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Curcumin effects on blood lipid profile in a 6-month human study

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Abstract

Studies in animals and a short-term human study have suggested that curcumin, a polyphenolic compound concentrated in the curry spice turmeric, decreases serum cholesterol concentration. However, no controlled human trials have examined the effect of curcumin on cholesterol. This study investigated the effects of consuming curcumin on the serum lipid profile in men and women. Elderly subjects ($n = 36$) consumed 4 g/d curcumin, 1 g/d curcumin, or placebo in a 6-month, randomized, double-blind trial. Plasma curcumin and its metabolites were measured at 1 month, and the serum lipid profile was measured at baseline, 1 month, and 6 months. The plasma curcumin concentration reached a mean of 490 nmol/L. The curcumin concentration was greater after capsule than powder administration. Consumption of either dose of curcumin did not significantly affect triacylglycerols, or total, LDL, and HDL cholesterol over 1 month or 6 months. However, the concentrations of plasma curcumin and serum cholesterol were positively and significantly correlated. Curcumin consumption does not appear to have a significant effect on the serum lipid profile, unless the absorbed concentration of curcumin is considered, in which case curcumin may modestly increase cholesterol.

Keywords: Cholesterol; Curcumin; High-density lipoprotein; Low-density lipoprotein; Triglycerides

1. Introduction

Curcumin is a polyphenolic molecule extracted from turmeric, the spice consisting of the powdered root of the *Curcuma longa* plant [1,2]. Turmeric contains about 5% curcumin, which gives the spice its yellow color but none of its flavor and thus is used as a yellow food coloring [1]. Because of its anti-inflammatory, anti-oxidant, and other potentially protective properties [1,2], curcumin has been tested in animal models of Alzheimer's disease (AD), with favorable results [3,4]. Curcumin decreased total plasma cholesterol in studies of mice, rats, and rabbits [5–7]. In human subjects, oral administration of 0.5 g/day of curcumin for 1 week decreased total serum cholesterol 12% and increased HDL cholesterol 29% [8]. Therefore, curcumin might serve as a cholesterol-lowering drug. As part of a double-blind, placebo-controlled, randomized trial of curcumin for possible treatment of Alzheimer's disease, we examined the effect of 1 g/day or 4 g/day oral curcumin on serum lipids and lipoproteins over periods of 1 month or 6 months.

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2. Subjects and methods

2.1. Subjects

Subjects were eligible for the study if they were aged 50 years or older, were ethnic Chinese living in Hong Kong, had progressive decline in memory and cognitive function for at least 6 months, had NINCDS-ADRDA diagnosis of probable or possible AD [9], and gave informed consent. For subjects unable to understand the study and their role in it, consent was obtained from the caregivers. Exclusion criteria were anticoagulant or antiplatelet treatment or bleeding risk factors, current smoking, or severe illness making completion of the study unlikely. The study was approved by the Clinical Research Ethics Committees for the regions including the participating sites (the New Territories East and Kowloon West regions of Hong Kong).

Thirty-four subjects and their caregivers were recruited. Two additional subjects were treated for only 1 month since the trial period was nearing conclusion. We excluded from analysis five subjects who did not take the assigned study drug for at least 1 day during the first month and who also did not take the assigned study drug for at least 30 days during the 6-month study, and who thus were unsuitable for analysis of results at either 1 month or 6 months. Table 1 describes the remaining 31 subjects.

2.2. Study design

The study was a double-blind, placebo-controlled, randomized, 6-month trial. Patients were randomized to the three oral, daily, curcumin doses: placebo, 1 g, or 4 g. Since the volume of drug was rather large and inconvenient to consume, patients were allowed to choose whether they preferred to take the study drug as either 10 capsules to be swallowed after a meal or as a packet of 4 g powder to be mixed with food and eaten. Yellow starch was used as placebo in order to match the color of curcumin. Patients continued to receive any drugs or other treatment deemed appropriate by their physicians and were also given as a standard AD treatment one capsule daily of 120 mg ginkgo leaf extract standardized to 24% flavone glycosides and 6% terpene lactones (ginkgolides and bilobalide) (Shanghai Charoma, Shanghai, China) [10]. Curcumin for packets was supplied as a gift from Kancor Flavours, Kerala, India, and curcumin for capsules was purchased from Arjuna Natural Extracts, Kerala, India. We did not measure the curcumin, demethoxycurcumin, and bisdemethoxycurcumin composition of the extracts. There is some natural variation in their ratios, thus the different sources may affect blood levels of curcumin components. To monitor safety, sodium, potassium, urea, creatinine, protein, albumin, bilirubin, alkaline phosphatase, and alanine aminotransferase (ALT/GPT) were measured.

