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Dietary total antioxidant capacity is related to glucose tolerance in older people: The Hertfordshire Cohort Study

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Abstract

Background and Aims—Dietary antioxidants may play a protective role in the aetiology of type 2 diabetes. However, observational studies that examine the relationship between the antioxidant capacity of the diet and glucose metabolism are limited, particularly in older people. We aimed to examine the relationships between dietary total antioxidant capacity (TAC) and markers of glucose metabolism among 1441 men and 1253 women aged 59–73 years who participated in the Hertfordshire Cohort Study, UK.

Methods and Results—Diet was assessed by food frequency questionnaire. Dietary TAC was estimated using published databases of TAC measured by four different assays: oxygen radical absorbance capacity (ORAC), ferric-reducing ability of plasma (FRAP), total radical-trapping antioxidant parameter (TRAP) and trolox equivalent antioxidant capacity (TEAC). Fasting and 120-min plasma glucose and insulin concentrations were measured during a standard 75-g oral glucose tolerance test. In men, dietary TAC estimated by all four assays was inversely associated with fasting insulin concentration and homeostasis model assessment of insulin resistance (HOMA-IR); with the exception of ORAC, dietary TAC was also inversely related to 120-min glucose concentration. There were no associations with fasting glucose or 120-min insulin concentrations. In women, with the exception of the association between ORAC and 120-min insulin concentration, dietary TAC estimated by all assays showed consistent inverse associations with fasting and 120-min glucose and insulin concentrations and HOMA-IR. These associations were more marked among women with BMI ≥ 30 kg/m².

Conclusion—These findings suggest dietary TAC may have important protective effects on glucose tolerance, especially in older obese women.

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Authors' contribution: HO created the TAC database for estimation from the FFQ, conducted the statistical analysis, interpreted the data and wrote the manuscript. HES analyzed the data and assisted statistical analysis. DIWP was responsible for the glucose tolerance data. AA-S, ED and CC were responsible for the design of the Hertfordshire Cohort Study and data collection. SMR was responsible for the collection analysis of dietary data, and assisted in data interpretation and manuscript preparation. All the authors provided suggestions during the preparation of the manuscript and approved the final version submitted for publication.

Competing interests: Nothing to declare.

Keywords

Total antioxidant capacity; Diet; Glucose tolerance; Epidemiology

Introduction

Oxidative stress, defined as an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defence system, plays an important role in the pathogenesis of insulin resistance and type 2 diabetes mellitus (T2DM) [1]. Attention has therefore been focused on the potential protective role of dietary antioxidants in the aetiology of impaired glucose tolerance and T2DM [2,3]. A meta-analysis of observational data from cohort studies showed protective effects of greater intakes of dietary antioxidants on risk of T2DM [4]. However this is not consistent with findings of randomized controlled trials of antioxidant vitamin supplements, that failed to show benefits of increasing antioxidant intake [5,6]. The reasons for the difference in findings are not clear. One possibility is that high intakes of antioxidant nutrients act as markers for high intakes of other food constituents that also have antioxidant properties, such as polyphenols, that were not included in nutrient supplements used in the trials [5,6]. Additionally, previous observational and intervention studies have commonly focused on the effects of single or selected antioxidants [5,6], which may not necessarily reflect the combined actions all of antioxidants existing in the diet [7].

The recent proposed concept of total antioxidant capacity (TAC) of the diet may therefore provide important insights into the influence of dietary antioxidants on glucose metabolism. TAC aims to assess the free radical reducing capacity of all antioxidants in the diet and takes into account synergistic effects between them [8]. Using this approach, the ATTICA study recently showed that a high dietary TAC, estimated by three different assays, was inversely associated with fasting glucose, insulin and homeostasis model assessment of insulin resistance (HOMA-IR) in middle-aged healthy adults [9]. Protective effects of dietary TAC on fasting glucose and insulin concentrations have also been described in healthy young adults [10]. However, little is known about the effects of dietary TAC in older people, or whether TAC influences postprandial metabolism [9,10]. Given the increases in oxidative stress and reductions in antioxidant defence system with aging [11], additional studies are needed to assess the effects of dietary TAC on glucose metabolism among older people.

