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ASSESSMENT OF CHANGES IN OXIDATIVE STRESS AND ANTIOXIDANT STATUS WITH HYPERTENSION, SMOKING AND PAST HISTORY OF SCHISTOSOMIASIS

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Abstract:

Objective : Oxidative stress has been implicated in pathophysiological conditions that affect the cardiovascular system. To assess changes in oxidative stress with hypertension, smoking and past history of schistosomiasis in Saudi middle aged males who don't exercise regularly, using the levels of total antioxidant capacity, vitamin C, superoxide dismutase, total thiol, ceruloplasmin and uric acid, indices of antioxidant status, as a reflection of oxidative stress.

Subjects and Methods : The study population consisted of 80 Saudi middle aged male volunteers (mean age 47.8 ± 2.6 years) divided into eight non-overlapping categories, ten persons each, of either normotensive subjects, smokers and non-smokers, with or without past history of schistosomiasis or hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Results : A generalized reduction in the levels of superoxide dismutase and vitamin C was observed in association with both hypertension and cigarette smoking. In contrast, serum levels of uric acid and ceruloplasmin were elevated in association with both hypertension and cigarette smoking. Neither hypertension nor smoking had any significant effect, either independently or jointly, on total thiol levels. Notably, both hypertension and smoking inflicted, independently, significant reductions in serum total antioxidant capacity levels that were mostly noticeable among subjects with no other complications. Moreover, association of both smoking and hypertension boosted the effect that either one alone had on total antioxidant capacity and superoxide dismutase levels.

Conclusion : The current study observed an important overall reduction in the antioxidant mechanisms. Hypertension and cigarette smoking had the strongest direct associations with changes in indices of antioxidant status while past history of schistosomiasis had very little association. Whether the low activity of the antioxidant system is the cause or the consequence of the increased oxidative status needs further evaluation, but the fact that the low activity included several systems points to the reduction being more a consequence than a cause.

Key words : antioxidant, oxidative stress, hypertension, smoking, schistosomiasis, Saudi.

Determination of SOD and uric acid concentrations: SOD levels were measured in plasma samples (separated from venous blood collected into EDTA sample tubes and centrifuged at 2000g for 10 minutes at VQ by ELISA using monoclonal antibody as described previously [24]. Serum uric acid concentration was measured using a COBAS MIRA, spectrophotometric analyzer with reagent kit purchased from Roche Diagnostic Systems, Inc. (Branchburg, NJ).

Statistical analysis: All data analyses were performed by means of the Statistical Package for the Social Science (SPSS version 10.0). Results were expressed as a mean ± standard deviation (SD). Student's t test was used to compare molecule levels between patients and controls. Statistical significance was assumed at a p value <0.05.

Results:

In order to evaluate the oxidative stress associated with hypertension, cigarette smoking and past history of schistosomiasis, blood levels of total antioxidant capacity, vitamin C, superoxide dismutase, total thiol, ceruloplasmin and uric acid, indices of antioxidant status, were measured and shown in Tables 1-6, respectively.

Serum level of total antioxidant capacity (TAC) was significantly lower for hypertensive non-smoking subjects compared to the normotensive non-smoking ones in the absence of any other contributing factor ($p<0.05$; p^4 and p^5, Table 1). A past history of schistosomiasis for the hypertensive non-smoking subjects markedly enhanced the statistical significance of the reduction in TAC, compared to the normotensive nonsmoking subjects with no history of schistosomiasis, above that attributed to hypertension alone (p^8 versus p^4 or p^5, Table 1). Moreover, subjects with all three factors, hypertension, smoking and past history of schistosomiasis, had the highest reduction in TAC compared to subjects who didn't have any of these factors ($p<0.0001$; p^9, Table 1). In contrast, serum levels of TAC were not different statistically for all equivalent subject groups that differed only in either their past history of schistosomiasis or their smoking habits (N.S.; p^1, p^2 and p^3, Table 1). The only exception was for normotensive smoking subjects with no past history of schistosomiasis who showed a statistically significant reduction in TAC compared to their nonsmoking counterparts ($p<0.05$; p^2, Table 1). Moreover, hypertension alone had no significant effect on the level of TAC among smoking subject groups (N.S.; p^6 and p^7, Table 1). Notably, a past history of schistosomiasis for the normotensive non-smoking subjects neutralized the impact hypertension and smoking had on the level of TAC (N.S.; p^{10} versus p^9, Table 1).

