than pre-exercise value in non-smokers ($P < 0.0001$). Hematocrit, plasma viscosity and plasma fibrinogen were also significantly higher post-exercise in smokers ($P < 0.01$, $P < 0.01$ and $P < 0.05$, respectively). The white cell count and whole blood viscosity were 17.8% and 9% higher, respectively, than pre-exercise values in smokers.

Conclusions: The study confirmed an acute rise in rheological parameters in smokers and non-smokers following submaximal exercise; however, smokers are more liable to cardiovascular effects of acute intense exercise, especially those with sedentary lifestyle.

Key Words: Exercise, hemorheological parameters, smokers

Nijeryalı Genç Sigara Tiryakilerinde Hafif Egzersizin Kan Parametreleri Üzerine Etkisi

Amaç: Düzenli fizik aktivitesi kalp damar hastalıkları riskini azaltır. Bu çalışmada, normalin altındaki akut egzersizin de aynı etkilere sahip olduğu hipotezi sigara içen ve içmeyenlerde test edilmiştir.

Yöntem ve Gereç: 18 sigara içen ve 15 sigara içmeyen 33 erkek üniversite öğrencisi çalışmaya dahil edildi. Öğrencilerin tümüne 30 dk süreyle sikloergometrede düşük düzeyde egzersiz yaptırıldı. Egzersizden 30 dakika önce ve sonra hemorolojik parametreleri ölçmek için kan örnekleri alındı, standart yöntemlerle analiz edildi.

Bulgular: 33 öğrenci çalışmaya alındı. Hemotokrit, plazma vizkositesi ve plazma fibrinojen düzeyleri sigara içenlerde anlamlı ölçüde daha yüksekti. Sigara içmeyenlerde egzersiz sonrası plazma vizkositesi egzersiz öncesinden daha yüksekti. Sigara içenlerde de egzersiz sonrası hemotokrit, plazma viskozitesi ve plazma fibrinojen düzeyleri daha yüksekti. Sigara içenlerde kan, beyaz küre ve vizkosite değerleri de egzersiz öncesi değerlere oranla sırasıyla % 17.8 ve % 9 daha yüksekti.

Sonuç: Bu çalışma hafif egzersizi takiben kan değerlerinde her iki grupta da akut bir yükselme olduğunu gösterdi. Ancak, sigara içenler ve özellikle de sedanter bir yaşamı olanlar hafif egzersizin kardiyovasküler etkilerine daha dayanıksızdı.

Anahtar Sözcükler: Egzersiz, kan değerleri, sigara içimi

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Introduction

Regular physical exercise is associated with reduced risk of cardiovascular diseases (CVD), although the mechanism is still unclear. Protective effects of exercise have been demonstrated for both coronary heart disease (1,2) and peripheral artery diseases (3,4). Protection is associated with metabolic improvements, a development which suggests that the beneficial effects of exercise may be mediated at least in part by reversal of the increase in conventional risk factors that are commonly associated with sedentary lifestyle (5). Lack of physical fitness is an independent risk factor for cardiovascular mortality (6); a dose-dependent relationship has been demonstrated between regular physical exercise and reduced risk of CVD (1). Alteration in plasma fibrinogen concentration has been suggested to be a likely mechanism through which the effects of exercise on cardiovascular risk may be partly mediated (7). Chronic exercise has been demonstrated to lower plasma fibrinogen levels; furthermore, an intense exercise generates an acute phase rise in plasma fibrinogen, a response that has been suggested to be influenced by G/A polymorphism of the β fibrinogen gene (8-10). Plasma fibrinogen has been reported to be higher in smokers than non-smokers (11,12). Intervention studies in the field of exercise rheology are few and the conclusions are not consistent (13). In this study, we tested the hypothesis that moderate physical exercise exerts the same effects on rheological parameters of smokers and non-smokers.

Materials and Methods

Subjects: Thirty-three male university undergraduates comprised of 18 smokers and 15 non-smokers who gave informed consent to participate actively in the study were included. Smokers were defined as people who regularly smoked at least one cigarette per day for at least five years. None of the subjects had clinical evidence of disease(s). Data on age, smoking habits and medical history were obtained by interviewer-administered questionnaire. Weight of participants was measured on a balanced scale with shoes off. Heavy outer garments were removed to minimize error. None of the subjects had been involved in any regular physical exercise for more than a year before the study.