In the present study, we examined the association between dietary TAC and glucose tolerance, among 1441 men and 1253 women aged 59–73 years who were participants in the Hertfordshire Cohort Study [12].

Methods

The Hertfordshire Cohort Study (HCS)

Details of the HCS have been published elsewhere [12]. From 1911–1948, midwives recorded information on all infants born in the county of Hertfordshire, UK. In 1998, 3822 men and 3284 women (born 1931–1939) were traced. Permission to contact 3126 men

(82%) and 2973 women (91%) was obtained from general practitioners; of these men and women, 1684 men (54%) and 1541 women (52%) agreed to a home interview; subsequently 1579 men (94%) and 1418 women (92%) attended a clinic for further investigations. The study had ethical approval from the Bedfordshire and Hertfordshire local research ethics committee and the West Hertfordshire local research ethics committee. Written informed consent was obtained from all participants.

Dietary assessment

Diet over a 3-month period before the home interview was assessed using a 129-item food frequency questionnaire (FFQ) that was administered by a trained research nurse [13,14]. The FFQ was developed for the European Prospective Investigation of Cancer, and previously validated against 16-day weighed records [13]. Information on frequency of consumption and quantities consumed of different types of alcoholic beverages was obtained separately. Energy intakes from foods and alcoholic beverages were calculated by multiplying the frequency of consumption of a portion of each food item by its energy content, according to the UK food composition database or manufacturers' composition data [15].

Dietary TAC

Dietary TAC was estimated using published databases in which TAC of individual foods was measured by four different assays: oxygen radical absorbance capacity (ORAC), ferric-reducing ability of plasma (FRAP), total radical-trapping antioxidant parameter (TRAP) and trolox equivalent antioxidant capacity (TEAC). For each type of TAC measurement, data from a single database (foods analyzed in one laboratory) was used. ORAC data were obtained from a database provided by the USDA [16,17], FRAP from a database of foods analyzed in one Norwegian laboratory [18], TRAP and TEAC databases were based on Italian foods analyzed in one laboratory [19,20].

Each food included in the FFQ was matched to an equivalent food in each database. When more than one TAC value was available, an average value was calculated. If foods were not directly matched, values were imputed, applying the following procedure: 1) for dry foods, the TAC values of fresh foods were converted on the basis of their water content, 2) for dish-based foods, the TAC values of the main ingredients and sauce were combined on the basis of the proportions from recipes and/or commercial products [21], and 3) if no data were available, data for similar food items (e.g. same botanical group) were used. Of the total of 129 food groups and 5 alcoholic beverages on the FFQ, 60 items could be assigned ORAC values from the composition database (food coverage=44.8%); the equivalent numbers were 132 for FRAP (98.5%), 60 for TRAP (44.8%) and 63 for TEAC (47.0%). Total dietary TAC (units/day) for each type of TAC assay was calculated for every participant by multiplying the TAC value for a portion of each food/beverage by their reported frequency of consumption, and summing these values. Information on dietary supplements was not available in three databases; supplements were not included in the calculations of dietary TAC.

75-g oral glucose tolerance test (OGTT)

At clinic, a standard 75-g OGTT was performed after an overnight fast. Venous blood samples were taken for measurements of plasma glucose and insulin concentrations at 0, 30 and 120-min [22]. Plasma glucose was measured by an automated hexokinase method and plasma insulin was measured by two-site immunometric assay [22]. Insulin resistance was assessed by calculation of HOMA-IR according to the following formula: $\text{HOMA-IR} = [(\text{fasting plasma insulin (pmol/l)}/6.9) \times \text{fasting plasma glucose (mmol/l)}]/22.5$

Assessment of lifestyle and anthropometric variables

Information on lifestyle, and social and medical background was obtained at the home interview. Physical activity was assessed from responses to questions about the frequency and duration of gardening, housework, climbing stairs and carrying loads in a typical week [23]. A standardized activity score, ranging 0–100, was calculated, with higher scores indicating a greater level of activity. Smoking status was classified as never, ex-smoker or current smoker. Alcohol consumption was calculated from the reported consumption of alcoholic drinks and converted to units/week (1 unit equivalent to 284 ml beer, 125 ml wine, 50 ml fortified wine or 25 ml spirits).