A highly significant reduction in the level of plasma vitamin C was observed in all equivalent groups as a result of hypertension with the significance of the reduction being more pronounced among non-smoking ($p<0.0001$; p^4 and p^5, Table 2) versus smoking groups ($p<0.005$; p^6 an p^7, Table 2). A similar highly significant reduction in the level of plasma vitamin C was observed in all equivalent groups as a result of smoking with the significance of the reduction being more pronounced among normotensive ($p<0.0001$; p^2 and p^3, Table 2) versus hypertensive groups ($p<0.005$ and $p<0.001$; p^2 and p^3, respectively, Table 2). A minor, yet statistically significant, reduction in

vitamin C level as a result of past history of schistosmoiasis was observed among all subject groups except the non-smoking hypertensive ones (p^1, Table 2). Notably, association of any of the other two factors, or both, with hypertension didn't cause any further enhancement of the statistical significance of the reduction in vitamin C level above that already observed with hypertension alone (p^4, p^5 and p^8 - p^{10}, Table 2).

A significant reduction in plasma superoxide dismutase (SOD) levels was observed in all equivalent non-smoking groups as a result of hypertension ($p<0.005$; p^4 and p^5 , Table 3), yet, no statistically significance reduction was observed as a result of hypertension among the equivalent smoking groups (N.S.; p^6 and p^7 , Table 3). Likewise, a similarly significant reduction in SOD levels was observed in all equivalent normotensive groups as a result of smoking ($p<0.005$; p^2 and p^3, Table 3). In contrast, smoking only exerted a negative effect on SOD levels among hypertensive subjects who had a past history of schistosomiasis ($p<0.05$; p^3, Table 3). Moreover, plasma SOD levels were not different statistically as a result of past history of schistosomiasis for all equivalent subject groups except the smokincr hypertensive ones (p^1, Table 3). Notably, association of any of the other two factors with hypertension didn't cause any further enhancement of the statistical significance of the reduction in SOD level above that already observed with hypertension alone ($p < 0.005$; p^4, p^5, p^8 and p^{10}, Table 3), however, association of all three factors markedly enhanced the statistical significance of the reduction above that observed with hypertension alone ($p<0.0001$; p^9, Table 3).

The plasma levels of total thiol didn't show any statistically significant differences among all subject groups in the presence of any of the investigated factors alone (N.S.; p^1 - p^7, p^{10} and p^{11}, Table 4). However, only a combination of past history of schistosomiasis and hypertension caused a statistically significant reduction in total thiol levels, regardless of the smoking habits of the investigated subjects ($p<0.05$; p^8 and p^9, Table 4).

As for ceruloplasmin, a significant elevation in its serum levels was observed for all equivalent non-smoking groups as a result of hypertension ($p<0.005$ and $p<0.0001$; p^4 and p^5, respectively, Table 5), in contrast, no statistically significant elevation in ceruloplasmin levels was observed as a result of hypertension among the equivalent smoking groups (N.S.; p^6 and p^7, Table 5). Likewise, a similarly significant elevation in ceruloplasmin levels was observed in all equivalent normotensive groups as a result of smoking, even though the elevation was stunningly more significant in groups with past history of schistosomiasis ($p<0.0005$; p^3 versus $p<0.05$; p^2 , Table 5). In contrast, smoking didn't have any statistically significant effect on serum ceruloplasmin levels among hypertensive subject groups (N.S.; p^2 and p^3, Table 5). Moreover, serum ceruloplasmin levels were also significantly elevated as a result of past history of schistosomiasis for all equivalent subject groups except the non-smoking, normotensive ones (p^1, Table 5). Furthermore, association of past history of schistosomiasis with hypertension moderately enhanced the statistical significance of the elevation in serum ceruloplasmin above that observed with hypertension alone ($p<0.0001$; p^8 versus $p<0.005$; p^4 , Table 5), yet, association of all three factors didn't

have any further enhancement effect ($p<0.0001$; p^9, Table 5).

