Supplementation with Quercetin Markedly Increases Plasma Quercetin Concentration without Effect on Selected Risk Factors for Heart Disease in Healthy Subjects1,2


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ABSTRACT The purpose of this double-blind study was to investigate the influence of adding a quercetin-containing supplement to the diet on plasma quercetin status, serum/platelet fatty acid levels and risk factors for heart disease. Healthy men and women with cholesterol levels of 4.0–7.2 mmol/L, consumed four capsules daily of either a quercetin-containing supplement (1.0 g quercetin/d) or rice flour placebo for 28 d. Quercetin intakes were ~50-fold greater than the dietary intakes associated with lower coronary heart disease mortality on the basis of epidemiologic studies. Subjects consuming quercetin-containing capsules had plasma quercetin concentrations ~23-fold higher than those of subjects consuming the control capsules. Quercetin supplementation did not modify serum total, LDL or HDL cholesterol or triglyceride levels. There were also no alterations of other cardiovascular disease or thrombogenic risk factors, including platelet aggregation, platelet thromboxane B2 production, blood pressure or resting heart rate. Furthermore, there was no effect on the levels of (n-6) or (n-3) polyunsaturated fatty acids in serum or platelet phospholipids. In conclusion, supplementation with quercetin-containing capsules markedly enhanced the plasma quercetin concentration but had no effect on other cardiovascular or thrombogenic risk factors. J. Nutr. 128: 593–597, 1998.

KEY WORDS: • humans • quercetin supplementation • cholesterol • thrombogenic factors • fatty acids

Quercetin is a member of a group of polyphenolic compounds known as flavonoids. Flavonoids, including quercetin, occur naturally in fruits, vegetables, nuts, seeds, flowers and bark. On the basis of recent studies, a normal diet contains 23–34 mg flavonoids/d on average (DeVries et al. 1997, Hertog et al. 1993b and 1995, Justesen et al. 1997); the majority of this is quercetin. Flavonoids have been reported to exhibit a wide range of biological effects including antibacterial, antiviral, anti-inflammatory, antiallergic and vasodilatory actions (reviewed by Cook and Samman 1996) as well as the inhibition of carcinogen-induced tumors in rats (Deschner et al. 1991) and colonic cell proliferation in vitro (Ranelletti et al. 1992). Recently, the Zutphen elderly study, the seven countries study and a Finnish study (Hertog et al. 1993a and 1995, Knekt et al. 1996) showed that there was a significant inverse association between dietary flavonoid intake and mortality from coronary heart disease (CHD)4 and a weaker inverse relation with incidence of myocardial infarction. Flavonol/flavone intake also was associated with a decreased risk of stroke in a Dutch cohort (Keli et al. 1996). It has been postulated that the antioxidant and free radical–scavenging properties of phenolic compounds, such as resveratrol and quercetin, present in red wine, may explain in part the low rate of CHD in the French population (reviewed by Cook and Samman 1996, Pace-Asciak et al. 1995).

Flavonoids, including quercetin (0.2 μmol/L–1 mmol/L), have been shown to scavenge superoxide anion, singlet oxygen and lipid peroxides (Husain et al. 1987, Robak and Gryglewski 1988, Takahama 1985) as well as to inhibit copper-catalyzed oxidation and cytotoxicity of LDL in vitro (Negrelewski 1988, Takahama 1985) as well as to inhibit copper-catalyzed oxidation and cytotoxicity of LDL in vitro (Negrelewski 1988, Takahama 1985). On the basis of recent studies, a normal diet contains 23–34 mg flavonoids/d on average (DeVries et al. 1997, Hertog et al. 1993b and 1995, Justesen et al. 1997); the majority of this is quercetin. Flavonoids have been reported to exhibit a wide range of biological effects including antibacterial, antiviral, anti-inflammatory, antiallergic and vasodilatory actions (reviewed by Cook and Samman 1996) as well as the inhibition of carcinogen-induced tumors in rats (Deschner et al. 1991) and colonic cell proliferation in vitro (Ranelletti et al. 1992).