At clinic, height was measured to the nearest 0.1 cm using a Harpenden pocket stadiometer (Chasmors, London UK) and weight to the nearest 0.1 kg on a Seca floor scale (Chasmors). Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2).

Statistical analysis

Of 2997 participants who attended clinic, 182 with previously diagnosed diabetes, and 44 with missing data on medical history of diabetes were excluded. A further 77 participants were excluded from the current analyses because of missing information on the variables used. The final sample consisted of 1441 men and 1253 women; 120-min insulin concentration were available for 980 men and 618 women. Plasma concentrations of glucose and insulin and HOMA-R were $\log_e$ transformed to normalise their distributions. Because older men and women may differ in associations between diet and function [24], their data were analysed separately. Descriptive data are presented, using means and standard deviation (SD), or frequency and percentage distribution. Mean and SD for $\log_e$ -transformed variables were back-transformed to geometric means and SD on the original scale of measurement.

Multiple linear regression analyses were performed to explore the associations of dietary TAC with glucose and insulin concentrations. These analyses were adjusted for potential confounders including age, BMI, smoking status, physical activity level, dietary supplement use and energy intake. In addition, effect modification by gender and these confounding factors was considered by adding interaction terms to the model. As shown previously [22,25], poor early growth is associated with impaired glucose metabolism in adulthood [26]. Interactions with birthweight and infant weight gain were therefore also considered. All statistical analyses were performed using Stata version 12 (Statacorp LP, College Station, TX, USA).

Results

Participant characteristics

In terms of lifestyle, men had slightly higher physical activity scores than women, but were more likely to be current or ex-smokers, fewer used dietary supplements and they had higher alcohol consumption (all $P < 0.001$, Supplemental Table 1). As expected, men had higher total energy intakes than women. Dietary TAC, estimated by FRAP and TEAC assays, was greater in men (both $P < 0.01$), but there were no differences between men and women in dietary TAC estimated by ORAC and TRAP assays. Men had higher fasting glucose concentrations and HOMA-IR than women, but lower 120-min glucose and insulin concentrations (all $P < 0.01$). There was no difference in fasting insulin concentration between men and women ($P = 0.07$).

Food and beverage contributors to dietary TAC

The relative contributions of different food groups to the four types of dietary TAC were very similar in men and women (Table 1). For dietary TAC estimated using the FRAP, TRAP and TEAC assays, the principal food/groups were coffee, tea, fruit and vegetables, contributing 75% to dietary TAC. In contrast, for dietary TAC estimated using the ORAC assay, the principal food/groups were fruit, vegetables, tea and potatoes. FRAP, TRAP and TEAC were strongly correlated with each other (range of Spearman correlation coefficients, $r = 0.90$ – 0.99), but were less highly correlated with ORAC (range $r = 0.09$ – 0.45).

Associations between dietary TAC and glucose tolerance

Table 2 shows the relationships of dietary TAC with fasting and 120-min glucose and insulin concentrations, before and after adjustment for potential confounding factors. In men, dietary TAC estimated by all assays was inversely related to fasting insulin concentration and HOMA-IR, but there was no association with fasting glucose concentration. These associations remained after adjustment for age, BMI and lifestyle variables. Dietary TAC estimated by FRAP, TRAP and TEAC was inversely related to 120-min glucose concentration, but comparable associations with 120-min insulin concentration were no longer evident after adjustment for confounding factors. In women, there was a clearer pattern of associations between dietary TAC and glucose and insulin concentrations. Dietary TAC estimated by all assays was inversely associated with fasting glucose, insulin concentrations and HOMA-IR. With the exception of dietary TAC estimated by ORAC, dietary TAC was also inversely associated with 120-min glucose and insulin concentrations. Adjustment for confounding variables made little difference to these associations. The effect sizes for the associations between dietary TAC and glucose and insulin concentrations were compared for men and women, but they did not differ (P for interaction all > 0.05).