Finally, the pattern of fluctuation in the serum levels of uric acid was analogous to that of ceruloplasmin, where a significant elevation in the serum levels of uric acid was observed in all equivalent non-smoking groups as a result of hypertension ($p<0.0001$ and $p<0.005$; p^4 and p^5, respectively, Table 6), in contrast, no statistically significance elevation in uric acid was observed as a result of hypertension among the equivalent smoking groups (N.S.; p^6 and p^7, Table 6). Likewise, a similarly significant elevation in uric acid levels was observed in all equivalent normotensive groups as a result of smoking, even though the elevation was stunningly more significant in groups with no history of schistosomiasis ($p<0.0001$; p^2 versus $p<0.01$; p^3, Table 6). In contrast, smoking didn't have any statistically significant effect on serum uric acid levels among hypertensive subject groups (N.S.; p^2 and p^3, Table 6). In a marked contrast to ceruloplasmin, uric acid levels were not significantly elevated as a result of past history of schistosomiasis for all equivalent subject groups except the non-smoking normotensive ones (p^1, Table 6). Furthermore, association of a past history of schistosomiasis with hypertension or association of all three factors didn't have any further enhancement effect on the elevation in serum uric acid levels above that observed with hypertension alone ($p<0.0001$; p^4, p^8, and p^9, Table 6).

observed TAC might be due to exogenously provided antioxidants. Moreover, a strong correlation between changes in antioxidant capacity and serum uric acid during lifestyle intervention has been reported [57]. Furthermore, vitamin C was shown to affect the overall antioxidant status [58]. Therefore, the observed reduction in TAC would be the product of the difference between reductions and elevations in the levels of individual antioxidants.

Apart from a minor, yet statistically significant, reduction in the levels of vitamin C and elevation in the level of ceruloplasmin, past history of schistosomiasis alone didn't have any effect on the levels of TAC, SOD. uric acid or total thiol. However, association of past history of schistosomiasis with hypertension noticeably boosted the effect on the levels of TAC, total thiol and ceruloplasmin above that observed with hypertension alone. In contrast, this association didn't cause any further enhancement of the effect on vitamin C. SOD and uric acid levels above that already observed with hypertension alone. Moreover. association of all three factors past history of schistosomiasis smoking and hypertension, markedly enhanced the effect on TAC, above that attributed to hypertension alone or hypertension and past history of schistosomiasis.

In hypertension, the mechanisms responsible for the increase of ROS species, superoxide, hydrogen peroxide and hydroxyl radical, are still not well understood, even though an increase in the ROS production and/or a decrease in the disposal of antioxidant mechanisms have been proposed. There are 3 key enzymes which besides the proton leakage across the mitochondrial membrane, account for the majority of the ROS generation: NADPH oxidase, uncoupled eNOS, and xanthine oxidase [59]. The role of NADPH oxidase is an important generator of ROS [60] and the implication of eNOS during deficiency states of arginine and tethrabiopterin were largely recognized in hypertensive states. Furthermore, it was recently found that spontaneously hypertensive rats were characterized by an increased level of oxyradical production from xanthine oxidase activity [60].