Table 1
Subject characteristics at baseline

	Value
Sex (male)	29%
Age (y)	73.4 ± 8.8 (55–88)
Total cholesterol (mmol/L)	5.5 ± 1.1 (3.5–7.8)
HDL cholesterol (mmol/L)	1.6 ± 0.4 (0.6–2.6)
LDL cholesterol (mmol/L)	3.2 ± 1.0 (1.2–5.2)
Triacylglycerol (mmol/L)	1.6 ± 0.8 (0.6–4.0)

$n = 31$ subjects. All values are mean ± standard deviation; range in parentheses.

2.3. Methods

At baseline, 1 month, and 6 months, blood samples were taken after an overnight fast (except for consumption of the study drug) for measuring serum lipid and lipoprotein concentrations and liver and kidney function parameters.

In a preliminary experiment, curcumin, demethoxycurcumin, and bisdemethoxycurcumin were measured in plasma from one non-demented control subject at various times after oral doses of 4 g curcumin either with or without food. No signal was detected unless the plasma was first treated with glucuronidase, demonstrating that little of the curcumin remained unmodified. The concentration of curcumin (including demethoxycurcumin and bisdemethoxycurcumin) peaked at 250 nmol/L at 1.5 h when taken with food, and at 270 nmol/L at 4 h when taken with only water. At 24 h, the concentration fell to 60 nmol/L. Based on these findings, we decided to measure curcumin by using plasma samples collected from subjects 2–2.5 h after ingestion of curcumin. At 1 month, samples were taken (2–2.5 h after consumption of the study drug) for assay of curcumin and metabolites. Curcumin, demethoxycurcumin, bisdemethoxycurcumin, tetrahydrocurcumin, ferulic acid, vanillic acid, and vanillin were measured in plasma by liquid chromatography–mass spectrometry-mass spectrometry (LC–MS-MS) after treatment with β-glucuronidase (Sigma, St. Louis, Missouri, USA).

2.4. Statistical analysis

Statistical calculations were performed using SPSS 14.0 software (SPSS, Chicago, Illinois, USA). The statistical significance of differences in continuous variables at baseline or of curcumin and metabolites were determined by testing for normality of the distributions using the Kolmogorov–Smirnov test with a Lilliefors significance level, and then using two-sided independent sample *t*-tests or ANOVA tests for normal distributions and Mann–Whitney or Kruskal–Wallis tests otherwise, with values of $p < 0.05$ being considered significant.

Following the recommendation of Vickers and Altman [11], the effects of treatment on total cholesterol, HDL cholesterol, LDL cholesterol, and triacylglycerol were assessed using linear regression models. In these models, the 1 month or 6 month measurement of the above outcomes was the dependent variable, while the predictor variables were the corresponding baseline measurement for the relevant variable, age, sex, and indicator variables for the 1 g dose group and 4 g dose groups. For example: $\mathrm{HDLC}_{1\ \mathrm{month}} = \beta_0 + \beta_1 \times \mathrm{HDLC}_{\mathrm{base}} + \beta_2 \times \mathrm{age} + \beta_3 \times \mathrm{sex} + \beta_4 \times \mathrm{I(dose = 1\ g)} + \beta_5 \times \mathrm{I(dose = 4\ g)} + \varepsilon$. The coefficients for the indicator variables then represent the estimated differences between the placebo group and either the 1 g and 4 g groups in the follow-up measurement of the outcome variable, adjusting for the baseline measurement of that variable, age, and sex. The statistical significance of the

adjusted differences is then determined by testing the null hypothesis that these coefficients are equal to zero. Similar models were used to assess the association between concentrations of curcumin and its metabolites and concentrations of serum lipids and lipoproteins.

Power analysis (using G*Power 3.0 software) using the effect size calculated from a previously published human curcumin study looking at cholesterol, in which 0.5 g curcumin in 10 subjects decreased total serum cholesterol by a mean of 24.8 mg/dl (standard deviation: 18.7 mg/dl) [8], showed that our study of 19 patients receiving curcumin for 1 month had a power of 99.98% to detect a significant ($\alpha = 0.05$) difference. Although the previously published study did not have a placebo control group, possibly exaggerating the effect size, if the effect size were cut in half (for example), the power of our study would still be 78%.

