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Dietary total antioxidant capacity is inversely associated with cardiovascular events and cardiometabolic risk factors: A cross-sectional study

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ABSTRACT

Objectives: Dietary total antioxidant capacity (dTAC) has been introduced as a useful tool to quantify the antioxidant content of a diet. However, few studies have evaluated the association of dTAC with cardiovascular disease (CVD) occurrence and cardiometabolic risk factors in people with established CVD events. Thus, we aimed to investigate the presence of an association between dTAC values, cardiovascular events, and cardiometabolic risk factors in individuals with previous CVD in a Brazilian multicenter study.
Methods: This study has a cross-sectional design. We evaluated baseline data from the Brazilian Cardioprotective Nutritional Program Trial. Sociodemographic, anthropometric, clinical, and food-consumption data were collected in face-to-face interviews. We estimated dTAC from the mean of two 24-h dietary recalls by values of ferric-reducing antioxidant power.
Results: We evaluated 2346 participants, most of whom were men (58.4%), older adults (64.2%), and overweight (68.6%), and had coronary artery disease (92.4%). The mean dTAC was equal to 5.6 (interquartile range, 3.9-7.8) mmol/1000 kcal. Participants in the third dTAC tertile (9.2 mmol/1000 kcal) had a 22%, 59%, and 69% lower chance, respectively, of having hypertriglyceridemic waist phenotype, abdominal aortic aneurysm, and amputation due to arterial disease in comparison to the first tertile (3.4 mmol/1000 kcal).
Conclusions: The dTAC was inversely associated with hypertriglyceridemic waist phenotype, abdominal aortic aneurysm, and amputation due to arterial disease in individuals undergoing secondary care for CVD. Our results can guide strategies for the prevention of new CVD and its consequences.

Introduction

Cardiovascular diseases (CVDs) lead to around 31% of global deaths annually. They are known as one of the main causes of morbidity and increased health care expenditure [1]. Certain lifestyle factors, such as tobacco use, unhealthy diet, physical inactivity, and alcohol abuse, predispose individuals to overweight, obesity, and high blood pressure and glucose, factors that can trigger CVDs such as heart attacks and strokes [1,2].

Oxidative stress has been proposed as one of the causes and consequences of cardiovascular and other chronic non-communicable diseases (NCDs) [3,4]. Reactive oxygen species (ROS) are produced naturally in all organisms by cellular metabolism. In moderate amounts, ROS have an essential physiological function.

A.C.B.-F., C.R.T., A.M., and B.W. designed the study. A.S., A.P.S.C., S.L.P., H.H.M.H., A. M., A.C.B.-F., C.R.T., B.W., and J.B. performed the data generation and contributed to the statistical analysis, interpretation of the data, and drafting of the manuscript. The final version of the manuscript for submission was approved by all authors.

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They stimulate the activation of signaling pathways in response to changes in the intra- and extracellular environment. However, in high concentrations, ROS induce damage to biomolecules such as lipids, proteins, and DNA [5,6]. Redox imbalance mediated by ROS can lead to a chronic inflammatory process, associated with atherosclerosis, which can induce the onset of CVD, diabetes mellitus type I and II, cancer, neurologic and pulmonary diseases, Alzheimer's disease, and age-associated macular degeneration, among others [7–11].

Eating habits are among the modifiable NCD risk factors [12]. A systematic review by Afshin et al. reports that dietary improvements based on the regular consumption of whole grains, fruits, vegetables, nuts, and seeds could prevent one in five deaths worldwide [13]. In addition to being rich in dietary fiber, these foods contain antioxidants, which can scavenge oxygen free radicals from ROS and neutralize them [5]. Thus, the consumption of foods high in antioxidants is one of the strategies adopted to prevent and treat NCDs [14–16].

In this sense, dietary total antioxidant capacity (dTAC) has been introduced as a useful tool to quantify the antioxidant content of a diet [17]. Some studies have reported that a high dTAC is beneficial against cancers, death from all causes, CVD, some clinical risk factors, and severity of coronary stenosis [18–22]. Another study shows an inverse relation between dTAC and central adiposity and metabolic and oxidative stress markers in healthy young adults [23]. Similarly, a systematic review has revealed an inverse association between dTAC and the risk of chronic diseases [24]. On the other hand, a recent study found no association between dTAC as derived by ferric-reducing antioxidant power, and obesity and waist circumference [25].

