

Plasma carotenoids in normal men after a single ingestion of vegetables or purified β -carotene¹⁻³

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ABSTRACT Changes in seven plasma carotenoids were measured in 30 men for 11 d after ingesting a single dose of pure β -carotene or a high carotenoid vegetable. A controlled, low-carotenoid diet was fed in a crossover design. Maximum plasma concentrations of β -carotene occurred 24–48 h after dosing with β -carotene (12 or 30 mg) or carrots (270 g). A large intake of broccoli (600 g) or tomato juice (180 g) did not change any plasma carotenoids. We concluded that 1) normal subjects vary widely, three to fourfold, in efficiency of carotenoid absorption; 2) peak plasma response to β -carotene in a capsule occurs at 24–48 h; 3) a large single intake of carrots produces a small increase in plasma β -carotene but single intakes of broccoli or tomato juice do not change plasma carotenoids; and 4) plasma response to pure β -carotene is greater than the response to a similar amount of β -carotene in carrots. *Am J Clin Nutr* 1989;49:1258–65.

KEY WORDS β -carotene, plasma carotenoids, plasma lipids, carotenoid absorption, vegetable carotenoids, interindividual variation

Introduction

Current epidemiologic evidence has indicated a possible association of increased dietary consumption of certain fruits and vegetables with lower rates of several epithelial cancers. Interest has centered on the carotenoid pigments in these foods as the constituents responsible for a cancer-modifying effect (1). These hypotheses have led to renewed efforts in assessing nutritional and metabolic aspects of carotenoids, especially because a recent, more sensitive analytical methodology, namely, high-performance liquid chromatography (HPLC), permits the determination of individual pigments in human plasma (2). Earlier studies of plasma changes after ingesting vegetables were limited to measuring plasma total carotenoid levels in relatively small numbers of subjects (3, 4). In a recent study HPLC was used to measure changes in plasma α - and β -carotene after subjects ingested carrots (5).

In the present study we determined by HPLC the serial changes in seven plasma carotenoid fractions in 30 healthy men maintained on a controlled, low-carotenoid diet after a single ingestion of either a food high in one or more carotenoids or a dose of purified β -carotene. Changes in various other plasma lipid components were also determined.

Subjects and methods

Thirty men aged 20–45 y, 64–88 kg, and in good health as determined by a screening history, medical evaluation, and lab-

oratory tests were studied. None were taking vitamin supplements or medications during the study. All were nonsmokers and had base-line plasma total carotenoid concentrations between 1.3 and 3.7 μ mol. Procedures for this study were approved by Human Studies Review Committees of the United States Department of Agriculture (USDA), The Georgetown University School of Medicine, and National Cancer Institute.

A crossover design (Fig 1) was used so that groups of subjects ingested a single dose of a vegetable or a placebo capsule and a capsule containing β -carotene. The subjects were divided into two strata on the basis of their fasting plasma total carotenoid concentration and then randomly assigned to two major groups to receive a carotenoid dose of either 12 or 30 mg. These two groups were further subdivided into three groups (placebo, vegetables, or pure β -carotene) for the crossover plan (Fig 1). The amounts of vegetables ingested and their contents of major carotenoids, and the amount of purified β -carotene are indicated in Table 1. The serving of vegetable was calculated from our HPLC analysis (below) to provide ~12 or 30 mg of the primary carotenoids in each vegetable (β -carotene in carrots, lutein plus β -carotene in broccoli, and lycopene in tomato juice).

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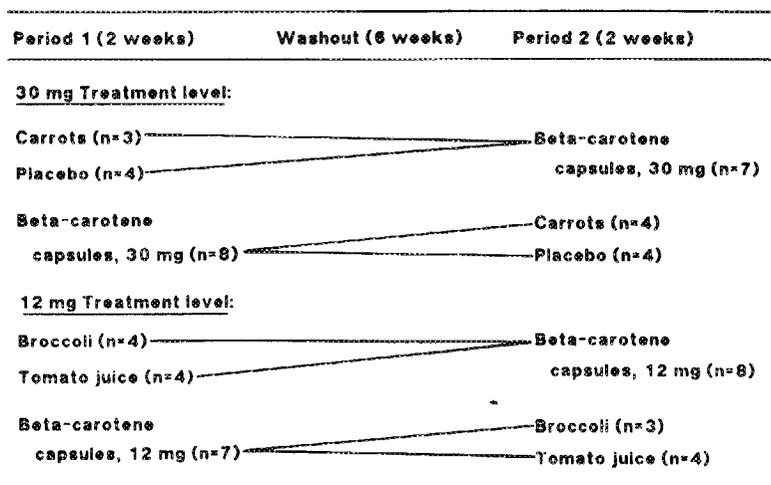


FIG 1. Crossover design of single ingestion of vegetables or β -carotene. Vegetables or β -carotene were fed on day 1 of periods 1 and 2. The subjects ingested self-selected diets between periods 1 and 2 (washout).