3. Results

3.1. Curcumin doses and lipid effects

Tables 2 and 3 show the baseline values, follow-up values and mean changes in serum lipids and lipoproteins for 1 month and 6 months, respectively. At baseline, serum concentrations of total cholesterol, HDL cholesterol, LDL cholesterol, and triacylglycerol did not significantly differ among treatment groups. Coefficients (beta) for the indicator variables for curcumin doses of 1 g and 4 g from the linear regression models are shown in Tables 2 and 3. The results indicate that there were no significant between-group differences in any of the 1 month or 6 month lipid or lipoprotein measurements after controlling for the baseline measurement, age and sex. When both curcumin treatment groups were combined for comparison with the placebo group, there were also no significant differences at either 1 month or 6 months (data not shown).

3.2. Curcumin absorption and lipid effects

Curcumin, including that used in this trial, actually consists of three similar molecules—curcumin, demethoxycurcumin, and bisdemethoxycurcumin—with the former being the most abundant. Curcumin, demethoxycurcumin, bisdemethoxycurcumin, tetrahydrocurcumin, ferulic acid, vanillic acid, and vanillin were measured after treating plasma with glucuronidase. Of the 22 subjects taking curcumin and providing a blood sample for curcumin measurement, the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin concentrations was 490 ± 740 nmol/L (mean ± S.D.) or 270 ± 620 nmol/L (median ± interquartile range), and the tetrahydrocurcumin concentration was 440 ± 470 nmol/L (mean ± S.D.) or 300 ± 480 nmol/L (median ± interquartile range), while other metabolites were less abundant: 110 ± 80 nmol/L ferulic acid (mean ± S.D.), 50 ± 90 nmol/L vanillic acid (mean ± S.D.), and no detectable vanillin. Capsules (taken fasting with only water on the day of curcumin measurement, rather than with a meal as normally; 10 subjects) produced greater concentrations than did powder (eaten with a small amount of food; 12 subjects) of the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin (mean ± S.D.: 940 ± 920 vs. 120 ± 140 nmol/L; median ± interquartile range: 620 ± 950 vs. 70 ± 260 nmol/L; Mann–Whitney test, $p = 0.0001$), but not of tetrahydrocurcumin, ferulic acid, or vanillic acid. This difference between capsules and powder occurred in both 4 g (*t*-test,

Table 2
Mean serum concentrations of total, LDL, and HDL cholesterol and triacylglycerol for baseline vs. 1 month

	Control group	1 g curcumin group	*P*	4 g curcumin group	*P*
Total cholesterol	$n = 10$	$n = 8$		$n = 11$	
Baseline (mmol/L)	5.41 ± 1.41	5.04 ± 0.61		5.90 ± 0.93	
One month (mmol/L)	5.35 ± 1.61	5.25 ± 0.97		6.06 ± 1.23	
Change (mmol/L)	−0.06 ± 0.62	0.21 ± 0.78		0.16 ± 0.51	
Beta (95% CI)*		0.29 (−0.28, 0.85)	0.30	0.53 (−0.15, 1.22)	0.12
HDL Cholesterol	$n = 10$	$n = 8$		$n = 11$	
Baseline (mmol/L)	1.58 ± 0.31	1.62 ± 0.33		1.56 ± 0.51	
One month (mmol/L)	1.66 ± 0.30	1.74 ± 0.41		1.67 ± 0.44	
Change (mmol/L)	0.08 ± 0.14	0.12 ± 0.31		0.11 ± 0.29	
Beta (95% CI)*		0.17 (−0.08, 0.42)	0.18	0.07 (−0.13, 0.28)	0.46
LDL Cholesterol	$n = 10$	$n = 8$		$n = 11$	
Baseline (mmol/L)	3.10 ± 1.19	2.70 ± 0.79		3.57 ± 0.97	
One month (mmol/L)	3.04 ± 1.49	2.84 ± 0.91		3.66 ± 1.09	
Change (mmol/L)	−0.06 ± 0.78	0.14 ± 0.48		0.08 ± 0.45	
Beta (95% CI)*		0.26 (−0.42, 0.94)	0.44	0.17 (−0.40, 0.74)	0.55
Triacylglycerol	$n = 10$	$n = 8$		$n = 11$	
Baseline (mmol/L)	1.61 ± 0.73	1.59 ± 1.04		1.70 ± 0.78	
One month (mmol/L)	1.45 ± 1.06	1.47 ± 1.20		1.62 ± 0.79	
Change (mmol/L)	−0.16 ± 0.63	−0.12 ± 0.22		−0.08 ± 0.45	
Beta (95% CI)*		0.05 (−0.52, 0.62)	0.86	0.10 (−0.37, 0.56)	0.68