Cigarette smoking is known to be a source of free radicals that lead to oxidative stress and antioxidant depletion [18]. Cigarette smoke extract increases superoxide by stimulation of NADPH, which, in turn, reduces NO bioactivity and results in endothelial dysfunction [61, 62]. Acrolein, an important constituent of cigarette smoke, mediates these effects and remains stable in blood along with other gas-phase oxidants in cigarette smoke and thus are capable of acting directly on the vascular endothelium [36]. The oxidative stress from smoking was shown to influence the cardiovascular system in 2 ways: by directly delivering free radicals to the vascular system and by consuming antioxidants that would normally be available to protect against endogenous free radicals resulting from the respiratory process. Although the mechanism(s) for the smoking-induced low plasma SOD levels is unknown, inhaled NO or superoxide produced by cigarette smoking [63] may decrease circulating SOD or, alternatively, other components of smoking may downregulate SOD production [30].

The role of oxidative stress in the pathogenicity of hypertension and/or cigarette smoking is still not well understood. However, as many of the cardiovascular risk factors, including hyperlipidemia, hypertension, diabetes and smoking, are associated

with overproduction of reactive oxygen species or increased oxidative stress, both of which reduce vascular nitric oxide bioavailability and promote cellular damage [64], hence, increased oxidative stress is considered to be a common pathogenic mechanism of the effect of risk factors on the endothelium [7, 64, 65].

In conclusion, the current study observed animportant overall reduction in the antioxidant mechanisms. Hypertension and cigarette smoking had the strongest direct associations with changes in indices of antioxidant status while past history of schistosomiasis had very little association. Even though the increment in ROS may upregulate the antioxidant enzymes under higher amounts of pure oxygen or related species, consumption by ROS can overcome the increased production, leading to the low activity observed. Whether the low activity of the antioxidant system is the cause or the consequence of the increased oxidative status needs further evaluation, but the fact that the low activity included several systems points to the reduction being, more a consequence than a cause.

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Collectively, these results demonstrate a generalized reduction in the levels of SOD and vitamin C in association with both hypertension and cigarette smoking. These results are consistent with prior reports demonstrating a reduction in the activity of SOD in hypertensive patients [27, 28] and cigarette smokers [29, 30]. Moreover, a reduction in the plasma levels of vitamin C was also previously reported for hypertensive patients [31-33] and cigarette smokers [34-36]. Another report [37] demonstrated a reduction in the extracellular SOD activities and serum vitamin C levels among, smokers.

This study also demonstrated a generalized elevation in the levels of uric acid and ceruloplasmin in association with both hypertension and cigarette smoking. Prior reports demonstrated elevations in the serum levels of uric acid in hypertensive patients [38-40] and cigarette smokers [41-43]. Moreover, uric acid is known to have proinflammatory effects on vascular smooth muscle cells that seem to be mediated by intracellular redox pathways [44] and recent observations in experimental hyperuricernia suggest that uric acid may in fact have a pathogenic role [45]. In addition, the increase in uric acid concentrations by smoking status was shown previously to be secondary to increased production through the xanthine oxidase pathway [43]. As for ceruloplasmin, elevations in its serum levels have been reported previously for cigarette smokers [46], in accordance with results from this study. In contrast, conflicting accounts of the association of serum ceruloplasmin with hypertension were reported, concluding either negative [47, 48] or positive association [49]. Metalloproteins, such as ceruloplasmin, are well known for their critical role in metal homeostasis and function as storage reservoirs and/or chaperones for essential trace metals. The antioxidant properties of these proteins have been attributed primarily to their binding of the redox active metals, thus minimizing their capacity to catalyze ROS production via the Fenton reaction. Evidence indicates that these proteins are induced during the acute-phase response [50, 51] and under oxidative stress [52, 53] which explains the observed elevation in its level in this study.

