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ACUTE CARDIOVASCULAR RESPONSE TO PRE-PRANDIAL AND POSTPRANDIAL EXERCISE IN ACTIVE MEN

RESPOSTA CARDIOVASCULAR AGUDA AO EXERCÍCIO PRÉ E PÓS-PRANDIAL EM HOMENS ATIVOS

RESPUESTA CARDIOVASCULAR AGUDA AL EJERCICIO PREPRANDIAL Y POSTPRANDIAL EN HOMBRES ACTIVOS

Márcio Rabelo Mota[1]
(Physical Education Professional)
Sandro Nobre Chaves[2]
(Physical Education Professional)
Maurílio Tiradentes Dutra[2]
(Physical Education Professional)
Ricardo Jacó de Oliveira[2]
(Physical Education Professional)
Renata Aparecida Elias Dantas[1]
(Physical Education Professional)
Filipe Dinato de Lima[2]
(Physical Education Professional)

1. Centro Universitário de Brasília (UniCEUB), Faculdade de Ciências da Educação e da Saúde, Brasília, DF, Brazil.
2. Universidade de Brasília (UnB), Faculdade de Educação Física, Brasília, DF, Brazil.

Correspondência:
Maurílio Tiradentes Dutra
Faculdade de Educação Física.
Campus Universitário Darcy Ribeiro, Asa Norte, Brasília, Distrito Federal, Brazil. 70910-970.
mauriliotiradentes@gmail.com

ABSTRACT

Introduction: Pre-prandial exercise promotes greater mobilization of fat metabolism due to the increased release of catecholamines, cortisol, and glucagon. However, this response affects how the cardiovascular system responds to exercise. Objective: To evaluate the response of systolic, diastolic, and mean blood pressure, heart rate (HR) and rate-pressure product (RPP) to pre- and postprandial exercise. Methods: Ten physically active male subjects (25.50 ± 2.22 years) underwent two treadmill protocols (pre- and postprandial) performed for 36 minutes at 65% of VO_2max on different days. On both days, subjects attended the laboratory on a 10-hour fasting state. For the postprandial session, volunteers ingested a pre-exercise meal of 349.17 kcal containing 59.3 g of carbohydrates (76.73%), 9.97 g of protein (12.90%), and 8.01 g of lipids (10.37%). Blood pressure, HR and RPP were measured before and after exercise. The 2x2 factorial Anova with the multiple comparisons test of Bonferroni was applied to analyze cardiovascular variables in both moments (pre- vs. postprandial). The significance level was set at p<0.05. Results: Systolic (121.70 ± 7.80 vs. 139.78 ± 12.91 mmHg) and diastolic blood pressure (66.40 ± 9.81 vs. 80.22 ± 8.68 mmHg) increased significantly after exercise only in the postprandial session (p<0.05). HR increased significantly (p<0.05) after both protocols (64.20 ± 15.87 vs. 141.20 ± 10.33 bpm pre-prandial and 63.60 ± 8.82 vs. 139.20 ± 10.82 bpm postprandial). RPP had a similar result (8052.10 ± 1790.68 vs. 18382.60 ± 2341.66 mmHg.bpm in the pre-prandial session and 7772.60 ± 1413.76 vs. 19564.60 ± 3128.99 mmHg.bpm in the postprandial session). Conclusion: These data suggest that fasted exercise does not significantly alter the blood pressure. Furthermore, the meal provided before the postprandial exercise may promote a greater blood pressure responsiveness during exercise.

Keywords: exercise; fasting; postprandial period; blood pressure; heart rate.