Despite these studies using dTAC, few have evaluated its association with CVD occurrence and cardiometabolic risk factors in individuals with established CVD events. Thus, we aimed to investigate the presence of an association between dTAC values and cardiovascular events and cardiometabolic risk factors in people with previous CVD from a Brazilian multicenter study. Additionally, we evaluated the food groups that most contributed to dTAC values.

Methods

Study design

This study has a cross-sectional design. We evaluated baseline data from the Brazilian Cardioprotective Nutritional Program Trial (BALANCE Program Trial), which is a randomized, multicenter, national trial to assess the effects of a Brazilian cardioprotective diet on reducing cardiovascular events and risk factors in individuals with established CVD. At least 2468 participants 45 y or older who had experienced one or more cardiovascular events in the preceding 10 years were required to compose the study. This total number of participants took into account a type I error rate of 5%, a statistical power of 80%, a 20% incidence rate of the primary outcome in the control group, and a relative risk reduction of 30% in the intervention group. Details about the eligibility criteria are described in the study protocol [26]. The insertion of participants in the study was conducted from March 2013 to April 2015. The study was in accordance with the Helsinki Declaration principles [27] and was duly registered on ClinicalTrials.gov (NCT01620398). All participants provided written informed consent.

Data collection

CVD diagnosis

The presence of cardiovascular events was obtained by medical diagnostics, as previously described [26]. Eligibility consisted of the occurrence of one or more of the following cardiovascular events:

- asymptomatic coronary artery disease (CAD): stable or unstable angina (clinical diagnosis including diagnosis without complementary tests or history of positive stress test)
- symptomatic CAD: angiography or computed tomography coronary angiography, showing atherosclerotic stenosis $\geq$ 70% of the diameter of any coronary artery
- treated CAD: presence of angioplasty, stent, or revascularization
- asymptomatic peripheral artery disease (PAD): Ankle-to-arm ratio $<$ 0.9 to systolic blood pressure in any resting leg; angiographic or Doppler study showing $>$70% stenosis in a non-cardia artery
- symptomatic PAD: intermittent claudication
- treated PAD: vascular surgery for atherosclerotic disease
- heart attack: history of myocardial infarction or acute coronary syndrome; history of abnormality in the segmental movement of the cardiac wall on echocardiography or a fixed segmental defect on scintigraphy
- abdominal aortic aneurysm (AAA)
- amputation due to arterial disease
- stroke: presence of a clinical diagnosis of stroke or evidence of previous stroke from computed tomography or magnetic resonance imaging

Sociodemographic and anthropometric data and clinical measurements

Data on sociodemographic (age, sex, level of education, income), lifestyle (smoking, physical activity, food consumption), and clinical (use of medications, family history of diseases, documented presence of cardiovascular events confirmed by a doctor) characteristics were collected in face-to-face interviews. Additionally, anthropometry data (body weight, height, waist circumference) and blood pressure were measured.

Participants 60 y or older were classified as older adults. Furthermore, the participants were classified in the following categories: illiterate or literate; smoker + former smoker, or non-smoker; sedentary or active; family history of CAD or no history; use or do not use lipid-lowering medications, antihypertensives, anticoagulants, or hypoglycemic agents; and presence or absence of each cardiovascular event as already described. Participants who had at least completed elementary school were considered literate, and those who undertook mild, moderate, or intense physical activity were considered active.

Height was measured twice with wall-mounted stadiometers (precision, 0.5 cm) with the participants barefoot in a standing position. Body weight was collected twice with calibrated scales (precision, 100 g) with the participants barefoot and wearing light clothes. Waist circumference was measured at the midpoint between the lower border of the costal arch and the iliac crest at the midaxillary line, and was classified as high or not [28]. Body mass index was calculated using the formula weight (kg)/(height (m))^2. Participants were classified as overweight or not overweight. Adults with a body mass index $>$25.00 kg/m^2 and older adults with body mass index $>$27.00 kg/m^2 were considered overweight.