Antioxidant capacity is the number of moles of a given free radical scavenged by a test solution, independently of the capacity of any one antioxidant present in the mixture [26]. In the case of plasma, being a heterogeneous solution of diverse antioxidants, the antioxidant status is better reflected by antioxidant capacity that is a combination of all redox chain antioxidants, including several analytes such as thiol bearing proteins, and uric acid. Indeed, an increase of TAC indicates improved in vivo antioxidant status, or the result of the activation of an adaptation mechanism to oxidative stress [54]. Alternatively, a decrease of TAC indicates deprived in vivo antioxidant status. Indeed, it is well established that smoking habits reduce the TAC of human plasma, a reduction which is reversed after stopping smoking [26]. Moreover, hypertension was reported to cause a similar reduction in TAC levels [55]. These previous findings are in line with the results presented in this study demonstrating a reduction of TAC in both hypertensive patients and cigarette smokers. Moreover, a synergism between the action of hypertension and cigarette smoking on TAC levels was also observed. Taking, into account the normal concentrations of endogenous analytes such as uric acid, ascorbate, albumin, bilirubin and lipoproteins the study of Kampa et al., [56], concluded that about 85% of the TAC is due to endogenous analytes, and only 15% of the

Discussion:

The primary defense against oxidative stress in extracellular fluids results from a number of low molecular weight antioxidant molecules being either water- (ex. Vitamin C) or lipid-soluble (ex. Vitamin E). These antioxidants are either generated during normal metabolism (ex. uric acid, bilirubin, albumin, thiols) or introduced in the body by the consumption of dietary products rich in antioxidants (olive oil, fruits and vegetables, tea, wine, etc) [25]. The sum of endogenous plus exogenous (food-derived) antioxidants represents the total antioxidant capacity (TAC of extracellular fluids. Changes of these antioxidants reflect their consumption during acute oxidative stress states. Oxidative stress, or the imbalance between reactive oxygen species and total antioxidant capacity, plays a role in multiple disease processes. In order to evaluate the oxidative stress associated with hypertension, cigarette smoking and past history of schistosomiasis, blood levels of several indices of antioxidant status were measured. As cooperation between different antioxidant pathways provides greater protection against attack by reactive oxygen or nitrogen radicals, compared to any single compound, TAC may give more relevant biological information compared to that obtained by the measurement of individual biomarkers, as it considers the cumulative effect of all antioxidants present in plasma and body fluids [26]. For that reason, TAC was measured, as well as concentrations of the individual antioxidants, vitamin C, SOD, uric acid, ceruloplasmin and thiol groups.

Assessment of antioxidant activities in hypertensive subjects indicated a highly significant reduction in the plasma level of vitamin C in association with hypertension among all equivalent subject groups. Moreover, a significant reduction in the plasma levels of SOD, in association with hypertension, was also observed, even though it was merely among, the equivalent non-smoking subject groups. In contrast, the serum levels of ceruloplasmin and uric acid were significantly elevated in association with hypertension among the same groups. As for cigarette smoking, a highly significant reduction in the plasma level of vitamin C, analogous to that observed in association with hypertension, was observed in association with cigarette smoking among all equivalent subject groups. Moreover, a significant reduction in plasma SOD levels in association with smoking was also observed, though mostly among equivalent normotensive subject groups. In contrast, the serum levels of ceruloplasmin and uric acid were significantly elevated in association with cigarette smoking among equivalent normotensive subject groups. Neither hypertension nor smoking had any significant effect, either independently or jointly, on total thiol levels. Notably, both hypertension and smoking inflicted, independently, a minor, yet significant, reduction in serum TAC levels that was only noticeable among subjects with no other complications. Moreover, association of both smoking and hypertension boosted the effect that either one alone had on TAC and SOD levels. With the exception of vitamin C, the effect attributed to either hypertension or cigarette smoking was mostly evident in groups lacking the other one probably because the presence of either one of them causes a background effect that either diminishes or completely masks the effect of the other one.

Table 6 : Serum concentration of uric acid in norrnotensive and hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Assessed parameter	Normotensive				Hypertensive			
	Non-smokers		Smokers		Non-smokers		Smokers	
	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS
Uric acid (mg/dL)	4.01 ±0.7	4.81 ±0.81	5.64 ±0.90	6.01 ±1.10	5.84 ±0.90	6.12 ±1.10	5.84 ±1.04	6.43 ±1.12
p^1	< 0.05		N.S.		N.S.		N.S.	
p^2	< 0.0001				N.S.			
p^3	< 0.01				N.S.			
p^4	< 0.0001							
p^5	< 0.005							
p^6	N.S.							
p^7	N.S.							
p^8	< 0.0001							
p^9	< 0.0001							
p^{10}	< 0.05							
p^{11}	N.S.							