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Other risk factors for heart disease have been inhibited by quercetin, including platelet aggregation in vitro (13–150 μmol/L quercetin; Chung et al. 1993, Xiao et al. 1995, Tseng et al. 1991), and, in laboratory animals (5–50 mg/kg; Xiao et al. 1993, Nuraliev and Avezof 1992), thromboxane formation and thromboxane receptor function (Tseng et al. 1991, Xiao et al. 1993). Dietary quercetin has also been shown to decrease serum total cholesterol in rats fed a cholesterol-enriched diet (Igarashi and Ohnuma 1995) or rats with alloxan diabetes (Nuraliev and Avezof 1992) and to increase high density...
lipoprotein (HDL) cholesterol in rats fed a high fat diet (Yugar et al. 1992). It is of further interest that vitamin E-deficient chicks fed quercetin had greater tissue essential fatty acid levels than controls (Jenkins and Atwal 1995). Other flavonoids, including morin, had similar effects.

To our knowledge, there have been no feeding trials published that evaluated the effects of quercetin supplementation on various risk factors for heart disease and thrombosis in humans. Previous reports of quercetin supplementation include estimated absorption (Gugler et al. 1975, Hollman et al. 1995 and 1997, Kühnau 1976, Nielsen et al. 1997, Noteborn et al. 1997); the most recent paper (Noteborn et al. 1997) suggests ~24 ± 9% absorption for quercetin aglycone.

The purpose of this study was to evaluate the potential for supplementary quercetin to increase concentrations of plasma quercetin and to influence selected cardiovascular and thrombotic risk factors.

### SUBJECTS AND METHODS

**Subjects and experimental design.** The subjects were 27 healthy men and women (cholesterol levels 4.0–7.2 mmol/L) selected from the Guelph community. Approval for this double-blind study was granted by the Human Ethics Committee of the University of Guelph and written informed consent was obtained from each subject. The subjects (aged 42.0 ± 2.6 y, mean ± SEM) were randomly assigned to the two supplementation groups, quercetin-supplemented (13 subjects) and control (14 subjects). The quercetin-containing and the placebo (rice flour) capsules were kindly provided by Roland Gahler of Gahler Enterprises, Burnaby, Canada. Quality control analysis showed that the quercetin capsules contained 250 mg purified quercetin anhydride, 250 mg mixed other bioflavonoids (composition unknown), 50 mg rutin, 50 mg bromelain and 4.2 mg magnesium stearate. The experimental group consumed four quercetin-containing capsules per day (total 1.6 g quercetin/d as well as 1.0 g bioflavonoids, 200 mg rutin and 200 mg bromelain), distributed throughout the day with their meals; the control group consumed four rice flour placebo capsules per day. Each group consumed the capsules for a period of 28 d beginning on d 0. After 28 d of capsule ingestion, both groups completed a washout period for 28 d during which there was no supplementation. Subjects were weighed on each visit (d 0, 28 and 56) and height was measured at entry; there were no significant differences between the groups at entry in these or other characteristics (Table 1). Three-day dietary records were obtained from each subject before supplementation. There were no differences between groups in daily energy intakes (~8 MJ/d) or intakes of saturated fat, cholesterol, monounsaturated fat or polyunsaturated fat (data not shown). The weight of the subjects was not affected throughout the supplementation period in either group. All dietary records were analyzed by the Can West Diet Analysis-Plus program which includes comparison with the Canadian Recommended Nutrient Intakes (West Publishing, St. Paul, MN). Compliance was monitored from a capsule count at the end of the study.