Blood samples were collected after a fasting period of 12 to 14 h. Traditional biochemical markers were analyzed with blood samples. Low-density lipoprotein cholesterol (LDL-c) was determined by the Friedewald formula, and triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-c), and glucose were measured by the enzymatic colorimetric method (Johnsons & Johnsons, Raritan, USA, VITROS 5600). Subjects were classified as having hipertriglyceridemia ($\geq$ 150 mg/dl) or not, high total cholesterol ($\geq$ 200 mg/dl) or not, high LDL-c ($\geq$ 130 mg/dl) or not, impaired fasting glucose ($\geq$ 100 mg/dl) or/and on hypoglycemic medication use or not, and low HDL-c (values $<$ 50 mg/dl for women and $<$ 40 mg/dl for men) or not.Subjects with high waist circumference ($\geq$ 94 cm for men and $\geq$ 80 cm for women) plus any two of the following four factors were classified as having metabolic syndrome: hipertriglyceridemia, hypertension, impaired fasting glucose, and low HDL-c [29,30]. In addition, the hypertriglyceridemic waist phenotype (HTGW phenotype) was classified by the simultaneous presence of high waist circumference and hypertriglyceridemia [31]. Blood pressure values were obtained following the recommendations of the American Heart Association. Subjects with high figures of systolic and diastolic blood pressure ($\geq$ 130 and 85 mmHg, respectively) or/and on antihypertensive medication use were classified as having hypertension [32].

Food intake and dTAC estimation

Food consumption was collected through two 24-h dietary recalls applied on non-consecutive, random days (holidays excluded). The multiple-pass method was used to standardize the collection of the food-consumption data and facilitate the maximum extraction of information about foods consumed [33]. Additionally, foods and preparations were grouped by similarity in chemical composition.

For dTAC estimation, we used a list of foods with TAC values proposed by Carlsen et al. coupled with supporting literature [17,34]. The list contains the TAC (mmol/100 g) of more than 3100 foods and preparations obtained according to ferric-reducing antioxidant potential. Thus, the dTAC is a sum of all TAC values from each food item consumed, in mmol/1000 kcal.

For foods whose TAC values were not available, data were used of foods that are botanically similar. Mixed preparations were broken down into ingredients, and the TAC of each preparation was calculated according to the quantity of each component of the recipe.

Statistical analysis

Data analysis was conducted with Stata software, version 13 (StataCorp LP, Coleege Station, Texas, USA). Data distribution was evaluated by the Kolmogorov–Smirnov test, graphical analysis, and asymmetry coefficients. The χ^2 test for

linear trends verified the relationship of the categorical variables across dTAC tertiles. Logistic regression was used to estimate the association between dTAC tertile (explanatory variable), HTGW phenotype, AAA, and amputation due to arterial disease. Regression analysis was performed only for outcomes with a statistically significant relationship with dTAC tertile observed in the χ^2 test of linear trends. Therefore, the results were presented as odds ratios and 95% confidence intervals. Variables used as an adjustment were selected based on the literature. Finally, to evaluate the groups that most contributed to dTAC, a quantile regression was performed and the pseudo r^2 values were extracted. For all analyses, the level of statistical significance used was 5%.

Results

A total of 2763 participants were assessed for eligibility, from which 2346 were included in the present study. The reasons for exclusions from the BALANCE Program Trial were not meeting eligibility criteria (n = 37), refusing to participate (n = 153), and other reasons (n = 39). An additional exclusion criterion for this study was lack of food-consumption data for dTAC calculation (n = 188).

Among the study participants, 1370 (58.4%) were men, with a median age of 63 y. Most participants had metabolic syndrome (73.3%), overweight (68.6%), were sedentary (66.1%), illiterate (64.1%), and smokers + former smokers (61.8%), along with having a family history of CAD (66.1%). Additionally, 39.3% presented the HTGW phenotype, 92.4% had CAD, 12% had stroke, and 11.3% had PAD. Among the PAD categories, 2.1% presented AAA and 1.6% were amputees due to arterial disease (Table 1). Furthermore, most participants were on medications such as lipid-lowering agents (85.7%), anticoagulants (90.2%), and antihypertensives (94.8%), and many were on hypoglycemic agents (41.3%).