Interactions between lifestyle factors and dietary TAC on glucose tolerance

Age, BMI and lifestyle factors (smoking status and physical activity level) were also considered as effect modifiers in the association between dietary TAC and glucose and insulin concentrations. An interactive effect was observed only for BMI among women, but not men, and not for the other factors examined (data not shown). Table 3 presents the

associations between dietary TAC and glucose and insulin concentrations in sub-groups of women, divided according to BMI ($<25.0 \text{ kg/m}^2$, $25.0\text{--}29.9 \text{ kg/m}^2$ or $\geq 30.0 \text{ kg/m}^2$). With the exception of TAC assessed by ORAC, dietary TAC differed in its associations with fasting and 120-min glucose and insulin concentrations according to BMI of the women studied. In each case more marked associations with dietary TAC were seen in women with BMI $\geq 30.0 \text{ kg/m}^2$ (P for interaction all <0.05). In contrast, interactive effects between dietary TAC estimated by ORAC and BMI were observed only in relation to 120-min glucose and insulin concentrations (P for interaction $=0.04$ and 0.008 , respectively).

Influence of early life on the associations between dietary TAC and glucose tolerance

Because impaired fetal and infant growth has previously been shown to be associated with poorer glucose tolerance in this cohort [22], our final analyses explored the influence of early growth on the associations between dietary TAC and glucose and insulin concentrations. Firstly we further adjusted the analyses for birth weight and infant growth; results were unaltered. Secondly we determined whether there was evidence of effect modification according to differences in early growth in the association between dietary TAC and glucose and insulin concentrations; no evidence for interaction was identified (data not shown).

Discussion

The principal finding of the present study was that higher dietary TAC was associated with better glucose tolerance in older men and women, as indicated by lower fasting and 120-min glucose and insulin concentrations. The second finding was that when we considered four different estimates of dietary TAC there were very consistent patterns of associations with measures of glucose tolerance for three assays (FRAP, TRAP, TEAC), but less consistent associations with ORAC.

There are few studies that have examined the relationship between dietary TAC and glucose and insulin concentrations and, to our knowledge, this has not been described in older adults before. Comparison with other studies is therefore limited to those of younger adults, who were studied in a fasting condition and in which data for men and women were combined [9,10]. The inverse relationships between dietary TAC and fasting glucose and insulin concentrations among the older men and women who participated in HCS are consistent with studies of younger (mean: 22 years) [10] and middle-aged (mean: 39 years) adults [9]. Additionally, our study extended these findings by examining glucose tolerance following a standard 75-g OGTT. Aging is directly associated with impaired antioxidant defences [11] which, together with oxidative stress accumulated over a lifetime, is thought to be a major risk factor for the development of T2DM accompanied by insulin resistance [27]. The beneficial effect of antioxidants from the diet might therefore be more important for older people whose antioxidant demands are high. Although our results require replication, the findings of the present study highlight the importance of dietary TAC and the potential for a beneficial role in glucose metabolism among older adults.

The inverse associations between dietary TAC and glucose and insulin concentrations in the present study differed according to BMI in women; more marked associations were found

among women who were obese. Although similar patterns of association were observed in men, tests for interaction were not statistically significant. One possible reason for different effects of dietary TAC on glucose metabolism according to obesity in women might be explained by greater oxidative stress in those who have a greater fat mass. Obesity is known to be a major contributor to oxidative stress, leading to insulin resistance, and which can be corrected by fat loss, exercise and dietary modification [28]. This raises the possibility that the protective effects of exogenous dietary TAC could be more important in obese individuals.