All subjects underwent a standardized submaximal exercise session on cycloergometer for 30 minutes. It consisted of a 30-minute cycling session with the first 10 minutes being a warm–up period followed by a 20-minute plateau at 85% of the theoretical maximal heart rate. Pedal speed was maintained at 60 rpm by the subjects. Blood samples were collected at 9 a.m. before exercise and 30 minutes after the exercise.

Blood Samples: Five milliliters of blood was collected from the cubital vein using a plastic syringe while applying tourniquet lightly over the arm until blood flow was established. Stasis was avoided during blood collection to prevent activation of clotting factors. 4.5 ml of blood was transferred into a plastic tube containing 0.5 ml of 3.8% sodium citrate. Blood and anticoagulant were mixed gently but thoroughly. Plasma was separated by centrifugation at 2,500 g for 15 minutes to obtain platelet poor plasma. The plasma fibrinogen was estimated using the clot weight method of Ingram (14). Blood for hematocrit, white cell count, plasma viscosity and whole blood viscosity determination was collected in 2 ml of 0.77 mol/L ethylenediamine-tetraacetic acid. Plasma viscosity was measured by the method of Reid and Ugwu (15). The white cell count and hematocrit were determined by standard methods (16).

Statistical Analysis: Instat graph pag^{Tm} was used for all statistical analyses. Alternate T-test was used to assess differences between the two groups (smokers and non-smokers) at baseline and after exercise. Two-tailed t test was used to test for differences before and after exercise. Hemorheological parameters before and after exercise in smokers and non-smokers were expressed as mean ± standard deviation. Significance was established as $P < 0.05$.

Results

A total of 33 undergraduates of the University of Benin were studied. They were grouped into non-smokers (n = 15) (mean age 23.8 ± 1.3 years, height 180.9 ± 1.2 cm, weight 77.9 ± 1.5 kg and body mass index 23.7 ± 0.3 kg/m^2) and smokers (n = 18) (mean age 20.7 ± 1.5, height 184.7 ± 2.2 cm, weight 80.9 ± 2.2 kg and body mass index 23.2 ± 0.5 kg/m^2).

Table 1 shows the clinical and rheological parameters of both smokers and non-smokers. The hematocrit, plasma viscosity and plasma fibrinogen concentration were significantly higher in smokers than non-smokers. Table 2 compares the rheological parameters pre- and

Table 1. Clinical and rheological data of smokers and non-smokers.

Parameter	Non-smokers (n = 15)	Smokers (n = 18)	P-value
Age (years)	23.9 ± 1.3	20.7 ± 1.5	N/s
Weight (kg)	77.9 ± 1.5	80.9 ± 2.2	N/s
Height (cm)	180.9 ± 1.2	184.7 ± 2.2	N/s
BMI (kg/m^2)	23.7 ± 0.3	23.2 ± 0.5	N/s
Hematocrit (%)	40.2 ± 4.65	43.25 ± 3.01	<0.05
Total WBC (x10^9/L)	3.4 ± 0.74	3.7 ± 1.4	>0.05
WBV (mPa.s)	3.52 ± 0.27	3.79 ± 0.58	>0.05
Plasma viscosity (mPa.s)	1.25 ± 0.05	1.37 ± 0.16	<0.01
Plasma fibrinogen (g/L)	1.66 ± 0.42	2.21 ± 0.62	<0.01

BMI: Body mass index. WBC: White blood count. WBV: Whole blood viscosity. N/s: Not significant.

Table 2. Effect of exercise on blood rheology in non-smokers.