Data are given as mean ±SD (n = 10) and statistical significance was assumed at a P value <0.05. PHS, past history of schistosomiasis. N.S., non significant; p^1, comparison between -ve PHS and +ve PHS; p^2, comparison between -ve PHS non-smokers and smokers; P^3, comparison between +ve PHS non-smokers and smokers; p^4, comparison between -ve PHS non-smokers normotensive and -ve PHS non-smokers hypertensive; p^5, comparison between +ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^6, comparison between -ve PHS smokers normotensive and -ve PHS smokers hypertensive; p^7, comparison between +ve PHS smokers normotensive and +ve PHS smokers hypertensive; p^8, comparison between -ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^9, comparison between -ve PHS non-smokers normotensive and +ve PHS smokers hypertensive; p^{10}, comparison between +ve PHS non-smokers normotensive and -ve PHS smokers hypertensive; p^{11}, comparison between -ve PHS smokers normotensive and +ve PHS non-smokers hypertensive.

Table 5 : Serum concentration of ceruloplasmin in normotensive and hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Assessed parameter	Normotensive				Hypertensive			
	Non-smokers		Smokers		Non-smokers		Smokers	
	-ve PHS	+ve PHS	-ve PHS	+VC PHS	-ve PHS	+ve PHS	-ve PHS	+VC PHS
Ceruloplasmin (mg/dL)	16.91 ±4.1	18.19 ±4.8	24.18 ±5.2	29.17 ±6.5	24.35 ±6.3	32.61 ±7.4	28.68 ±7.1	34.7 ±7.9
p^1	N.S.		< 0.05		< 0.01		< 0.05	
p^2	< 0.05				N.S.			
p^3	< 0.0005				N.S.			
p^4	< 0.005							
p^5	< 0.0001							
p^6	N.S.							
p^7	N.S.							
p^8	< 0.0001							
p^9	< 0.0001							
p^{10}	< 0.001							
p^{11}	< 0.005							

Data are given as mean ±SD (n = 10) and statistical significance was assumed at a p value <0.05. PHS, past history of schistosomiasis. N.S., non significant; p^1, comparison between -ve PHS and +ve PHS; p^2, comparison between -ve PHS non-smokers and smokers; p^3, comparison between +ve PHS non-smokers and smokers; p^4, comparison between -ve PHS non-smokers normotensive and -ve PHS non-smokers hypertensive; p^5, comparison between +ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^6, comparison between -ve PHS smokers normotensive and -ve PHS smokers hypertensive; p^7, comparison between +ve PHS smokers normotensive and +ve PHS smokers hypertensive; p^8, comparison between -ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^9, ocmparison between -ve PHS non-smokers normotensive and +ve PHS smokers hypertensive; p^{10}, comparison between +ve PHS non-smokers normotensive and -ve PHS smokers hypertensive; p^{11}, comparison between -ve PHS smokers normotensive and +ve PHS non-smokers hypertensive.

Table 4 : Plasma concentration of total thiol in normotensive and hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Assessed parameter	Normotensive				Hypertensive			
	Non-smokers		Smokers		Non-smokers		Smokers	
	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS
Total Thiol (nmol/L)	1.82 ±0.61	1.69 ±0.58	1.53 ±0.54	1.48 ±0.52	1.52 ±0.54	1.39 ±0.49	1.42 ±0.51	1.31 ±0.46
P^1	N.S.		N.S.		N.S.		N.S.	
P^2	N.S.				N.S.			
P^3	N.S.				N.S.			
P^4	N.S.							
P^5	N.S.							
P^6	N.S.							
P^7	N.S.							
P^8	< 0.05							
P^9	< 0.05							
P^{10}	N.S.							
P^{11}	N.S.							