RESUMO

Introdução: O exercício pré-prandial promove maior mobilização do metabolismo de gordura devido ao aumento da liberação de catecolaminas, cortisol e glucagon. Contudo, tal resposta afeta a forma como o sistema cardiovascular responde ao exercício. Objetivo: Avaliar a resposta da pressão sistólica, diastólica e média, a frequência cardíaca (FC) e o duplo produto (DP) ao exercício pré e pós-prandial. Métodos: Dez indivíduos ativos (25,50 ± 2,22 anos) foram submetidos a dois protocolos de exercício em esteira (pré e pós-prandial) realizados durante 36 minutos a 65% do $VO_2máx$ em dias diferentes. Em ambos os dias, os indivíduos compareceram ao laboratório em jejum de 10 horas. Para a sessão pós-prandial, os voluntários ingeriram uma refeição pré-exercício de 349,17 kcal, contendo 59,3 g de carboidratos (76,73%), 9,97 g de proteína (12,90%) e 8,01 g de lipídeos (10,37%). A pressão sanguínea, a FC e o DP foram medidos antes e depois do exercício. A Anova fatorial (2 X 2) com as comparações múltiplas de Bonferroni foi aplicada para análise das variáveis nos dois momentos (pré e pós-prandial). O nível de significância foi fixado em p < 0,05. Resultados: A pressão sanguínea sistólica (121,70 ± 7,80 vs. 139,78 ± 12,91 mmHg) e a diastólica (66,40 ± 9,81 vs. 80,22 ± 8,68 mmHg) aumentaram significantemente após o exercício somente na sessão pós-prandial (p < 0,05). A FC aumentou significantemente (p < 0,05) após ambos os protocolos (64,20 ± 15,87 vs. 141,20 ± 10,33 bpm pré-prandial e 63,60 ± 8,82 vs. 139,20 ± 10,82 bpm pós-prandial). O DP teve resultado semelhante (8.052,10 ± 1.790,68 vs. 18.382,60 ± 2.341,66 mmHg.bpm na sessão pré-prandial e 7.772,60 ± 1.413,76 vs. 19.564,60 ± 3.128,99 mmHg.bpm na sessão pós-prandial). Conclusão: Esses dados sugerem que o exercício em jejum não altera significantemente a pressão sanguínea. Além disso, a refeição fornecida antes do exercício pós-prandial pode promover maior responsividade da pressão sanguínea durante o exercício.

Descritores: *exercício; jejum; período pós-prandial; pressão sanguínea; frequência cardíaca.*

RESUMEN

Introducción: El ejercicio preprandial promueve una mayor movilización de metabolismo de la grasa debido al aumento de la liberación de catecolaminas, cortisol y glucagón. Sin embargo, tal respuesta afecta la forma en que el sistema cardiovascular responde al ejercicio. Objetivo: Evaluar la respuesta de la presión sistólica, diastólica y media, la frecuencia cardíaca (FC) y el doble-producto (DP) al ejercicio pre y postprandial. Métodos: Diez hombres activos

(25,50 ± 2,22 años), fueron sometidos a dos protocolos de ejercicio en cinta rodante (pre y postprandial) realizados durante 36 minutos a 65% del VO₂máx en días diferentes. En ambos días, los individuos asistieron al laboratorio después de un ayuno de 10 horas. Para la sesión postprandial, los voluntarios ingirieron una comida pre-ejercicio de 349,17 kcal, que contenía 59,3 g de hidratos de carbono (76,73%), 9,97 g de proteínas (12,90%) y 8,01 g de lípidos (10,37%). La presión sanguínea, la FC y el DP se midieron antes y después del ejercicio. Se aplicó el ANOVA factorial (2 x 2) con las comparaciones múltiples de Bonferroni para analizar las variables en los dos momentos (pre y postprandial). El nivel de significación se ha fijado en p < 0,05. Resultados: La presión sanguínea sistólica (121,70 ± 7,80 vs. 139,78 ± 12,91 mmHg) y la diastólica (66,40 ± 9,81 vs. 80,22 ± 8,68 mmHg) aumentaron significativamente después del ejercicio sólo en la sesión postprandial (p < 0,05). La FC aumentó significativamente (p < 0,05) después de ambos protocolos (64,20 ± 15,87 vs. 141,20 ± 10,33 lpm preprandial y 63,60 ± 8,82 vs. 139,20 ± 10,82 lpm postprandial). El DP tuvo un resultado similar (8.052,10 ± 1.790,68 vs. 18.382,60 ± 2.341,66 mmHg. lpm preprandial y 7.772,60 ± 1.413,76 vs. 19.564,60 ± 3.128.99 mmHg.lpm postprandial). Conclusión: Estos datos sugieren que el ejercicio en ayunas no altera significativamente la presión sanguínea. Además, la comida suministrada antes del ejercicio postprandial puede promover una mayor capacidad de respuesta de la presión sanguínea durante el ejercicio.