Subjects consumed controlled diets low in carotenoids for 3 d before being dosed, continuing for 11 d after. The same 24-h menu was repeated for the 5 d postdosing, then 7-d menus were provided for variability. The treatment, vegetable or capsule, was given on day 0 with breakfast. This meal contained \sim 800 kcal, 45% from fat primarily in bacon, butter, and milk.

Breakfast and dinner from Monday through Friday were eaten under the supervision of a dietitian at the Human Studies Facility of the Beltsville Human Nutrition Research Center. Lunches, the weekend meals, and holiday meals were packaged and taken home. A low calorie, lemon and lime carbonated beverage and instant coffee and tea were provided for consumption while away from the diet facilities. No other food or beverage was allowed. Weighed portions of the foods were adjusted to maintain body weights throughout the study. Menus were designed to be relatively low in carotenoids but not excessively so. The primary sources of carotenoids were the single daily serving of green beans, zucchini squash, lima beans, or peas. The 3000-kcal menu provided 0.5–1.6 mg total carotenoids and 1240–2400 mg RE vitamin A (retinol) by calculation. Forty percent of calories were from fat, 40% from carbohydrate, and 20% from protein. Nutrient content was calculated by using a USDA data set (6), the carotenoid data of Souci et al (7), and the University of Minnesota food tables (8).

Frozen carrots and broccoli (Hanover Foods, Hanover, PA) and canned tomato juice (Giant Foods, Inc, Washington, DC) prepared for household consumption were obtained from a sin-

gle processing lot. The carrots and broccoli were cooked according to those conditions recommended by the food supplier and were served or analyzed immediately after preparation. The β -carotene capsules contained dry gelatin beads of 10% β -carotene compounded with butylated hydroxytoluene (BHT), butylated hydroxyanisole (BHA), and sodium benzoate preservatives (Hoffman-LaRoche, Nutley, NJ). The β -carotene was 95% pure by HPLC analysis. The placebo contained the beads without β -carotene and was similar in appearance to the β -carotene capsules.

Fasting blood samples were obtained in each experimental period 3 d before dosing (the beginning of the controlled diet), day 0 (dosing day), and days 1, 2, 3, 4, 7, and 11 after the test dose. Two nonfasting blood samples were collected at 8 and 32 h after the test dose. After the 11th day subjects were free-living for 6 wk and then crossed over to begin the second treatment period as indicated in Figure 1.

Food analysis

Five units of each vegetable or tomato juice (0.45 kg bag or 1 L bottle) were randomly selected and prepared for consumption. Each unit of cooked vegetable was homogenized and analyzed immediately. About 2 g tomato juice or 4 g cooked broccoli was weighed, β -apo-8'-carotenol (internal standard) in hexane was added, and the sample extracted with acetone and petroleum ether (20 mL each). The organic layer was removed and the water and acetone layer was further vortexed four times with petroleum ether. The organic layers were combined and evaporated to dryness. The residue was dissolved in 50 mL HPLC solvent and filtered before HPLC analysis.

Because of the fibrous nature of carrots, \sim 4 g cooked carrots along with β -apo-8'-carotenol were ground for 4 min with 30 mL acetone in an Omnimixer[®] (Sorvall Instruments, Newtown, CT). An equal volume of petroleum ether was added and stirred and the liquid decanted onto a vacuum filter. The food was reextracted three to four times until the liquid phase was colorless. The combined extracts were evaporated to 15 mL, transferred to a screw-cap tube, extracted with petroleum ether, and prepared for HPLC analysis as described above.

The chromatography system consisted of three Beckman model 114M solvent pumps with a Beckman model 421 controller. Samples (20 μ L) were injected into a 3 cm \times 4.6 mm guard cartridge (Speri-5-C₁₈, 5 μ m particles; Brownlee Labs,

TABLE 1
Carotenoid content of the various supplements

Source	Amount	Carotenoids	Amount
			mg
Carrots	272 g	β -carotene	29
		α -carotene	9
Broccoli	600 g	Lutein	5
		β -carotene	6
Tomato juice	180 g	Lycopene	12
Purified β -carotene	capsule	β -carotene	30
Purified β -carotene	capsule	β -carotene	12

Santa Clara, CA) and a stainless steel 25 cm × 4.6 mm separation column (Microsorb C₁₈, 5 μm particles, Rainin Instrument Co, Woburn, MA). A Beckman model 164 variable wavelength detector was interfaced to a Shimadzu Model CR3A integrator and data processor.