**Blood collection.** At d 0 (presupplementation), 28 (supplementation) and 56 (washout) blood was collected by antecubital venipuncture from fasting subjects into siliconized tubes containing no anticoagulant (for isolation), the anticoagulants ACD (25 g/L Na Citrate, 20 g/L dextrose, 14 g/L citric acid, all from Fisher Chemicals, Nepean, Canada) or Na Citrate (3.2%). Whole blood was treated as follows: 1) For tubes without anticoagulant, whole blood was centrifuged at 1250 × g for 15 min to obtain serum. Serum was used for measurement of total, HDL and low density lipoprotein (LDL) cholesterol levels, triglyceride levels, serum quercetin concentration and serum total phospholipid fatty acid level as described (Conquer and Holub 1996). Serum was stored at −70°C until all samples were collected and thawed just before analyses. 2) For tubes containing Na Citrate, whole blood was centrifuged at 200 × g for 17 min to obtain platelet-rich plasma (PRP), which was removed; the remaining blood was centrifuged at 1250 × g for 15 min to obtain platelet-poor plasma (PPP) (Conquer and Holub 1996). The PRP was used in aggregation studies as described. 3) For tubes containing ACD, washed platelet suspensions were prepared according to the method of Turini et al. (1994).

**Plaquelet aggregation.** Platelet aggregation was performed within 3 h of blood collection. Platelets were counted in PRP using a Coulter Counter, model ZM (Coulter Electronics, Burlington, Canada) and adjusted to a final concentration of 2.3–2.7 × 10^11 platelets/L by using autologous PPP. Aliquots (0.5 mL) of adjusted PRP were preincubated for 1 min in siliconized cuvettes with stirring at 900 rpm at 37°C in a dual-channel 800B aggregometer (Payton Instruments, Ion Trace, Scarborough, Canada) before addition of the aggregating agent, Collagen (Hormone-Chemie, Munchen, Germany) was added at various levels to determine the amount of collagen necessary to induce ~60% of maximal aggregation (at 10 mg/L). The platelets were allowed to aggregate for 2 min after the addition of agonist and then the level of aggregation was measured (Born 1962).

**Cholesterol and triglyceride measurement.** Total cholesterol was measured enzymatically with a diagnostic test (Sigma Diagnostics Procedure No. 352, St. Louis, MO). HDL cholesterol was isolated by using a dextran sulfate and magnesium ion solution to precipitate the very low density lipoprotein (VLDL) and LDL from the PPP sample. The HDL fraction was then assayed by an enzymatic assay (Sigma Diagnostics Procedure No. 352-3). Triglyceride was measured enzymatically with a diagnostic test (Sigma Diagnostics Procedure No. 339). LDL cholesterol was calculated by using the formula validated by Friedewald et al. (1972). Analyses of all samples for each volunteer were performed in a single assay.

**Total phospholipid and fatty acid analysis.** The fatty acid compositions of total phospholipid from serum and washed platelet suspensions from a random sample of subjects (6 from each group; before and after supplementation) were determined after lipid extraction, TLC, transmethylation of fatty acid residues and gas-liquid chromatography by using procedures similar to those previously described (Conquer and Holub 1996 and 1997).

**Thromboxane production.** Aliquots of PRP (0.5 mL) were stimulated with collagen (concentration of collagen for each subject depended on the level required to induce ~60% of maximal aggregation on d 0 and was kept constant on d 28 and 56) for 2 min; reactions were stopped by the addition of 250 μmol/L (final concentration) indomethacin (Sigma) and 4.5 g/L NaCl (final concentration) and samples were immediately chilled on ice (Conquer and Holub 1996). Samples were centrifuged at 2000 × g for 15 min at 0°C. The supernatant was removed and stored at −70°C until analysis using the thromboxane B2 (TXB2) Biotrack enzyme immunoassay system (Amsham Canada, Oakville, Canada).

**Quercetin analysis.** Plasma quercetin was analyzed as described (Maiani et al. 1997) with slight modifications. Samples were enzymatically hydrolyzed with β-glucuronidase (545 IU) and sulfatase (50 IU), and supernatants extracted with ethyl acetate. After hydrolysis with HCl and methanol, samples were extracted again with ethyl acetate.

### Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Quercetin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>41.5 ± 2.9</td>
<td>42.0 ± 2.7</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.7 ± 0.0</td>
<td>1.7 ± 0.0</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>74.3 ± 3.7</td>
<td>73.4 ± 3.1</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.0 ± 1.3</td>
<td>26.2 ± 1.1</td>
</tr>
<tr>
<td>Protein, % energy</td>
<td>15.4 ± 0.4</td>
<td>15.9 ± 0.5</td>
</tr>
<tr>
<td>Fat, % energy</td>
<td>34.9 ± 2.2</td>
<td>30.5 ± 1.9</td>
</tr>
<tr>
<td>Carbohydrate, % energy</td>
<td>48.5 ± 2.6</td>
<td>51.2 ± 2.0</td>
</tr>
<tr>
<td>Alcohol, % energy</td>
<td>1.2 ± 0.4</td>
<td>2.4 ± 1.2</td>
</tr>
<tr>
<td>Energy, kJ/d</td>
<td>8460 ± 749</td>
<td>7422 ± 368</td>
</tr>
</tbody>
</table>

1 Values are reported as means ± SEM, n = 13 (quercetin group) or n = 14 (control) for age, height, weight and BMI, or n = 11 for protein, fat, carbohydrate, alcohol and energy intakes. No significant differences between the groups were found for the above variables.

2 BMI, body mass index.
TABLE 2

Plasma quercetin concentrations in fasting subjects after placebo or quercetin supplementation for 28 d

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Quercetin</th>
</tr>
</thead>
<tbody>
<tr>
<td>ng/mL</td>
<td>μmol/L</td>
<td>ng/mL</td>
</tr>
<tr>
<td>Day 0</td>
<td>ND</td>
<td>27.7 ± 25.7</td>
</tr>
<tr>
<td>Day 28</td>
<td>18.5 ± 16.4</td>
<td>0.07 ± 0.06</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM, n = 8 (control) or n = 7 (quercetin). *Significantly different than d 0, P < 0.05; ND, nondetectable.

RESULTS

Plasma quercetin did not differ between the groups at d 0. A dramatic increase was observed in the experimental group (change was seen in the control group. Selected cardiovascular risk factors were not significantly altered by the 28 d of quercetin or placebo supplementation (Table 3). This included no effects on the total cholesterol:HDL cholesterol or the LDL cholesterol:HDL cholesterol ratios, two very important risk factors for heart disease (Kinosian et al. 1994 and 1995). Triacylglyceride levels also did not change throughout the supplementation period in either group (Table 3). Platelet aggregation, and platelet release of thromboxane A2 (TxB2) (d 28), based on measurements of TxB2 postsupplementation (d 28) were not significantly different than d 0 for either treatment group and were not significantly different between the groups at any time point. Systolic and diastolic blood pressures and resting heart rates did not differ between groups at any time nor did they change due to quercetin or placebo supplementation (Table 3).

Treatment groups had similar serum and platelet total phospholipid fatty acid compositions (based on a randomly chosen subset of subjects) at entry (data not shown). No significant differences were observed with quercetin supplementation for any of the fatty acids examined nor was there any effect of quercetin supplementation on the desaturation indices of certain pathways [22:5(n-3) + 22:6(n-3)/20:5(n-3) ratio] and [20:3(n-6) + 20:4(n-6)/18:2(n-6) ratio] (data not shown).

DISCUSSION

This investigation examined the effects of a quercetin-rich supplement (containing 250 mg quercetin, 50 mg rutin and 250 mg other bioflavonoids) on certain cardiovascular risk factors. To our knowledge, this is the first report of plasma quercetin concentrations in a controlled intervention trial in which quercetin-containing capsules were fed over an extended interval. Supplementation with 1 g quercetin/d increased plasma quercetin concentrations in fasting subjects 14-fold (from 0.10 to 1.5 ± 0.3 μmol/L; or from 27.7 to 427.1 ± 89.2 ng/mL). Our results agree with related studies that suggest quercetin absorption after oral consumption of quercetin aglycone (Hollman et al. 1995 and 1997, Nielsen et al. 1997, Noteborn et al. 1997). In 1995, Hollman et al. suggested that ~25% of quercetin is absorbed after con-

TABLE 3

Effect of supplementation with quercetin or placebo for 28 d on selected thrombogenic and other cardiovascular risk factors in healthy subjects