In our sample, the median dTAC was 5.6 mmol/1000 kcal (interquartile range, 3.9–7.8), or 7.6 mmol/d. Regarding food consumption, the foods groups that contributed most to dTAC were coffee and infusions (16.0%), fruits (9.3%), beans and preparations (3.2%), and savory bread (1.8%) (Fig. 1). Other food groups contributed a total of 4.1% to dTAC, and consist of the sum of groups that contributed less than 1% each (oils and margarine, 0.76%; poultry and preparations, 0.74%; processed meats, 0.28%; sweet breads, 0.27%; oilseeds, 0.33%; rice and preparations, 0.18%; alcoholic beverages, 0.18%; fried foods, 0.18%; fish, 0.18%; sugar and sugar substitutes, 0.16%; corn and preparations, 0.14%; sauces and condiments, 0.07%; roots and tubers, 0.12%; vegetables, 0.5%; and seafoods, 0.05%).

From the dTAC tertiles, we oberved significant tendencies of low prevalence of women, adults, sedentary lifestyle, HTGW phenotype, AAA, and amputation due to arterial disease, while the prevalence of illiterate increased (Table 1). In brief, most of the participants with the HTGW phenotype, AAA, and amputation were older adults, smokers or former smokers, sedentary, overweight, hypertensive, presenting low values of high-density lipoprotein cholesterol, presenting CAD, and on several medications (Table 2).

Table 1
Characteristics of the participants, according to dietary total antioxidant capacity tertiles

Characteristic		dTAC tertile			
	Total (n = 2346)	T1 (lowest)	T2	T3 (highest)	P
dTAC, mmol/1000 kcal	5.6 (3.9–7.8)	3.4 (2.6–3.9)*	5.6 (5.1–6.2)†	9.2 (7.8–11.6)‡	<**0.001**
Adults	35.8	35.1	34.6	30.3	**0.035**
Older adults	64.2	32.4	32.6	35.0	
Men	58.4	29.7	33.6	36.6	<**0.001**
Women	41.6	38.4	32.9	28.7	
Illiterate	64.1	31.2	33.6	35.2	**0.011**
Smokers + former smokers	61.8	33.4	33.0	33.6	0.994
Sedentary	66.1	34.9	33.1	32.0	**0.007**
Overweight	68.6	34.2	33.6	32.2	0.072
High WC	82.4	34.0	33.2	32.8	0.078
HTGW phenotype	39.3	36.1	32.6	31.3	**0.016**
Family history of CAD	66.1	33.3	33.7	33.0	0.749
High LDL	15.3	34.2	33.0	32.7	0.687
High total cholesterol	22.6	35.9	33.5	30.6	0.079
Hypertriglyceridemia	44.6	35.2	33.1	31.7	0.051
Impaired fasting glucose	65.2	32.7	34.2	33.1	0.827
Low HDL	58.6	33.0	33.7	33.3	0.919
Hypertension	96.0	33.2	33.5	33.3	0.697
Metabolic syndrome	73.3	34.0	33.4	32.6	0.124
Stroke	12.0	36.5	29.8	33.7	0.534
CAD					
Asymptomatic	16.6	30.6	33.7	35.7	0.174
Symptomatic	36.3	32.7	34.3	33.0	0.916
Treated	69.0	32.3	34.4	33.3	0.382
Heart failure	50.0	33.7	34.1	32.2	0.363
PAD					
Asymptomatic	4.0	41.1	33.7	25.3	0.054
Symptomatic	6.5	35.5	32.2	32.2	0.608
Treated	4.1	41.7	27.1	31.2	0.202
AAA	2.1	49.0	30.6	20.4	**0.013**
Amputation due to arterial disease	1.6	55.3	28.9	15.8	**0.003**

AAA, abdominal aortic aneurysm; CAD, coronary artery disease; dTAC, dietary total antioxidant capacity; HDL, high-density lipoprotein; HTGW phenotype, hypertriglyceridemic waist phenotype; LDL, low-density lipoprotein; PAD, peripheric artery disease; WC, waist circumference
Data are presented as percentages except for dTAC values, which are presented as median (interquartile range)
P values were obtained through χ^2 test of the linear trend or Kruskal–Wallis test followed by Mann–Whitney U test
Values with different superscript symbols are statistically different from each other
Boldface indicates statistical significance