Isocratic separation of carotenoids in carrot extracts and β-apo-8'-carotenal was accomplished with methanol (15%), acetonitrile (65%), methylene chloride (10%), and hexane (10%) at a flow rate of 1 mL/min. Chromatographic runs (~25 min) for carrot extracts were monitored at 445 nm for β-apo-8'-carotenal and at 450 nm for α- and β-carotene. Lycopene in tomato juice was quantified with the above isocratic eluent at a flow of 1.4 mL/min and was monitored at 445 nm for the internal standard and at 470 nm for lycopene. The separation of the primary carotenoids in extracts of cooked broccoli used an isocratic mixture of methanol (15%), acetonitrile (75%), methylene chloride (5%), and hexane (5%) at 0.8 mL/min for 12 min followed by a gradient that terminated at 28 min with a composition of methanol (15%), acetonitrile (40%), methylene chloride (22.5%), and hexane (22.5%). Chromatographic runs (~20 min) were monitored at 445 nm for lutein and β-apo-8'-carotenal and 450 nm for β-carotene.

Standards used were *all-trans* α- and *all-trans* β-carotene and lycopene (Sigma, St Louis, MO) and β-apo-8'-carotenal (Fluka Chemical Corp, Hauppauge, NY). Lutein was isolated from kale by using thin layer chromatography (9). All solvents were HPLC-grade (Fisher Scientific, Pittsburgh, PA). Purity of standards was checked by HPLC; at least 96% of the detectable carotenoids were in a single component. Final concentrations were established spectrophotometrically by using extinction coefficients (10).

Plasma analysis

Blood was collected in all-plastic syringes (Monovet®, Sarstedt, Princeton, NJ) with 4.5 U sodium heparin/mL blood. Samples were protected from light and centrifuged within 1 h for 20 min at 2260 × g at 12 °C. Aliquots of plasma were stored at -70 °C until analyzed. Triglycerides, cholesterol, and high-density lipoprotein (HDL) cholesterol were analyzed on a Centrifchem Analyzer® (Baker Instruments Corp, Allentown, PA) by using standardized procedures. The low-density lipoprotein (LDL) cholesterol was estimated by the formula of Friedewald et al (11). Plasma retinol, α-tocopherol, individual carotenoids, and their totals were determined by the modified HPLC methods of Bieri et al (2, 12) as reported by Craft et al (13). Although all seven fractions obtained by this method were used to calculate total carotenoids, only the five major peaks, α-carotene, β-carotene, cryptoxanthin, lycopene, and lutein plus zeaxanthin (hereafter referred to as lutein), are reported in detail. For all HPLC analyses a laboratory plasma control sample was analyzed daily to verify precision. Chromatograms were individually checked for proper integration of peak areas.

Two isocratic HPLC systems were run in parallel for the plasma analyses. They consisted of the Beckman model 114M solvent delivery system, Beckman model 160 UV/VIS detector equipped with 436nm (carotenoids) and 280-nm (retinol and α-tocopherol) filters, a Beckman 450 Data System/Controller, an Altex Model 270A injector (Beckman Instruments, Inc, San Ramon, CA), and a Rheodyne Model 7125 injector (Rainin Instrument Co). For the retinol and tocopherol analysis, a Supelcosil C₁₈ 5 μm (250 × 4.6 mm) column paired with a matching precolumn (Supelco, Bellefonte, PA) was used. Carotenoid analysis was performed by using an Ultrasphere® C₁₈-5 μm column (Beckman Instruments, Inc) with an NH₂ precolumn (Alltech Associates, Deerfield, IL). Flow rate for both systems was 1.8 mL/min and run time was < 12 min per sample. Reti-

nol, α-tocopherol, and α-tocopheryl acetate (Sigma) were used as standards for the retinol and tocopherol analysis. Crystalline α-carotene, β-carotene, lycopene (Sigma), zeaxanthin, cryptoxanthin, and echinenone (Hoffman-LaRoche) were used as standards for carotenoid analysis. All solvents were HPLC-grade (Fisher Scientific, Silver Spring, MD). All samples for each subject were run on a single day to minimize within-day variation. Graphical checks for outliers were based on visible detection of unusual values. These assays were repeated. If discrepancy was observed, outlier values were deleted and treated as missing values.