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>Quercetin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L serum</td>
<td>5.10 ± 0.25</td>
<td>5.14 ± 0.28</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L serum</td>
<td>1.57 ± 0.12</td>
<td>1.54 ± 0.11</td>
</tr>
<tr>
<td>Total cholesterol:HDL cholesterol, mol/mol</td>
<td>3.2 ± 0.2</td>
<td>3.3 ± 0.2</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L serum</td>
<td>2.88 ± 0.17</td>
<td>2.94 ± 0.25</td>
</tr>
<tr>
<td>LDL cholesterol:HDL cholesterol, mol/mol</td>
<td>2.0 ± 0.2</td>
<td>1.9 ± 0.2</td>
</tr>
<tr>
<td>Triglyceride, mmol/L serum</td>
<td>1.41 ± 0.23</td>
<td>1.40 ± 0.21</td>
</tr>
<tr>
<td>Platelet aggregation, % of maximum aggregation with 10 mg/L collagen</td>
<td>64.9 ± 3.7</td>
<td>58.9 ± 8.1</td>
</tr>
<tr>
<td>TxB2 production, ng/2.5 × 10^8 platelets³</td>
<td>49.8 ± 16.7</td>
<td>105.5 ± 40.8</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>120.4 ± 3.5</td>
<td>118.0 ± 4.0</td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>75.5 ± 2.4</td>
<td>72.5 ± 2.8</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>72.8 ± 3.3</td>
<td>70.4 ± 3.4</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM, n = 13 (quercetin) or 14 (control). No significant differences due to supplementation were found.

2 Values for platelet aggregation are for the concentration of collagen (0.5–5 mg/L) necessary to induce ~60% of maximum aggregation (10 mg/L collagen) on d 0.

3 TxB2, thromboxane B2; bpm, beats per minute.
sumption of quercetin aglycone in ileostomy patients. Recently, this was confirmed in subjects with an intact colon (Hollman et al. 1997, Nielsen et al. 1997, Noteborn et al. 1997). The mean serum quercetin concentration observed in our study after supplementation (427.1 ng/mL; 1.5 μmol/L) is much lower than that observed in rat studies in which rats fed diets containing 0.125% quercetin (equivalent to ~36.6 g quercetin/d in humans on a weight for weight basis) had plasma quercetin metabolites at levels of ~45 μmol/L, but was higher than that seen in plasma of subjects given a one-time dose of quercetin (225 ng/mL; 0.8 μmol/L) in various forms (Hollman et al. 1997). The elimination half-life of quercetin has been suggested to be ~25 h, implying that repeated dietary intake would lead to an accumulation in plasma (Hollman et al. 1997). Furthermore, peak levels of plasma quercetin occur from 0.7 to 7 h after ingestion. The samples in our study were taken from subjects who had fasted for at least 10 h, suggesting that the levels of quercetin as measured herein do not reflect immediate prior consumption but do reflect the minimum levels of quercetin expected in subjects with extended and consistent daily intakes.

Our levels of plasma quercetin obtained in vivo (1.5 μmol/L) were higher than those required to inhibit superoxide generation (Robak et al. 1988) and 50% of LDL oxidation in vitro studies (IC50) (Vinson et al. 1995a). As little as 0.25 μmol/L of quercetin significantly prolonged the lag phase, and 1 μmol/L of quercetin drastically affected the duration of the lag phase; the conjugated diene appearance was also abolished (Manach et al. 1995). The IC50 for inhibition of in vitro oxidation of lipoproteins (0.224 μmol/L) can be compared with the IC50 for β-carotene (4.30 μmol/L), vitamin E (2.40 μmol/L), vitamin C (1.45 μmol/L), butylated hydroxyaniline (BHA) (0.181 μmol/L) and BHT (0.270 μmol/L) (Vinson et al. 1995a). However, because quercetin is strongly bound to albumin in blood, it is not known whether any in vivo effects would be similar to those seen in vitro (Manach et al. 1995). Although in vivo evidence is lacking for antioxidant activity of quercetin, total reactive antioxidant potential assays, which provide a quantitative measure of the global antioxidant power of plasma, before and after ingestion of red wine and tea, suggest that flavonoids do have in vivo antioxidant activity (Maxwell et al. 1994, Serafini et al. 1994).