Descriptores: *ejercicio; ayuno; periodo posprandial; presión sanguínea; frecuencia cardíaca.*

INTRODUCTION

Aerobic exercise in the fasting state has been used as a strategy for body fat reduction due to increased mobilization of fat metabolism[1,2]. During physical activity in the fasting state, lipolysis is increased and promotes a subsequent increase in plasma concentrations of triglycerides and its subcomponents glycerol and free fatty acids[3,4]. This phenomenon occurs as a consequence of a marked catecholamine secretion and a greater sensitivity of the adipose tissue to these hormones generated by exercise[5]. In addition, exercise in a fasting state causes an increased release of glucagon and cortisol, favoring the use of fat as energy substrate once glycogenolysis becomes restricted by the depletion of muscle and liver glycogen[6-8]. In this sense, the mobilization of triglycerides reserves with decreased metabolism of carbohydrates aims to preserve the glucose concentration in the blood, in order to supply the central nervous system and red blood cells[9].

The variation of the endocrine response induced by food intake or by fasting seems to influence the response of the cardiovascular system to physical activity. For instance, regulation of blood pressure (BP) responds to plasma catecholamine[10]. Meanwhile, the variation in plasma concentration of norepinephrine necessary for maintaining normal levels of BP is facilitated by the ingestion of glucose and the subsequent release of insulin[11]. Thus, the reduction in the concentration of this neurotransmitter may limit the activation of β-adrenergic receptor and reduce cardiac contractility[12]. Accordingly, abnormally large falls in BP after a meal (postprandial hypotension) occurs due to the absence of elevated levels of catecholamine and may indicate a dysfunction of the sympathetic nervous system[13]. In addition, insulin response induced by glucose consumption can cause changes in the stimulation of the autonomic nervous system affecting the regulation of BP[14].

On the other hand, the increase of insulin secretion has a vasodilator effect, which is elicited by an increase of nitric oxide release by the vascular endothelial cells, causing a reduction in BP[15]. This increase in insulin secretion alters the action of the arterial baroreceptors, resulting in a reduction in baroreflex sensitivity. This phenomenon causes tachycardia in response to insulin[16]. However, BP appears to respond to other factors related to food intake, such as the volume ingested, and not only the glucose concentration. There is evidence in previous literature that a high volume may attenuate postprandial hypotension because of the increased gastric distension[17]. Yet, the magnitude of these changes is dependent on several factors such as age and gender[18].

Despite the diversity of studies evaluating the cardiovascular changes induced by fasting and by glucose intake, little evidence is available about the acute cardiovascular responses to exercise in fasting and after food intake. Therefore, the aim of this study was to analyze the acute response of BP, heart rate (HR) and rate-pressure product (RPP) of young men during exercise in fasting and postprandial conditions.

MATERIALS AND METHODS

Ten men aged between 20 and 30 years were invited to participate. All subjects provided written informed consent (consent form). They were not smokers or drinkers and had no musculoskeletal injuries. None of the participants had medical history of metabolic or cardiovascular pathologies and were not taking antihypertensive medications. All subjects were physically active with a minimum volume of 150 minutes per week of aerobic exercise for at least 12 months. This study was approved by the Research Ethics Committee of the University Center of Brasília, Brazil (protocol 858.452/2014).

Assessments were carried out on three different days with an interval of 72 hours. Subjects were asked to refrain from physical activity and ergogenic resources from 48 hours before the start of the study until the end of the tests. On the first day, participants underwent anthropometric measurements, assessment of maximal oxygen consumption and BP at rest. The level of physical activity was assessed adopting a questionnaire designed by the researchers. Body mass was measured to the nearest 50g on a digital scale (Filizola, São Paulo, Brazil). Height was measured to the nearest 0.1cm on a stadiometer (Sanny, American Medical of Brazil, São Paulo, Brazil). Body mass index (BMI) was calculated as weight divided by height squared. Body fat percentage was estimated by skinfold method (Cescorf Sporting Equipment, Porto Alegre, Brazil) adopting a 7-fold protocol[19].

Maximum oxygen uptake was estimated by a maximum incremental treadmill test (RUN 700, Techno Gym, Rio de Janeiro, Brazil) without inclination. The test consisted of an initial speed of 5km/h with increments of 1 km/h every minute. The test stopped when the subject reached volitional exhaustion or when maximum HR estimated by the formula 220-age was achieved. To estimate VO₂max, the ventilometer VO₂ Pro-Fitness (CEFISE Biotechnology Sports, Nova Odessa, Brazil) was used.