Parameter	Pre-exercise (n = 15)	Post-exercise (n = 15)	% Increase
Packed cell volume (%)	40.20 ± 4.65	40.60 ± 3.36	1N/s
White blood count (x 10^9/L)	3.24 ± 0.88	3.4 ± 0.74	4.9N/s
Whole blood viscosity (mPa.s)	3.52 ± 0.27	3.92 ± 0.68	11.4N/s
Plasma viscosity (mPa.s)	1.25 ± 0.05	1.42 ± 0.01	13.6****
Plasma fibrinogen (g/L)	1.56 ± 0.68	1.66 ± 0.42	6.4N/s

N/s: Not significant. **** P < 0.0001.

post-exercise in non-smokers. Post-exercise plasma viscosity was significantly higher than pre-exercise value (P < 0.0001). Whole blood viscosity, hematocrit and plasma fibrinogen were also higher, but the differences between pre- and post-exercise values were not significant. The effect of exercise on blood rheology in smokers is shown in Table 3. The hematocrit, plasma viscosity, and plasma fibrinogen concentration were all significantly higher immediately post-exercise (P < 0.01, P < 0.01 and P < 0.05, respectively). The white cell count and whole blood viscosity were also higher than pre-exercise values (17.8% and 9%, respectively), but the differences were not significant. Comparison of the hemorheological parameters of non-smokers and smokers post-exercise (Table 4) revealed that the hematocrit and fibrinogen were still significantly higher in smokers than non-smokers after exercise (P < 0.001). The white cell count and plasma viscosity were also higher (P < 0.01 and P < 0.05, respectively).

Discussion

There is a body of literature on blood rheology and exercise (1-5,17), but the conclusions have not been univocal (13). In this study, we examined the hypothesis that exercise has the same effect on rheological parameters of smokers and non-smokers. We found that submaximal exercise is associated with alterations in blood rheology in both smokers and non-smokers. Triphasic rheological changes had been described following exercise (18). In this study, we found a 7.2% increase in hematocrit above basal level after exercise in

Table 3. Effect of exercise on blood rheology in smokers.

	Pre-exercise (n = 18)	Post-exercise (n = 18)	Increase (%)[b]
[a]Hct (%)	43.25 ± 3.01	46.38 ± 3.61	7.2**
WCC (x 10^9/L)	3.7 ± 1.4	4.36 ± 1.3	7.8N/s
WBV (mPa.s)	3.79 ± 0.58	4.13 ± 0.71	9N/s
PV (mPa.s)	1.37 ± 0.16	1.56 ± 0.21	13.9**
PFC (g/L)	2.21 ± 0.62	2.62 ± 0.50	18.6*

[a] Hct: Hematocrit. WCC: White cell count. WBV: Whole blood viscosity. PV: Plasma viscosity. PFC: Plasma fibrinogen concentration.
[b] ** P < 0.01. * P < 0.05. N/s: Not significant.

Table 4. Effect of exercise on hemorheological parameters of smokers and non-smokers.

	Post-exercise Non-smokers (n = 15)	Post-exercise Smokers (n = 18)
[a]Hct (%)	40.60 ± 3.36	[b]46.38 ± 3.61****
WCC (x 109)	3.24 ± 0.88	4.36 ± 1.3**
WBV (mPa.s)	3.92 ± 0.68	4.13 ± 0.71N/s
PV (mPa.s)	1.42 ± 0.10	1.56 ± 0.21*
PFC (g/L)	1.66 ± 0.42	2.62 ± 0.5****

[a] Hct: Hematocrit. WCC: White cell count. WBV: Whole blood viscosity. PV: Plasma viscosity. PFC: Plasma fibrinogen concentration.
[b] N/s: Not significant. * P < 0.05. ** P < 0.01. **** P < 0.0001.

smokers, and this agrees with previous studies where both maximal and submaximal exercise sessions have been reported to increase hematocrit (19). These changes have been described as hemoconcentration - a complex mechanism resulting from redistribution of red cells in the vascular bed, and entrapment of water into muscles among others (20,21). The increase in plasma viscosity found in this study is in agreement with the findings of Peyreigne et al. (22), which attributed the rise in plasma viscosity to water loss from sweating as a result of the increase in growth hormone secretion during exercise.

The 9% increase in whole blood viscosity in smokers and 11.4% increase in non-smokers could be explained by the corresponding rise in hematocrit, plasma fibrinogen and plasma viscosity.