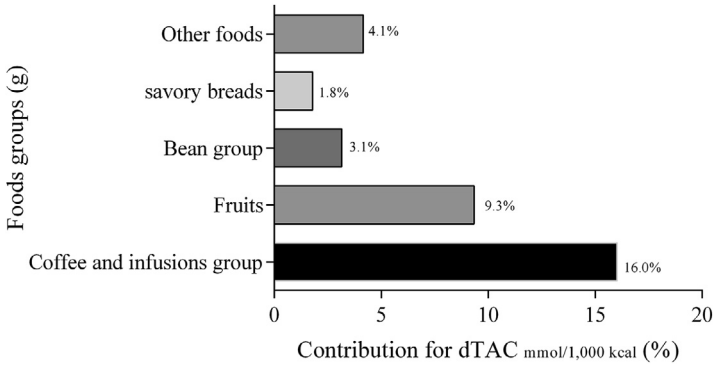

Fig. 1. Food groups that contribute most to dietary total antioxidant capacity. Percentages were obtained by quantile regression. Other foods are a sum of the food groups that contribute <1% to total antioxidant capacity, but with statistical significance.

Table 2
Characteristics of participants with hypertriglyceridemic waist phenotype, abdominal aortic aneurysm, and amputation due to arterial disease

Characteristic	HTGW phenotype (n = 8)	AAA (n = 49)	Amputation due to arterial disease (n = 38)
dTAC, mmol/1000 kcal	5.5 (3.8–7.6)	4.7 (3.5–6.9)	4.3 (2.6–6.1)
Older adults	61.2	65.3	57.9
Men	55.4	53.1	50.0
Illiterate	68.2	55.0	48.4
Smokers + former smokers	63.2	65.3	71.7
Sedentary	68.2	77.1	81.6
Overweight	83.2	57.1	57.9
Hypertension	96.7	98.0	94.7
Impaired fasting glucose	72.4	64.6	81.181
High LDL-c	18.8	22.9	18.9
High TC	34.2	33.3	27.0
Low HDL-c	72.8	58.3	56.8
CAD	93.1	85.7	86.8
Stroke	11.0	51.0	68.4
Lipid-lowering agents	86.1	71.4	71.1
Hypoglycemic agents	48.1	16.3	39.5
Antihypertensives	95.7	93.9	89.5
Anticoagulants	90.2	79.6	78.9

AAA, abdominal aortic aneurysm; CAD, coronary artery disease; dTAC, dietary total antioxidant capacity; HDL-c, high-density lipoprotein cholesterol; HTGW phenotype, hypertriglyceridemic waist phenotype; LDL-c, low-density lipoprotein cholesterol; TC, total cholesterol
Data are presented as percentages except for dTAC values, which are presented as median (interquartile range)

Moreover, the chance of having the HTGW phenotype, AAA, and amputation due to arterial disease was 22%, 59%, and 69% lower, respectively, among participants in the third dTAC tertile compared to those in the first tertile (Table 3), regardless of sex, age, use of any medications, presence of CAD, or stroke.

Table 3
Associations between dietary total antioxidant capacity tertile (explanatory variable) and cardiometabolic risk factors and cardiovascular events

Outcome	dTAC tertile (model 1)*		
	T1 (lowest)	T2	T3 (highest)
HTGW phenotype (n = 933)†	Reference	0.80 (0.65–0.99)	**0.78 (0.63–0.97)**
AAA (n = 49)‡		0.67 (0.34–1.32)	**0.41 (0.18–0.91)**
Amputation due to arterial disease (n = 38)‡		0.50 (0.22–1.12)	**0.31 (0.12–0.81)**

AAA, abdominal aortic aneurysm; dTAC, dietary total antioxidant capacity; HTGW phenotype, hypertriglyceridemic waist phenotype
Data are presented as odds ratio (95% confidence interval) according to multiple logistic regression
Boldface indicates statistical significance
*Model 1: Data were adjusted for sex, age, medication use (lipid-lowering agents, hypoglycemic agents, anticoagulants, and antihypertensives), presence of coronary artery disease, and stroke.
†Adjusted by model 1 + presence of any peripheral artery disease.
‡Adjusted by model 1 + presence of HTGW phenotype.

Discussion

In this article, we aimed to evaluate the association between dTAC and the occurrence of risk factors and cardiovascular events in individuals with previous CVD from a Brazilian multicenter study. We observed that the chance of having the HTGW phenotype, AAA, and amputation due to arterial disease was lower in participants in the third dTAC tertile compared to the first tertile (Fig. 2). In our sample, dTAC values were lower than other studies reporting dTAC levels associated with health benefits [35].