Experimental protocol

The volunteers performed two sessions of aerobic exercise (Fasting and Postprandial). Sessions were randomized and separated by 72 hours.

Both protocols occurred in the morning (7 am) and were preceded by a fasting period of 10 hours. In the fasted protocol, subjects remained at rest for 15 minutes prior to exercise. In the postprandial protocol, participants were provided with a 349.17 kcal meal consisting of yoghurt, whole wheat cookies, bananas, and a cereal bar, containing 59.3g of carbohydrate (76.73%), 9.97g of proteins (12.90%) and 8.01g of lipids (10.37%). After feeding, participants remained at rest for 15 minutes before the beginning of exercise.

Aerobic exercise was performed on a treadmill without inclination for 36 minutes at an intensity of 65% of the estimated VO_2max. The speed of the exercise was calculated according to the formula proposed by the American College of Sports Medicine[20]:

$$VO_2 = 3{,}5 + (0{,}2 * speed) + (0{,}9 * speed * \% \ grade)$$

In both situations, participants were submitted to two BP measurements with an automatic device (Microlife BP A100, Microlife Corporation, Widnau, Switzerland): after 15 minutes of rest and immediately after exercise. Heart rate was measured and recorded (FT1, Polar, Finland) on two occasions in both situations: after 15 minutes of rest and at the last minute of the exercise. The higher HR found in the last minute of the protocol was considered the post exercise HR. RPP was calculated as HR multiplied by systolic BP.

Statistical analyses

All analysis was performed using the Statistical Package for Social Sciences (IBM SPSS, IBM Corporation, Armonk, NY, USA, 21.0) for Mac OS X. Data normality distribution was verified using the Shapiro--Wilk test. Analysis of variance (repeated measures Factorial ANOVA) 2x2 (status x moment) was employed to evaluate systolic, diastolic and mean BP, as well as HR and RPP before and after exercise in both experimental conditions. Where significant differences and interaction between the effects were found, the Bonferroni multiple comparison test was applied. The effect size (ES) of the differences at pre-and post-moments was calculated according to the following equation[21].

$$Pre\text{-}Post \ ES = (Posttest \ mean - Pretest \ mean) / Pretest \ SD$$

Paired t-test was adopted to compare the percentage of maximum HR achieved at the end of testing in both conditions. The level of significance was set at p<0.05. Data are expressed as mean and standard deviation (SD).

RESULTS

Sample characteristics are shown in Table 1. All ten male subjects completed the experiment. None of them requested for interrupting the exercise in any of the two protocols. Running velocity in fasting and postprandial conditions was 8.54 ± 1.12 km/h. The percentage of maximum HR reached in the last minute of the two protocols showed no significant difference (72.59 ± 5.23% vs 71.62 ± 5.50%; p=0.593). In addition, analysis of variance showed that dependent variables (BP, HR and RPP) were not significantly different between fasting and postprandial conditions at pre-and post exercise moments.

Table 2 presents the outcomes of BP (systolic, diastolic and mean), HR and RPP before and after exercise in the fasting condition. HR and RPP were significantly higher after the exercise (p < 0.01), while BP did not change significantly (p > 0.05) (Table 2).

Results from the exercise in the postprandial condition are presented in Table 3. Similar to the fasting condition, HR and RPP increased significantly after the protocol (p < 0.01). However, contrary to the fasting condition, systolic, diastolic and mean BP presented a significant increase after the postprandial protocol (p ≤ 0.01) (Table 3).

In general, the present results show that ES values ranged from small (0.35-0.80) to large (> 1.5)[21]. Of note, higher ES values were observed for all variables in the postprandial condition (Tables 2 and 3).

Table 1. Sample characteristics.

Variable	Mean ± Standard deviation
Age (years)	25.5 ± 2.22
Weight (kg)	88.43 ± 10.71
Height (m)	1.80 ± 0.06
BMI (kg/m²)	27.11 ± 4.28
Body fat (%)	22.39 ± 8.26
Absolute VO_2max (L/min)	4.23 ± 0.59
Relative VO_2max (ml/kg/min)	48.75 ± 6.67
Maximal HR-estimated (bpm)	194.5 ± 2.22
Resting HR (bpm)	68.67 ± 10.95
Resting SBP (mmHg)	125.9 ± 10.57
Resting DBP (mmHg)	71.1 ± 8.62

BMI: Body mass index; VO_2max: Maximal oxygen consumption; HR: Heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure.