The increase in white blood cell count observed in both smokers and non-smokers following exercise is in line with existing literature where both white cell activation and oxidant stress were implicated in the hemorheological effects of exercise (23,24). The increase in the number of leukocytes after exercise may be explained by the increase in blood flow which recruits leukocytes from the marginal pool and/or hormonal changes, which are likely to be mediated by β adrenergic receptors. In addition, a decrease in leukocyte filterability, an indication of leukocyte activation during exercise, has also been reported as a likely mechanism for the rise in white cell count (23,25).

Fibrinogen is an acute phase protein that is synthesized by the liver in response to cytokines (26). Whereas regular exercise may reduce fibrinogen levels, severe exercise might paradoxically cause an acute-phase rise, simulating a procoagulant effect. Thus, the beneficial effects of regular exercise may be lost if the exercise is very intense (27,28), and this may acutely elevate the risk of myocardial infarction, especially in those with a sedentary lifestyle (29). The rise in plasma fibrinogen in non-smokers (6.4%) and smokers (18.6%), however, differs from the earlier study of Montgomery et al. (10), in which an acute rise in plasma fibrinogen was found on days 1 (22.7%) and 2 (37.1%) of a 10-week training exercise. This acute phase increase in levels was suggested to be influenced by the G/A polymorphism of the β gene. It is possible that our post-exercise samples were taken at the point when fibrinogen was just beginning to increase.

While all the rheological parameters were acutely increased following exercise in smokers, the effects seemed to be greater on plasma viscosity (13.9%), white cell count (17.8%), and fibrinogen (18.6%). In non-smokers, the effects were more pronounced in whole blood viscosity (11.4%), plasma viscosity (13.6%) and fibrinogen (6.4%). This observation indicates that blood rheology is altered following exercise in both smokers and non-smokers. However, fibrinogen is more adversely affected in smokers than non-smokers. This may not be totally unexpected, as fibrinogen has been reported to be higher in smokers (11,12). It could therefore be argued that smokers are more likely to suffer from the cardiovascular effects of acute intense exercise than non-smokers.

Furthermore, the significant change in hematocrit immediately after exercise in smokers is noteworthy, as a negative correlation between hematocrit and physical fitness had been reported (30). This again portends an adverse immediate post-exercise effect in smokers.

In conclusion, although the theory of acute rise in rheological parameters following submaximal or maximal exercise has been debated, this study, despite its small sample size, has confirmed an acute rise in hemorheological parameters following a moderate exercise in smokers and non-smokers. The higher rise in hematocrit (7.2%) and fibrinogen (18.6%) in smokers compared to non-smokers may indicate that smokers are more liable to cardiovascular effects of acute intense exercise, especially in those with a sedentary lifestyle.