Dietary TAC is a relatively new tool proposed by Carlsen et al. to measure all or most antioxidants consumed in a diet based on the ferric-reducing antioxidant potential values of foods [17]. Antioxidants are molecules capable of neutralizing free radicals at the cellular level, aimed at homeostasis. Among antioxidants, those of non-enzymatic origin, such as vitamins, minerals, and bioactive compounds, exert a crucial role against oxidant agents and oxidative stress. Fruits, vegetables, oilseeds like the nuts group, berries, tea, coffee, and other foods, especially natural foods, are good sources of antioxidants [36–38]. Among the most studied antioxidants and those with scientific evidence-based health benefits in humans, we can cite vitamins C and E, selenium, and bioactive compounds like polyphenols (myricetin, quercetin, luteolin, kaempferol, and others) and phytosterols [39,40]. Studies have

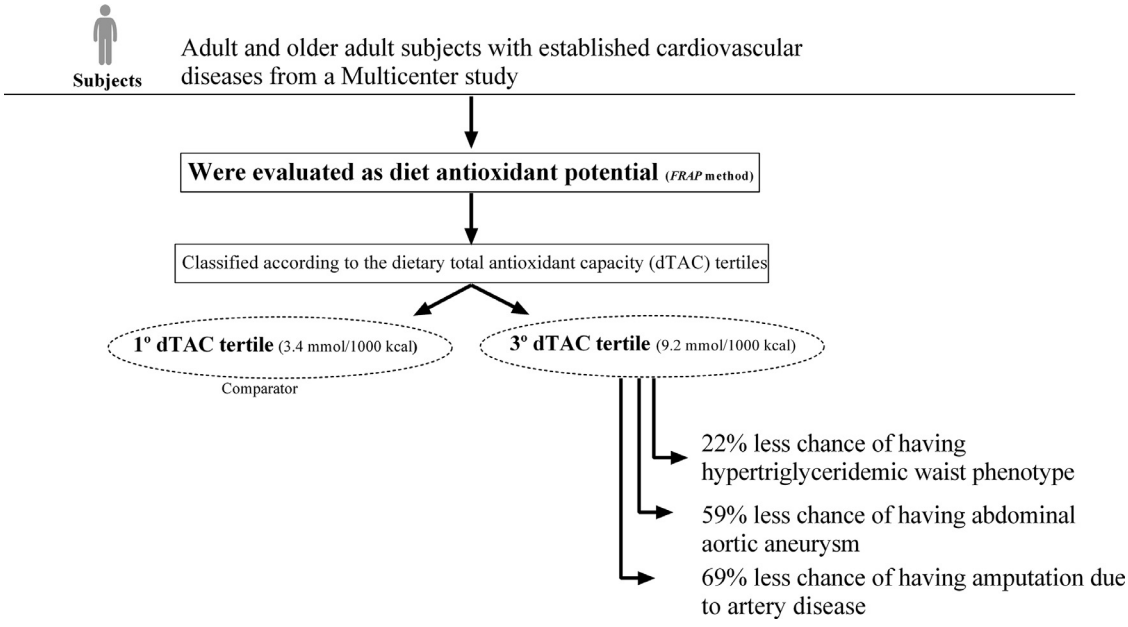

Fig. 2. Schematic representation of the main findings of the study.

shown that dietary antioxidants can inhibit LDL oxidation and prevent endothelial dysfunction, whereas oxidized LDL drives the inflammatory process and ROS production in the endothelium, contributing to the triggering of atherosclerosis.

Participants in our third dTAC tertile had a lower chance of having the HTGW phenotype, which has been closely associated with an increased risk of abnormal glucose metabolism [41] and has been proposed as a tool to measure cardiometabolic risk [31]. Adipose tissue expansion promotes ROS production due to hypoxia and tissue inflammation [42]. High ROS in the adipose tissue is associated with increased expression of NADPH oxidase and reduced expression of antioxidant enzymes, such as superoxide dismutase and catalase [43]. Some morbidities related to obesity, like insulin resistance, excessive free fatty acids in the blood, and dyslipidemias, are also events related to ROS production and contribute to the oxidative stress process. Once established, oxidative stress initiates a cycle characterized by intensive ROS production leading to the development of morbidities like NCDs, perpetuating ROS production.