Data are given as mean ±SD (n = 10) and statistical significance was assumed at a p value <0.05. PHS, past history of schistosomiasis. N.S., non significant; p^1, comparison between -ve PHS and +ve PHS; p^2, comparison between -ve PHS non-smokers and smokers; p^3, comparison between +ve PHS non-smokers and smokers; p^4, comparison between -ve PHS non-smokers normotensive and -ve PHS non-smokers hypertensive; p^5, comparison between +ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^6, comparison between -ve PHS smokers normotensive and -ve PHS smokers hypertensive; p^7, comparison between +ve PHS smokers normotensive and +ve PHS smokers hypertensive; p^8, comparison between -ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^9, comparison between -ve PHS non-smokers normotensive and +ve PHS smokers hypertensive; p^{10}, comparison between +ve PHS non-smokers normotensive and -ve PHS smokers hypertensive; p^{11}, comparison between -ve PHS smokers normotensive and +ve PHS non-smokers hypertensive.

Table 3 : Plasma concentration of superoxide dismutase (SOD) in normotensive and hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Assessed parameter	Normotensive				Hypertensive			
	Non-smokers		Smokers		Non-smokers		Smokers	
	-ve PHS	+ve PHS	-ve PHS	+VC PHS	-ve PHS	+ve PHS	-ve PHS	+VC PHS
Superoxide Dismutase (ng/ml)	85.07 ±13.91	79.46 ±13.4	67.32 ±11.9	62.12 ±11.6	72.84 ±12.2	68.01 ±11.6	66.10 ±11.4	57.01 ±10.1
p^1	N.S.		N.S.		N.S.		< 0.05	
p^2	< 0.005				N.S.			
p^3	< 0.005				< 0.05			
p^4	< 0.005							
p^5	< 0.005							
p^6	N.S.							
p^7	N.S.							
p^8	< 0.005							
p^9	< 0.0001							
p^{10}	< 0.005							
p^{11}	N.S.							

Data are given as mean ±SD (n = 10) and statistical significance was assumed at a p value <0.05. PHS, past history of schistosomiasis. N.S., non significant; p^1, comparison between -ve PHS and +ve PHS; p^2, comparison between -ve PHS non-smokers and smokers; p^3, comparison between +ve PHS non-smokers and smokers; p^4, comparison between -ve PHS non-smokers normotensive and -ve PHS non-smokers hypertensive; p^5, comparison between +ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^6, comparison between -ve PHS smokers normotensive and -ve PHS smokers hypertensive; p^7, comparison between +ve PHS smokers normotensive and +ve PHS smokers hypertensive; p^8, comparison between -ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^9, comparison between -ve PHS non-smokers normotensive and +ve PHS smokers hypertensive; p^{10}, comparison between +ve PHS non-smokers normotensive and -ve PHS smokers hypertensive; p^{11}, comparison between -ve PHS smokers normotensive and +ve PHS non-smokers hypertensive.

Table 2 : Plasma concentration of vitamin C in normotensive and hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Assessed parameter	Normotensive				Hypertensive			
	Non-smokers		Smokers		Non-smokers		Smokers	
	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS
Vitamin C (µmol/L)	45.57 ±6.9	40.01 ±5.4	29.59 ±4.1	26.42 ±4.1	31.2 ±5.02	28.06 ±4.7	24.07 ±3.9	21.20 ±3.3
p^1	< 0.05		< 0.05		N.S.		< 0.05	
p^2	< 0.0001				< 0.005			
p^3	< 0.0001				< 0.001			
p^4	< 0.0001							
p^5	< 0.0001							
p^6	< 0.005							
p^7	< 0.005							
p^8	< 0.0001							
p^9	< 0.0001							
p^{10}	< 0.0001							
p^{11}	N.S.							