All values are mean ± S.D. CI: confidence interval. *Regression coefficients for dummy variables for 1 g and 4 g doses from multiple linear regression models (with 1-month values for the outcomes) as the dependent variable and baseline values for the outcome, age, and sex as additional covariates. The coefficients represent the between-group difference (1 g vs. placebo and 4 g vs. placebo) in 1-month outcome adjusted for the baseline outcome, age and sex.

Table 3
Mean serum concentrations of total, LDL, and HDL cholesterol and triacylglycerol for baseline vs. 6 months

	Control group	1 g curcumin group	*P*	4 g curcumin group	*P*
Total cholesterol	$n=8$	$n=7$		$n=11$	
Baseline (mmol/L)	5.86 ± 1.37	5.19 ± 0.48		5.90 ± 0.93	
Six months (mmol/L)	5.48 ± 1.33	5.49 ± 0.85		5.85 ± 1.14	
Change (mmol/L)	−0.39 ± 0.77	0.30 ± 0.69		−0.06 ± 0.72	
Beta (95% CI)		0.76 (−0.29, 1.81)	0.15	0.41 (−0.37, 1.18)	0.29
HDL Cholesterol	$n=8$	$n=7$		$n=11$	
Baseline (mmol/L)	1.68 ± 0.45	1.71 ± 0.23		1.56 ± 0.51	
Six months (mmol/L)	1.71 ± 0.45	1.97 ± 0.34		1.65 ± 0.48	
Change (mmol/L)	0.04 ± 0.27	0.27 ± 0.41		0.09 ± 0.37	
Beta (95% CI)		0.22 (−0.26, 0.70)	0.35	0.00 (−0.37, 0.37)	0.98
LDL Cholesterol	$n=8$	$n=7$		$n=10$	
Baseline (mmol/L)	3.41 ± 1.14	2.91 ± 0.54		3.57 ± 0.97	
Six months (mmol/L)	3.23 ± 1.24	2.96 ± 0.71		3.52 ± 1.17	
Change (mmol/L)	−0.19 ± 0.83	0.04 ± 0.61		−0.12 ± 0.59	
Beta (95% CI)		0.27 (−0.71, 1.24)	0.57	0.14 (−0.62, 0.89)	0.71
Triacylglycerol	$n=8$	$n=7$		$n=11$	
Baseline (mmol/L)	1.69 ± 0.80	1.24 ± 0.38		1.70 ± 0.78	
Six months (mmol/L)	1.20 ± 0.52	1.24 ± 0.71		1.86 ± 1.52	
Change (mmol/L)	−0.50 ± 0.93	0.00 ± 0.46		0.16 ± 1.03	
Beta (95% CI)		0.42 (−0.86, 1.69)	0.50	0.63 (−0.34, 1.60)	0.19

All values are mean ± S.D. CI: confidence interval. *Regression coefficients for dummy variables for 1 g and 4 g doses from multiple linear regression models (with 6-month values for the outcomes) as the dependent variable and baseline values for the outcome, age, and sex as additional covariates. The coefficients represent the between-group difference (1 g vs. placebo and 4 g vs. placebo) in 6-month outcome adjusted for the baseline outcome, age and sex.

$p=0.01$) and 1 g (Mann–Whitney test, $p=0.004$) doses. Of all 11 subjects on the 1 g dose, the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin had a mean ± S.D. of 540 ± 1000 nmol/L and median of 190 ± 620 nmol/L; tetrahydrocurcumin had a mean ± S.D. of 430 ± 620 nmol/L and median of 170 ± 450 nmol/L; ferulic acid had a mean ± S.D. of 120 ± 90 nmol/L and median of 130 ± 180 nmol/L; and vanillic acid had a mean ± S.D. of 50 ± 100 nmol/L and median of 0 ± 0 nmol/L. Of all 11 subjects on the 4 g dose, the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin had a mean ± S.D. of 450 ± 390 nmol/L and median of 330 ± 460 nmol/L; tetrahydrocurcumin had a mean ± S.D. of 440 ± 270 nmol/L and median of 410 ± 560 nmol/L; ferulic acid had a mean ± S.D. of 100 ± 60 nmol/L and median of 120 ± 150 nmol/L; and vanillic acid had a mean ± S.D. of 50 ± 90 nmol/L and median of 0 ± 130 nmol/L.