References

1. Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. AM J Epidemiol 1990; 132: 612-628.
2. Morris JN. Exercise in the prevention of coronary heart disease: today's best buy in public health. Med Sci Sports Exerc 1994; 26: 807-814.
3. Ernst E. Physical exercise for peripheral vascular disease – a review. VASA 1987; 16: 227-231.
4. Ernst E. Peripheral vascular disease, benefits of exercise. Sports Med 1991; 12: 149-151.
5. Wood P. Physical activity, diet, and health: independent and interactive effects. Med Sci Sports Exerc 1994; 26: 838-843.
6. Slattery ML, Jacobs DR. Physical fitness and cardiovascular disease mortality. The US Railroad Study. Am J Epidemiol 1999; 127: 571-580.
7. Simonsick EM, Lafferty ME, Phillips CL, Mendes de Leon CF, Kasl SV, Seeman TE et al. Risk due to inactivity in physically capable older adults. Am J Public Health 1993; 83: 1443-1450.
8. Morris JN, Heady JA, Raffle PAB, Roberts CG, Parks JW. Coronary heart disease and physical activity of work. Lancet 1953; 2: 1111-1120.
9. Ernst E. Regular exercise reduces fibrinogen levels: a review of longitudinal studies. Br J Sports Med 1993; 27: 175-176.
10. Montgomery HE, Clarkson P, Nwose OM, Mikailidis DP, Jagroop IA, Dollery C et al. The acute rise in plasma fibrinogen concentration with exercise is influenced by the G-453-A polymorphism of the beta-fibrinogen gene. Arterioscler Thromb Vasc Biol 1996; 16(3): 386–391.
11. Haustein KO, Krause J, Haustein H, Rasmussen T, Cort N. Effects of cigarette smoking or nicotine replacement on cardiovascular risk factors and parameters of haemorheology. J Intern Med 2002; 252(2): 130-139.
12. Awodu OA, Famodu AA, Ajayi OI, Borgu MZ. Effects of smoking on haemorheological parameters of Africans. Haema 2005; 8(1): 99-102.
13. Moxley RT, Brakman P, Astrup T. Resting levels of fibrinolysis in blood in inactive and exercising men. J Appl Physiol 1970; 28: 549-552.
14. Ingram GIC. A suggested schedule for the rapid investigation of acute haemostatic failure. J Clin Path 1961; 14: 356-360.
15. Reid HL, Ugwu AC. A simple technique of rapid determination of blood viscosity. Nig J Physiol Sci 1987; 3: 45-48.
16. Dacie JV, Lewis SM, editors. Practical haematology. 8th ed. Edinburgh: Churchill Livingstone; 1994. pp. 326-28.
17. Ernst E, Matrai A, Schmolz C, Magyarosy I. Dose-effect relationship between smoking and blood rheology. Br J Haematol 1987; 65: 485-487.
18. Brun JF, Khaled S, Raynaud E, Bouix D, Micallef JP, Orseti A. The triphasic effects of exercise on blood rheology: which relevance to physiology and pathophysiology? Clin Hemorheol Microcirc 1998; 19(2): 89-104.
19. Brun JF, Frons C, Supparo I, Mallard C, Orsetti A. Could exercise-induced increase in blood viscosity at high shear rate be entirely explained by haematocrit and plasma viscosity changes? Clin Hemorheol 1993; 13: 187-189.
20. Sjogaard G, Adams RP, Saltin B. Water and ion shifts in skeletal muscle of humans with intense dynamic knee extension. Am J Physiol 1985; 248: R190-R196.

21. Martins e Silva J. Blood rheological adaptation to physical exercise. Rev Port Hemorrel 1988; 2: 63-67.

22. Peyreigne C, Bouix D, Micallef JP, Mercier J, Bringer J, Prefaut C et al. Exercise-induced growth hormone secretion and haemorheology during exercise in elite athletes. Clin Hemorheol Microcirc 1998; 19: 169-176.

23. Ernst E, Marshall M, Reduced leucocyte filterability after acute physical stress. Clin Hemorheol 1991; 11: 129-132.

24. Gurcan N, Erbas D, Ergen E, Bilgerhan A, Dundar S, Aricioglu A et al. Changes in blood haemorheological parameters and submaximal exercise in trained and untrained subjects. N Physiol Res 1998; 47: 23-27.

25. Brun JF. Exercise haemorheology as a three acts play with metabolic actors: Is it of clinical relevance? Clin Hemorheol Microcirc 2002; 26: 155-174.

26. Dalmon J, Laurent M, Courtois G. The human beta fibrinogen promotor contains a hepatocyte nuclear factor 1-dependent interleukin-6-responsive element. Mol Cell Biol 1993; 13: 1183-1193.

27. Arai M, Yorifuji H, Ikematsu S, Nagasawa H, Fujimaki M, Fukutake K et al. Influences of strenuous exercise (triathlon) on blood coagulation and fibrinolytic system. Thromb Res 1990; 57: 465-471.

28. Paffenbarger RS, Hyde RT, Wing AL, Hsieh C. Physical activity, all-cause mortality, and longevity of college alumni. N Engl J Med 1986; 314: 605-613.

29. Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Mullers JE. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. N Engl J Med 1993; 329: 1678-1683.

30. Brun JF, Bouchahda C, Chaze D, Aissa Benhadddad A, Micallef JP, Mercier J. The paradox of haematocrit in exercise physiology: which is the "normal" range from an hemorheologist's viewpoint? Clin Hemorheol Microcirc 2000; 22: 287-303.