As a variety of assays have been developed to measure dietary TAC, it has been proposed that data from different assays should be used to obtain clear information about its role [8,29]. In the present study, we used four different estimates of dietary TAC (ORAC, FRAP, TRAP and TEAC). With the exception of ORAC, similar patterns of association between dietary TAC and glucose and insulin concentrations were observed. This is consistent with the high correlations seen between FRAP, TRAP and TEAC ($r=0.90-0.99$), and lower correlations with ORAC ($r=0.09-0.45$). The differing findings for ORAC may be explained by a more limited number of foods assigned values from the ORAC database. In particular, the lack of data for coffee in the ORAC database may be important [16,17], as it contributed more than 25% to estimates of dietary TAC using the three other assays. Protective effects of coffee consumption on T2DM have been described in meta-analyses [30], and the effect of dietary TAC estimated by ORAC in the present study may therefore be underestimated. It is notable that the three other estimates of TAC showed such consistency in their associations with glucose and insulin concentrations, despite being based on different assay processes.

There are several strengths to our study. These included studying a large well-characterised population of community-dwelling older men and women, our use of four different TAC assays, as well as our consideration of a range of confounding influences, including early growth measurements, on the relationship between dietary TAC and glucose tolerance. However the study did have some limitations. Firstly, dietary information was collected using an administered FFQ. There are concerns that participants can over-report intake in response to FFQs, although their ability to describe types of diets and patterns of food consumption is well-established [15]. Measurement error would be expected to attenuate associations and we do not think that mis-reporting on the FFQ could explain the associations we describe. Secondly, we used databases of TAC values developed in other countries as there is no TAC database for foods commonly consumed in UK, and matching of foods was not complete. It is possible that the dietary TAC values used in our analyses could underestimate the antioxidant capacity of the diet because of a lack of TAC values in the composition databases to assign to the foods included in the FFQ (coverage rate=44.8%–98.5%). However, despite these limitations, the associations between TAC and glucose and insulin concentrations in this study were consistent with observations in younger adults [9,10], adding weight to the suggestion that dietary TAC may also be of importance for older adults. Finally, it is possible that high intakes of dietary antioxidants are acting as markers, either of more health conscious lifestyles or of other important differences in diet, which could confound associations with glucose and insulin concentrations. While we

adjusted for a large number of potential factors, some of these (such as physical activity) may be underestimated, and the possibility of residual confounding cannot be ruled out.

In conclusion, higher dietary TAC is associated with better glucose tolerance, particularly among women with higher BMI. Oxidative damage is linked to a number of health outcomes in older age. Further studies of dietary TAC in relation to other outcomes are needed, together with a better understanding of the requirement for dietary antioxidants in older adults [7].

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1
Food and beverage contributors to dietary TAC according to the type of assay