Although we observed elevated concentrations of quercetin in the plasma of quercetin-supplemented individuals, we did not observe modifications of various risk factors for cardiovascular disease (CVD). Despite the studies that have shown that quercetin could inhibit platelet aggregation in vitro (Chung et al. 1993, Tseng et al. 1991, Xiao et al. 1995) and in vivo in laboratory animals (Nuraliev and Avezoff 1992, Xiao et al. 1993), we did not observe any inhibition of either platelet aggregation or platelet release of TXA2. This may be due to the fact that the concentration of quercetin in the plasma of our supplemented subjects (1.5 μmol/L) did not reach high enough levels to exert an inhibitory effect on platelet activity, or quercetin in plasma is not in a form able to exert antioxidant/CVD risk factor–modifying activities. Alternatively, the presence of other bioflavonoids or the high levels of quercetin (1 g) used in this study may have had an inhibitory effect on modification of CVD risk factors. It is also possible that despite quercetin’s antioxidant effects (Vinson et al. 1995a) and its ability to inhibit lipoxygenase activity (Showell et al. 1981), it may have had no effect on cyclooxygenase activity or platelet aggregation. The first suggestion is the most likely on the basis of other studies (reviewed by Middleton and Kandaswami 1992, Pace-Asciak et al. 1995) that have shown flavonoid inhibition of platelet aggregation, adhesion and secretion as well as cyclooxygenase activities. In vitro studies (Chung et al. 1993, Tseng et al. 1991, Xiao et al. 1995) on quercetin-inhibited platelet aggregation (by agonists such as arachidonic acid, ADP, thrombin and collagen) have used levels of 20–500 μmol/L quercetin, 13–333 times the value for plasma quercetin (1.5 μmol/L) that we observed in our quercetin-supplemented individuals. On the basis of these studies, inhibition of platelet aggregation appears to require a minimum of ~10 μmol/L quercetin. Interestingly, when quercetin and rutin were injected into an extracorporeal stream of blood, calculated plasma concentrations of 0.03 μmol/L were enough to cause inhibition of the deposition of platelet thrombi on a blood-superfused collagen strip (Grzylewski et al. 1987).

Administration of 10 and 50 mg quercetin/kg rat weight (rats with aloxan diabetes) was sufficient to reduce the levels of total cholesterol and LDL cholesterol (Nuraliev and Avezoff 1992). However, in nondiabetic rats fed a cholesterol-free diet, quercetin supplementation did not lower cholesterol levels and an extremely high intake (0.2% of diet) was required to lower cholesterol levels in rats fed a high cholesterol diet (Igarashi and Ohnuma 1995). Oral daily supplementation of an equivalent level of quercetin (13.6 mg/kg based on an average of 73 kg) in humans (in our study, 1 g daily quercetin supplementation for 28 d), did not lower either total or LDL cholesterol or triglyceride or increase HDL cholesterol. It is possible that certain rodent models may be much more sensitive to quercetin-mediated blood lipid alterations than we have found in human subjects.

In summary, we have shown that prolonged ingestion of 1 g quercetin/d (28 d) raises plasma quercetin levels at least 14-fold, to mean levels of 1.5 μmol/L. Although this level has been shown in vitro to inhibit superoxide generation (Robak et al. 1988) and 50% of LDL oxidation in other studies (Vinson et al. 1995a), there appeared to be insufficient quercetin in serum to cause a significant reduction in platelet aggregation or TXA2 production or to modify other risk factors (including lipid/lipoprotein levels and platelet reactivity) for CVD. Epidemiologic data (Hertog et al. 1993b and 1995, Knekt et al. 1996) have indicated a protective effect against CVD of foods providing up to 16–24 mg quercetin and 23–34 mg total flavonoids daily (compared with 1000 mg quercetin and 2200 mg total bioflavonoid in our supplementary trial). Our results suggest that any protective effect of foods containing quercetin may be mediated via effects on risk factors other than those we have measured (inhibition of LDL oxidation or reduction in free radical production). Alternatively, the protective effect of quercetin-containing foods may be due to factors other than quercetin in those foods.

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