Linear regression models with follow-up lipid and lipoprotein concentrations as outcomes and curcumin components or metabolite concentrations as predictors (adjusted for age, sex, and baseline values) showed a significant positive association between 1 month total cholesterol concentrations and the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin ($p=0.006$, Fig. 1) and with ferulic acid ($p=0.008$) and vanillic acid ($p=0.006$) but a non-significant positive association with tetrahydrocurcumin ($p=0.13$). Associations with 6-month total cholesterol were also significant for ferulic ($p=0.04$) and vanillic acid ($p=0.02$) and not significant for the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin ($p=0.05$). LDL cholesterol at 1 month was significantly and positively associated with the sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin ($p=0.04$) and with ferulic acid ($p=0.04$) and vanillic acid ($p=0.04$), while 6-month triacylglycerol concentrations were positively associated with ferulic acid ($p=0.03$). All other associations between lipid and lipoprotein measurements and these compounds were positive and non-significant.

3.3. Safety

To monitor safety, sodium, potassium, urea, creatinine, protein, albumin, bilirubin, alkaline phosphatase, and alanine

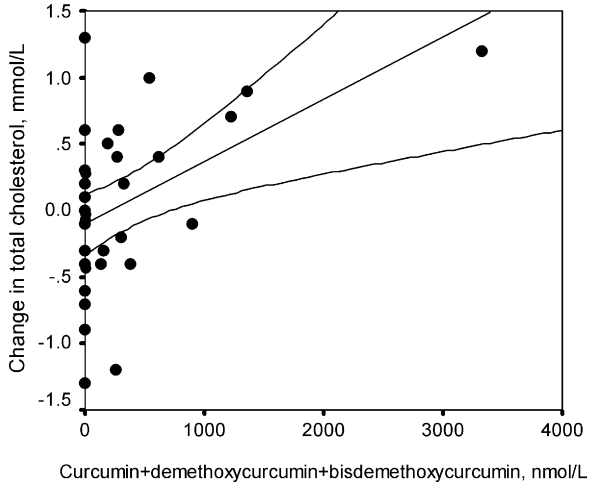

Fig. 1. The sum of curcumin, demethoxycurcumin, and bisdemethoxycurcumin concentrations vs. the change in total cholesterol from baseline to 1 month. Linear regression line ($r^2=0.23$) and 95% confidence intervals are shown. Many subjects had a curcumin concentration of zero because the placebo group was included.

Table 4
Adverse events at any time during the trial

Adverse event	Placebo ($n=13$)	1 g Curcumin ($n=12$)	4 g Curcumin ($n=11$)
Constipation	1	1	0
More delusions	0	1	1
Diarrhea	0	1	0
Nausea	1	0	0
Broken hip	0	1	0
Fall	1	0	0
Pneumonia	1	0	0
Chest infection	1	0	0
Cold	0	1	0
Dizziness	0	0	1
Ankle edema	1	0	0
Bruise	0	1	0
Hearing impairment	1	0	0
Total	7	6	2

aminotransferase (ALT/GPT) were measured. At any time point, only ALT/GPT differed among dose groups by ANOVA at $p<0.05$ (baseline, $p=0.008$; 1 month, $p=0.04$; 6 months, $p=0.06$). The difference appeared to be due to elevated ALT/GPT in the 1 g/day curcumin group at baseline. The elevation did not change during the trial. At 6 months, mean ALT/GPT levels were 16.8 for the placebo group, 24.6 for the 1 g/day curcumin group, and 16.5 for the 4 g/day curcumin group. Adverse events were recorded (Table 4).

4. Discussion

Consuming curcumin did not significantly alter serum cholesterol or triacylglycerol concentrations in this double-blind, placebo-controlled trial, contrary to previous reports of cholesterol-lowering effects in animals and humans [5–8]. However, the absorbed concentration of curcumin was positively associated with cholesterol concentration, suggesting that curcumin may increase rather than decrease cholesterol concentration. This is surprising as it is the opposite of the result expected based on previous studies in animals and humans [5–8]. Among the results in our study, the discrepancy between the non-significance of the effect of curcumin consumption and the significance of the association with curcumin concentrations might be due to individual variability in absorption of curcumin. The number of subjects was small, thus possibly obscuring a small effect of consumed (rather than absorbed) curcumin on cholesterol or triacylglycerol concentrations.