ORAC	(%)	FRAP	(%)	TRAP	(%)	TEAC	(%)
Men (n = 1441)							
Fruit	31.9	Tea	26.3	Coffee	46.1	Coffee	37.5
Tea	16.3	Coffee	25.4	Tea	30.9	Tea	27.7
Potatoes	15.5	Fruit	12.0	Fruit	8.9	Fruit	11.3
Vegetables	14.6	Vegetables	11.6	Vegetables	5.7	Vegetables	7.5
Cereals and cereal products	7.0	Alcoholic beverages	6.7	Alcoholic beverages	2.6	Alcoholic beverages	6.0
Sweet, sugar and snack	5.1	Cereals and cereal products	4.2	Cereals and cereal products	1.8	Chocolate	3.3
Chocolate	3.6	Potatoes	3.5	Potatoes	1.5	Sweet, sugar and snack	1.9
Nuts	0.9	Sweet, sugar and snack	3.4	Chocolate	0.9	Potatoes	1.8
Non-alcoholic energy-containing beverage	0.5	Meat and meat products	1.1	Sweet, sugar and snack	0.7	Cereals and cereal products	1.5
Alcoholic beverages	0.5	Non-alcoholic energy-containing beverage	1.1	Non-alcoholic energy-containing beverage	0.6	Nuts	1.1
Women (n = 1253)							
Fruit	39.5	Coffee	26.5	Coffee	46.8	Coffee	39.1
Vegetables	15.4	Tea	25.0	Tea	28.0	Tea	26.2
Tea	14.4	Fruit	16.1	Fruit	11.7	Fruit	15.1
Potatoes	11.2	Vegetables	13.1	Vegetables	6.1	Vegetables	8.2
Cereals and cereal products	7.2	Cereals and cereal products	4.3	Alcoholic beverages	2.1	Chocolate	3.0
Sweet, sugar and snack	4.1	Potatoes	2.8	Cereals and cereal products	2.0	Alcoholic beverages	2.3
Chocolate	2.9	Sweet, sugar and snack	2.7	Potatoes	1.1	Cereals and cereal products	1.8
Nuts	0.7	Alcoholic beverages	2.7	Chocolate	0.8	Sweet, sugar and snack	1.6
Non-alcoholic energy-containing beverage	0.7	Non-alcoholic energy-containing beverage	1.4	Sweet, sugar and snack	0.6	Potatoes	1.4
Alcoholic beverages	0.4	Miscellaneous foods	1.1	Non-alcoholic energy-containing beverage	0.5	Nuts	0.9

Abbreviations: TAC, total antioxidant capacity; ORAC, oxygen radical absorbance capacity; FRAP, ferric-reducing ability of plasma; TRAP, total radical-trapping antioxidant parameter; TEAC, trolox equivalent antioxidant capacity.

Table 2
Associations between dietary TAC and glucose and insulin concentrations among 1441 men and 1253 women