Our study showed that participants with higher antioxidant intake have lower chances of presenting AAA and amputation due to arterial disease. This result suggests that high dTAC values could be protective against certain types of CVD, especially PAD. A close relationship between PAD and all CVDs with some form of oxidative stress has been documented [3], where this relation is built on the atherosclerosis process. After the internalization of oxidized LDL, macrophages release proinflammatory cytokines and ROS to sustain LDL oxidation, contributing to the progression of atherosclerosis and stimulation of endothelial molecule recruitment, which lead to apoptosis, necrosis, and thrombosis of the atherosclerotic plaque [3]. In PAD, a state of tissue hypoxia is present as result of atherosclerotic plaque formation, which blocks normal blood flux, and as a result, lower extremities of the arteries are more affected. All these processes contribute to ROS formation. Additionally, ROS is produced due to cycles of ischemia and reperfusion, causing increased oxidative stress [44].

A study has shown higher oxidant status and oxidative stress index in participants with AAA than those without [45]. Another study reports that vitamin C, β-carotene, ubiquinone, zinc, and selenium in blood are negatively and significantly correlated with AAA size in participants with AAA [46]. Antioxidant therapy has been an approach to treating and preventing PAD complications [44]. Despite this, to our knowledge this is the first study to show an inverse association between dTAC and AAA and amputation due to arterial disease. Previous studies have shown an association between low dTAC and risk of NCD in older adults [24]. In addition, a recent meta-analysis with prospective studies observes that a 5-mmol/d increment in dTAC (evaluated through ferric-reducing antioxidant power) reduces the risk of all-cause mortality, including CVD, by 7% [18]. Given these developments, it seems that dTAC is a suitable tool to measure antioxidants in the diet.

Interestingly, we found a lower prevalence of women, adults, and sedentary people according to the increase in dTAC tertiles, while the prevalence of illiterate patients increased. Contrary to our results, one study showed a higher prevalence of older adult women and of individuals with ≥4 years of an education level according to the increase in dTAC tertiles [48]. A lower prevalence of sedentary people in the higher dTAC tertiles may suggest that, despite being in secondary attention to CVD, the higher prevalence of non-sedentary people is associated with the ingestion of higher amounts of dietary antioxidants. The practice of physical activities, as well as the consumption of a healthy diet, rich in antioxidants, are recognized recommendations for maintaining a healthy lifestyle and preventing diseases [1]. Corroborating our results, one study showed that 37.5% of patients with nonalcoholic steatohepatitis with higher dTAC were sedentary, while 62.5% were active [50].

Regarding food consumption, the coffee and infusions group contributed most to dTAC values. Coffee is widely consumed in Brazil [47] and the population seems to resort to coffee, fruits, and beans according to our study in order to increase dietary antioxidants. Similarly, coffee was the main contributor of dTAC among older adult Brazilians studied by Nascimento-Souza et al. [48]. Several antioxidants are present in coffee and other infusions like tea. Among them, phenolic compounds like chlorogenic acid have beneficial effects on health [49]. Despite the high quantity of antioxidants in fruits and vegetables, coffee consumption was highlighted in our study, which confirms its consumption among Brazilians with previous CVD. Strategies to improve diet quality in terms of

antioxidants are necessary. The findings of this study may be of great interest to participants in secondary care, as well as a guide for professionals in the field.

The broad sample of participants already diagnosed with CVD can be considered a strength of our study. On the other hand, our study has a cross-sectional design that does not establish causal relationship. Furthermore, plasma antioxidant and oxidant markers were not evaluated, which impairs comprehension of the effect of a high-dTAC diet on the oxidative status of individuals in secondary CVD prevention.

Conclusion

In conclusion, dTAC was inversely associated with hypertriglyceridemic waist phenotype, AAA, and amputation due to arterial disease in people undergoing secondary care for CVD from a large Brazilian multicenter study.

These results suggest that a diet with a high antioxidant profile can help control the development and progression of cardiometabolic risk and CVD. We emphasize the need for more investigations, including long-term studies, to evaluate the relation between antioxidant intake and CVD progression and its morbidities.

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