Table 2. Dependent variables in the fasting condition (Mean ± SD).

Variable	Pre	Post	p	ES
Systolic BP (mmHg)	126.3 ± 7.0	130.1 ± 12.2	0.93	0.54
Diastolic BP (mmHg)	71.6 ± 8.6	80.5 ± 7.0	0.20	1.03
Mean BP (mmHg)	89.8 ± 7.5	97.0 ± 7.8	0.25	0.96
HR (bpm)	64.2 ± 15.9	141.2 ± 10.3	0.0001	4.84
RPP (mmHg*bpm)	8052.1 ± 1790.7	18382.6 ± 2341.7	0.0001	5.77

BP: Blood pressure; HR: Heart rate; RPP: Rate pressure product; ES: Effect size.

Table 3. Dependent variables in the postprandial condition (Mean ± SD).

Variable	Pre	Post	p	ES
Systolic BP (mmHg)	121.7 ± 7.8	139.8 ± 12.9	0.01	2.32
Diastolic BP (mmHg)	66.4 ± 9.8	80.2 ± 8.7	0.003	1.41
Mean BP (mmHg)	84.8 ± 2.8	100.1 ± 2.2	0.001	5.46
HR (bpm)	63.6 ± 8.8	139.2 ± 10.8	0.0001	8.59
RPP (mmHg*bpm)	7772.6 ± 1413.8	19564.6 ± 3128.9	0.0001	8.34

BP: Blood pressure; HR: Heart rate; RPP: Rate pressure product; ES: Effect size.

DISCUSSION

This study investigated BP, HR and RPP responses to a 36 minutes' duration aerobic exercise at 65% of the VO_2max performed in the fasting state and after feeding. The results showed a significant increase in systolic, diastolic and mean BP only after the postprandial exercise condition, while HR and RPP increased significantly in both protocols. It is known that systolic BP increment during exercise is related to the maximization of the sympathetic activity in order to elevate HR and cardiac output and to promote a greater blood flow to the working muscles[22]. Furthermore, systolic BP augmentation induced by exercise is related to the endothelial vasodilator function[23].

Interestingly, the findings presented in this study appear to be the opposite of those reported in previous literature. Although exercise in the fasting state stimulate the secretion of catecholamines[5], the present report shows that there was an increase in systolic BP only in the postprandial protocol. In this sense, food intake seems to have influenced systolic BP prior to exercise. According to Vanis et al.[13], glucose intake promotes insulin secretion, resulting in a greater release of norepinephrine and epinephrine. When this phenomenon does not occur (e.g. because of a failure in the sympathetic nervous system), postprandial hypotension may happen[13]. Worthy of note, none of the subjects presented postprandial hypotension in the present investigation.

The non-occurrence of a significant rise in systolic BP after exercise in the fasting state would suggest a dysfunction in sympathetic activity of the participants with a consequent failure to catecholamine secretion[12]. Nevertheless, HR increased significantly in both situations. Moreover, participants of this study were physically active and used to the exercise intensity adopted in the experimental protocol. In this sense, cardiovascular alterations during exercise were not expected to be so high.

Although the results of this study do not indicate a significant difference between pretest measurements in both protocols, systolic BP at the postprandial session presented a lower resting value compared to the fasting session (121.70 ± 7.80 vs 126.30 ± 6.98mmHg; p>0.05). Therefore, the non-occurrence of a significant rise in systolic BP after exercise in the fasting state may be related to the higher resting values observed at this session.

On the other hand, glucose intake and the increase in plasma insulin concentration promote a higher activity of the sympathetic nervous system, stimulating alterations in cardiac autonomic function, HR variability and BP[14]. Thus, the significant increase in systolic BP induced by exercise in the postprandial condition can also be explained by the high glycemic load contained in the provided meal, since glucose intake induces a marked increase of insulin production and release[24]. Also, insulin has vasodilating properties related to the increase in nitric oxide secretion by vascular endothelial cells, resulting in a reduction of peripheral vascular resistance and BP. Hereupon, the behavior of BP in response to exercise in fasting and postprandial conditions relates to the variation in insulin concentration, caused by the ingestion or restriction of food. This behavior appears to depend on a balance between the vasodilator effect of insulin (e.g. associated with the release of nitric oxide) and the consequent reduction in peripheral vascular resistance, and its vasoconstrictor activity characterized by the stimulation of the sympathetic nervous system and the secretion of norepinephrine[15].