	Men (n = 1441)				Women (n = 1253)			
	Crude		Adjusted		Crude		Adjusted	
	$\beta \pm SE$	<i>P</i> value	$\beta \pm SE^a$	<i>P</i> value	$\beta \pm SE$	<i>P</i> value	$\beta \pm SE^a$	<i>P</i> value
Glycemic status								
Fasting glucose (mmol/l) ^b								
ORAC	-0.000 ± 0.001	0.49	-0.000 ± 0.001	0.88	-0.001 ± 0.001	0.02	-0.001 ± 0.001	0.03
FRAP	-0.001 ± 0.001	0.40	-0.000 ± 0.001	0.80	-0.002 ± 0.001	0.005	-0.002 ± 0.001	0.002
TRAP	-0.000 ± 0.0004	0.43	-0.000 ± 0.0004	0.54	-0.001 ± 0.0004	0.01	-0.001 ± 0.0004	0.005
TEAC	-0.001 ± 0.001	0.43	-0.000 ± 0.001	0.63	-0.002 ± 0.001	0.006	-0.002 ± 0.001	0.002
120-min glucose (mmol/l) ^b								
ORAC	-0.003 ± 0.002	0.09	-0.001 ± 0.002	0.43	-0.005 ± 0.002	<0.001	-0.006 ± 0.002	<0.001
FRAP	-0.006 ± 0.002	0.001	-0.004 ± 0.002	0.02	-0.009 ± 0.002	<0.001	-0.010 ± 0.002	<0.001
TRAP	-0.003 ± 0.001	0.01	-0.002 ± 0.001	0.03	-0.005 ± 0.001	<0.001	-0.005 ± 0.001	<0.001
TEAC	-0.005 ± 0.002	0.006	-0.004 ± 0.002	0.03	-0.009 ± 0.002	<0.001	-0.010 ± 0.002	<0.001
Insulin sensitivity								
Fasting insulin (pmol/l) ^b								
ORAC	-0.010 ± 0.003	0.001	-0.007 ± 0.003	0.01	-0.008 ± 0.003	0.009	-0.009 ± 0.003	0.002
FRAP	-0.010 ± 0.003	0.002	-0.009 ± 0.003	0.003	-0.009 ± 0.003	0.01	-0.011 ± 0.003	<0.001
TRAP	-0.004 ± 0.002	0.08	-0.004 ± 0.002	0.04	-0.005 ± 0.002	0.03	-0.005 ± 0.002	0.005
TEAC	-0.008 ± 0.004	0.03	-0.008 ± 0.003	0.02	-0.008 ± 0.004	0.02	-0.010 ± 0.003	0.002
120-min insulin (pmol/l) ^{b,c}								
ORAC	-0.017 ± 0.006	0.004	-0.007 ± 0.006	0.21	-0.008 ± 0.005	0.10	-0.006 ± 0.005	0.23
FRAP	-0.021 ± 0.006	<0.001	-0.010 ± 0.006	0.07	-0.016 ± 0.006	0.004	-0.014 ± 0.006	0.02
TRAP	-0.006 ± 0.004	0.12	-0.003 ± 0.003	0.42	-0.009 ± 0.003	0.007	-0.008 ± 0.003	0.02
TEAC	-0.014 ± 0.006	0.03	-0.006 ± 0.006	0.30	-0.016 ± 0.006	0.009	-0.014 ± 0.006	0.02
HOMA-IR ^b								
ORAC	-0.010 ± 0.003	0.002	-0.008 ± 0.003	0.02	-0.009 ± 0.003	0.005	-0.011 ± 0.003	0.001
FRAP	-0.011 ± 0.004	0.002	-0.010 ± 0.003	0.004	-0.010 ± 0.004	0.004	-0.013 ± 0.003	<0.001
TRAP	-0.004 ± 0.002	0.07	-0.004 ± 0.002	0.04	-0.006 ± 0.002	0.02	-0.007 ± 0.002	0.002
TEAC	-0.009 ± 0.004	0.03	-0.008 ± 0.003	0.02	-0.010 ± 0.004	0.008	-0.012 ± 0.004	0.001

Abbreviations: TAC, total antioxidant capacity; SE, standard error; ORAC, oxygen radical absorbance capacity; FRAP, ferric-reducing ability of plasma; TRAP, total radical-trapping antioxidant parameter; TEAC, trolox equivalent antioxidant capacity; OGTT, oral glucose tolerance test; HOMA-IR, homeostatic model assessment of insulin resistance.

Values are regression coefficients indicating the change in glucose tolerance measurements per 1 unit increase in dietary TAC. An increment of 1 dietary TAC unit is equivalent to approximately 0.5-1 orange or 1-1.5 cups of tea.

^a Multiple linear regression model was adjusted for age (years, continuous), body mass index (kg/m², continuous), smoking status (never, ex or current), physical activity level (0-100, continuous), usage of dietary supplement (yes or no) and energy intake (kcal/d, continuous).

^b Glucose tolerance measurements were log_e transformed for analyses.

^c Because of data availability, 980 men and 618 women were included in the analyses for the association of dietary TAC with 120-min insulin concentration.

Table 3
Associations between dietary TAC and glucose and insulin concentrations among women, according to BMI