Our study had a small sample size and thus lacks power to detect a small difference due to curcumin treatment. HDL cholesterol exhibited trends toward increases in the 1 g and 4 g groups, and these increases may contribute to elevations in total cholesterol. Curcumin at a near-physiological concentration of 2 μmol/L was reported to induce expression of ABCG1, thus increasing HDL-dependent lipid efflux and plasma HDL cholesterol levels [12]. Curcumin was reported to raise plasma HDL cholesterol in rats [5]. Larger studies in humans may determine whether the small increases in HDL cholesterol that we observed are due to curcumin.

All subjects received ginkgo extract as a standard AD treatment, therefore the lack of an effect on cholesterol of treatment with curcumin vs. placebo occurred on top of ginkgo and all other treatments or diets that subjects received. Evidence is mixed regarding whether ginkgo extract affects cholesterol, with rat studies finding either no effect or a reduction in circulating cholesterol levels [13,14]. If ginkgo does decrease cholesterol in humans, it is possible that a decrease due to curcumin in our study would be harder to detect.

Curcumin in plasma in our study was nearly entirely glucuronidated, and the question arises as to whether glucuronidated curcumin could affect cholesterol levels. Previous studies reported that the vast majority of curcumin in plasma of rodents or humans was glucuronidated or sulfated [15–17], yet cholesterol levels were reported in other studies to be decreased by treatment with curcumin [5–8], and thus by the presence of conjugated curcumin.

A study of curcumin and cholesterol in rabbits used lower doses of curcumin: 1.66 mg/kg in rabbits vs. approximately 10 or 40 times that dose in this trial [6]. LDL cholesterol was increased at the higher dose, 3.2 mg/kg, thus perhaps the doses used in our trial were too high to reduce cholesterol concentrations—high enough perhaps to increase cholesterol, as we observed by correlation with absorbed curcumin concentrations. On the other hand, a study in rats and a previous human study that both reported cholesterol-lowering effects used 50 mg/kg and 500 mg curcumin, respectively, similar to the doses in this trial, thus differences in dosage between this trial and previous studies might not explain the discrepant results [5,8].

The mechanism by which curcumin decreased serum cholesterol concentrations in previous studies is not known. One hypothesis is that curcumin prevented increases in serum cholesterol concentrations in the animal studies by inhibiting dietary cholesterol absorption [5]. The relatively low absorption efficiency of curcumin is consistent with this hypothesis since the much greater curcumin concentration in the gut than in the blood makes an effect of curcumin on cholesterol absorption somewhat more plausible than an effect on cholesterol synthesis. The rat and rabbit studies used diets high in fat and cholesterol to increase serum cholesterol concentrations, whereas subjects in this trial were not selected as suffering hypercholesterolemia and were not fed a special diet [5,6]. The different effects of curcumin in this trial as compared to animal studies might then be explained if the subjects in this trial derived a larger proportion of serum cholesterol from synthesis rather than diet as compared to the animals in previous studies because, according to the above hypothesis, curcumin would affect cholesterol absorption but not synthesis. Administering curcumin to animals or humans receiving diets high or low in cholesterol may provide further evidence to support or refute this hypothesis.

Tetrahydrocurcumin, ferulic acid, and vanillic acid are metabolites of curcumin [15,18,19]. The observation that capsules or fasting produce greater plasma concentrations of curcumin, demethoxycurcumin, and bisdemethoxycurcumin but not of tetrahydrocurcumin, ferulic acid, or vanillic acid

suggests that capsules or fasting may enhance absorption but not metabolism of curcumin as compared to consumption of curcumin with food. Alternatively, food might delay or accelerate absorption of curcumin and metabolites in such a way as to result in lower concentrations at the time when samples were taken; the initial pharmacokinetic study, although of only one subject, indeed suggested that food appeared to accelerate absorption.

Curcumin did not appear to cause side effects (rather, there was a tendency toward fewer adverse events with the 4 g/day curcumin treatment). Thus, human trials of curcumin to test effects on other outcomes may be safely conducted.

To summarize, curcumin did not appreciably affect serum cholesterol or triacylglycerol concentrations unless the absorbed concentration of curcumin was taken into consideration, in which case curcumin modestly increased cholesterol.

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