It is shown in the literature that diastolic BP response to exercise is mediated by the vasodilation capacity of peripheral vessels and the consequent reduction in peripheral vascular resistance[25]. Ordinarily, diastolic BP remain constant or show small increases during submaximal or maximal aerobic exercise[22,26]. In this study, diastolic BP increased significantly only after exercise in the postprandial condition. This result could be explained by a hyperactivity of the sympathetic nervous system and increased vascular response to adrenergic stimulation[25,27]. However, this pattern should be also noted after the exercise in the fasting state, since that condition promotes a greater secretion of catecholamines[5]. So, it is more likely that this diastolic BP result be related to the same reasons mentioned for systolic BP.

Both protocols promoted a substantial increase in HR. This increase occurs mainly due to the increasing blood flow and cardiac output requirement, to redirect the oxygenated blood to active muscles[22]. Even though glucose intake and increased insulin concentrations induce changes in HR response to exercise[14], the present investigation showed that postprandial exercise promotes a similar HR response compared to fasted exercise.

The RPP, also referred to as Double Product (systolic BP multiplied by HR), is a surrogate measure of myocardial oxygen demand and cardiac workload[28]. According to Nagpal et al.[29], RPP correlates well with myocardial oxygen consumption (MVO_2) in patients with ischemic heart disease. Thus, the assessment of RPP can potentially indicate a dysfunction of the autonomic nervous system (e.g. sympathetic stimulation)[30]. Despite the expected sympathetic activation difference between exercise in fasting and postprandial state, the present study shows that RPP increased significantly and similarly after both protocols. Once RPP is the product of systolic BP and HR, this result may be related to an increase in sympathetic stimulation to the heart combined to a decrease in sympathetic stimulation to the blood vessels[31].

The present investigation has some limitations. Firstly, the sample was small and consisted only of trained young men with no musculoskeletal, metabolic or cardiovascular diseases. Therefore, these results should not be extrapolated to women, patients suffering from diseases, obese or sedentary. Secondly, the exercise protocol adopted in this study consisted of 36 minutes of aerobic exercise on a treadmill at 65% of the VO_2max. So, these results should not be generalized to different intensities or exercise duration, since BP and HR respond differently to distinct efforts. Finally, plasma catecholamines were not quantified and could elucidate some of the issues raised in this report.

Nonetheless, to the best of our knowledge, this is the first study to compare cardiovascular responses to exercise in fasting and postprandial states in a sample of physically active men. Hence, the present report provides novel and important information on the topic. Future studies should be conducted trying to address the limitations of the present investigation, with special attention to plasma catecholamines concentration.

CONCLUSION

In summary, moderate aerobic exercise performed after the postprandial session promoted a significant increase in systolic, diastolic and mean BP. This result was not observed after the fasting protocol. Despite of the expected difference of the endocrine response between the two protocols, both conditions induced significant increases of HR and RPP. Based on this results, it may be suggested that the increased secretion of catecholamines in the fasted exercise is not enough to significantly alter BP. Moreover, it is possible to infer that the meal provided before the postprandial exercise caused a greater responsiveness of BP to exercise.

All authors declare no potential conflict of interest related to this article.

AUTHORS' CONTRIBUTIONS: Each author made significant individual contributions to this manuscript. MRM (0000-0003-0881-305X)* designed the study, supervised data collection and interpretation, wrote the draft, revised the manuscript. SNC (0000-0003-4936-1109)* performed data collection, statistical analysis and helped to wrote the manuscript. MTD (0000-0001-6245-3337)* helped to interpret data and to wrote the manuscript, revised the manuscript. RJO (0000-0002-1232-0488)* and RAED (0000-0002-2935-8642)* designed the study, supervised data collection, and revised the manuscript. FDL (0000-0001-5748-7540)* designed the study, performed data collection, statistical analysis and data interpretation, helped to wrote the manuscript. *ORCID (Open Researcher and Contributor ID).

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