Women							
BMI: <25.0 kg/m ² (n = 416)			BMI: 25.0–29.9 kg/m ² (n = 500)		BMI: 30.0 kg/m ² (n = 337)		<i>P</i> _{interaction}
$\beta \pm \text{SE}^a$	<i>P</i> _{value}	$\beta \pm \text{SE}^a$	<i>P</i> _{value}	$\beta \pm \text{SE}^a$	<i>P</i> _{value}		
Glycemic status							
Fasting glucose (mmol/l) ^b							
ORAC	−0.001 ± 0.001	0.25	−0.001 ± 0.001	0.21	−0.002 ± 0.002	0.36	0.68
FRAP	0.000 ± 0.001	0.87	−0.001 ± 0.001	0.19	−0.005 ± 0.002	0.002	0.003
TRAP	0.000 ± 0.001	0.73	−0.001 ± 0.001	0.27	−0.003 ± 0.001	0.003	0.003
TEAC	0.000 ± 0.001	0.84	−0.001 ± 0.001	0.19	−0.005 ± 0.002	0.003	0.003
120-min glucose (mmol/l) ^b							
ORAC	−0.002 ± 0.003	0.51	−0.007 ± 0.003	0.009	−0.009 ± 0.003	0.01	0.04
FRAP	−0.005 ± 0.003	0.09	−0.010 ± 0.003	<0.001	−0.015 ± 0.003	<0.001	0.009
TRAP	−0.003 ± 0.002	0.10	−0.005 ± 0.002	0.004	−0.008 ± 0.002	<0.001	0.04
TEAC	−0.005 ± 0.003	0.08	−0.009 ± 0.003	0.002	−0.014 ± 0.003	<0.001	0.03
Insulin sensitivity							
Fasting insulin (pmol/l) ^b							
ORAC	−0.004 ± 0.005	0.46	−0.013 ± 0.005	0.007	−0.008 ± 0.006	0.22	0.53
FRAP	−0.003 ± 0.005	0.60	−0.007 ± 0.005	0.19	−0.025 ± 0.006	<0.001	0.05
TRAP	−0.001 ± 0.003	0.75	−0.002 ± 0.003	0.63	−0.015 ± 0.004	<0.001	0.02
TEAC	−0.002 ± 0.006	0.74	−0.005 ± 0.005	0.41	−0.026 ± 0.006	<0.001	0.02
120-min insulin (pmol/l) ^{b,c}							
ORAC	0.008 ± 0.009	0.36	−0.016 ± 0.009	0.07	−0.015 ± 0.011	0.19	0.008
FRAP	−0.005 ± 0.010	0.58	−0.002 ± 0.009	0.80	−0.033 ± 0.011	0.003	0.002
TRAP	−0.004 ± 0.006	0.48	0.001 ± 0.006	0.84	−0.021 ± 0.006	0.001	0.001
TEAC	−0.006 ± 0.010	0.52	0.000 ± 0.010	0.96	−0.036 ± 0.011	0.001	0.001
HOMA-IR ^b							
ORAC	−0.005 ± 0.006	0.37	−0.015 ± 0.005	0.006	−0.009 ± 0.007	0.19	0.62
FRAP	−0.003 ± 0.006	0.65	−0.008 ± 0.006	0.14	−0.031 ± 0.007	<0.001	0.02
TRAP	−0.001 ± 0.004	0.82	−0.002 ± 0.003	0.53	−0.018 ± 0.004	<0.001	0.005
TEAC	−0.002 ± 0.006	0.78	−0.006 ± 0.006	0.32	−0.032 ± 0.007	<0.001	0.006

Abbreviations: TAC, total antioxidant capacity; BMI, body mass index; SE, standard error; ORAC, oxygen radical absorbance capacity; FRAP, ferric-reducing ability of plasma; TRAP, total radical-trapping antioxidant parameter; TEAC, trolox equivalent antioxidant capacity; OGTT, oral glucose tolerance test; HOMA-IR, homeostatic model assessment of insulin resistance.

^aValues are regression coefficients indicating the change in glucose tolerance measurements per 1 unit increase in dietary TAC. An increment of 1 dietary TAC unit is equivalent to approximately 0.5–1 orange or 1–1.5 cups of tea. Multiple linear regression model was adjusted for age (years, continuous), smoking status (never, ex or current), physical activity level (0–100, continuous), usage of dietary supplement (yes or no) and energy intake (kcal/d, continuous).

^bGlucose tolerance measurements were log_e transformed for analyses.

^c Because of data availability, 618 women were included in the analyses for the association of dietary TAC with 120-min insulin concentration.