Data are given as mean ±SD (n = 10) and statistical significance was assumed at a p value <0.05. PHS, past history of schistosomiasis. N.S., non significant; p^1, comparison between -ve PHS and +ve PHS; p^2, comparison between -ve PHS non-smokers and smokers; p^3, comparison between +ve PHS non-smokers and smokers; p^4, comparison between -ve PHS non-smokers normotensive and -ve PHS non-smokers hypertensive; p^5, comparison between +ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^6, comparison between -ve PHS smokers normotensive and -ve PHS smokers hypertensive; p^7, comparison between +ve PHS smokers normotensive and +ve PHS smokers hypertensive; p^8, comparison between -ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^9, comparison between -ve PHS non-smokers normotensive and +ve PHS smokers hypertensive; p^{10}, comparison between +ve PHS non-smokers normotensive and -ve PHS smokers hypertensive; p^{11}, comparison between -ve PHS smokers normotensive and +ve PHS non-smokers hypertensive.

Table 1 : Serum concentration of total antioxidant capacity (TAC) in normotensive and hypertensive subjects, smokers and non-smokers, with or without past history of schistosomiasis.

Assessed parameter	Normotensive				Hypertensive			
	Non-smokers		Smokers		Non-smokers		Smokers	
	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS	-ve PHS	+ve PHS
Total antioxidant capacity(mmol/L)	1.203 ±0.201	1.076 ±0.213	1.017 ±0.187	0.935 ±0.184	1.010 ±0.180	0.924 ±0.173	0.943 ±0.177	0.831 ±0.170
p^1	N.S.		N.S.		N.S.		N.S.	
p^2	< 0.05				N.S.			
p^3	N.S.				N.S.			
p^4	< 0.05							
p^5	< 0.05							
p^6	N.S.							
p^7	N.S.							
p^8	< 0.005							
p^9	< 0.0001							
p^{10}	N.S.							
p^{11}	N.S.							

Data are given as mean ±SD (n = 10) and statistical significance was assumed at a p value <0.05. PHS, past history of schistosomiasis. N.S., non significant; p^1, comparison between -ve PHS and +ve PHS; p^2, comparison between -ve PHS non-smokers and smokers; p^3, comparison between +ve PHS non-smokers and smokers; p^4, comparison between -ve PHS non-smokers normotensive and -ve PHS non-smokers hypertensive; p^5, comparison between +ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^6, comparison between -ve PHS smokers normotensive and -ve PHS smokers hypertensive; p^7, comparison between +ve PHS smokers normotensive and +ve PHS smokers hypertensive; p^8, comparison between -ve PHS non-smokers normotensive and +ve PHS non-smokers hypertensive; p^9, comparison between -ve PHS non-smokers normotensive and +ve PHS smokers hypertensive; p^{10}, comparison between +ve PHS non-smokers normotensive and -ve PHS smokers hypertensive; p^{11}, comparison between -ve PHS smokers normotensive and +ve PHS non-smokers hypertensive.

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تقييم أثر ارتفاع ضغط الدم والتدخين والإصابة السابقة بالبلهارسيا على الجهد التأكسدى ومضادات الأكسدة

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يوضح البحث تأثير إختزال مضادات الأكسدة كنتيجة لإرتفاع ضغط الدم والتدخين مع خلفية الإصابة السابقة بالبلهارسيا فى ذكور متوسطى العمر من السعوديين .

تمت الدراسة على ٨٠ متطوع يتراوح أعمارهم حول ٤٧ عاماً قسموا إلي ٨ مجموعات من المدخنين وغير المدخنين ذو تاريخ سابق للإصابة بالبلهارسيا وضغط الدم المرتفع من عدمه .

تم قياس مستوى كل من سوبر كسيد ديسميوتيز وفيتامين ج وحمض البوليك والسيريلوبلازمين وكذلك الثيول لتقدير مدى تأثير مضادات الأكسدة مع ضغط الدم.

وأظهرت النتائج أن التدخين مع إرتفاع ضغط الدم وسلبيه الإصابة بالبلهارسيا لهم دوراً فى اختزال مضادات الأكسدة .