Statistical methods

Summary statistics were calculated for the carotenoids (response variables) for each of the six groups. The distributions of response variables were compared by using the *t* test for two independent groups and analysis of variance (ANOVA) for more than two independent groups, as well as the nonparametric counterparts, the Wilcoxon two sample rank sum test, and the Kruskal-Wallis test (14). Paired *t* tests and the signed rank test were used to compare for significant changes above base line. Estimates of association among response variables were based on Spearman correlation coefficients. Reported significance levels were unadjusted for multiple testing.

An adaptation of the response variable, area under the curve (AUC), was calculated by using the trapezoidal rule to approximate the integration formula (15). The plasma concentration-time curve was represented by a series of trapezoids formed by straight lines connecting each of the observed values and the base formed by a straight line parallel to the *x* axis through the pretreatment observed value. The integral of the AUC is the sum of the series of trapezoids formed by these points. Areas determined by plasma values that decreased below the baseline level were subtracted.

Results

Base-line values for plasma cholesterol, triglycerides, vitamins A and E, and carotenoids before both treatment periods are shown in Table 2. All blood indices were within acceptable normal ranges. The high correlations between periods for the various carotenoid levels indicate a relatively constant pattern for individuals while on their free-living diets. Tests for a period effect, ie, a difference in response variables for test dose 1 vs test dose 2, were not significant. The absence of a period effect allowed pooling data for the two periods when comparing treatment groups.

Plasma carotenoids of control subjects fed the low-carotenoid diet for 14 d (3 d predosing, 11 d placebo) did not uniformly decline. A representative plot of the five known carotenoids is shown in Figure 2. Lycopene, the most abundant pigment in these men (Table 2), declined progressively in all subjects throughout the 2-wk period. Lutein, the next most abundant carotenoid, showed a moderate increase in six of the nine subjects. β-carotene remained essentially constant as did α-carotene and cryptoxanthin. The two unknown peaks (not shown) remained constant.

For a comparison of plasma responses to the various intakes of carotenoids, three response variables were used: difference between the maximum concentration

TABLE 2
Concentrations of plasma lipid components for the two treatment periods and their correlations before starting experimental diets*

Measurement	Test dose 1	Test dose 2	r†
Cholesterol (mmol/L)	4.8 ± 0.9	4.8 ± 0.9	0.92
Triglycerides (mmol/L)	0.75 ± 0.3	0.77 ± 0.2	0.77
HDL cholesterol (mmol/L)	1.2 ± 0.3	1.3 ± 0.3	0.74
LDL cholesterol (mmol/L)	3.2 ± 0.7	3.2 ± 0.8	0.93
Retinol (μmol/L)	2.3 ± 0.3	2.4 ± 0.4	0.90
Tocopherol (μmol/L)	26.1 ± 5.2	26.4 ± 5.1	0.83
Lutein plus zeaxanthin (nmol/L)	434.2 ± 161.7	411.4 ± 131.9	0.84
Precryptoxanthin (nmol/L)	103.1 ± 41.6	97.6 ± 34.3	0.66
Cryptoxanthin (nmol/L)	153.7 ± 95.8	135.6 ± 56.1	0.68
Lycopene (nmol/L)	842.1 ± 329.8	842.1 ± 355.8	0.79
α-Carotene (nmol/L)	80.1 ± 65.2	93.2 ± 70.8	0.77
β-Carotene (nmol/L)	340.9 ± 177.0	329.8 ± 163.9	0.86
Total carotenoids (nmol/L)	2180 ± 558.9	2142 ± 521.6	0.74

* $\bar{x} \pm SD$; $n = 30$ except for $n = 29$ for retinol in test period 2. Periods were 8 wk apart.

† Spearman correlation coefficient; probability < 0.0001 except total carotenoids: probability < 0.0005.

reached after dosing and the predosing (base line) concentration; the relative maximum change (maximum change divided by the base-line concentration); and the AUC. In subjects given capsules containing β -carotene or those fed carrots, plasma β -carotene showed peak concentrations after 24–48 h followed by a slow decline that did not return to base line after 11 d. Figure 3 shows two

contrasting responses to 30 mg β -carotene. Subject A had a maximum increase in plasma β -carotene at 48 h of only 249.6 nmol/L (85% above base line) whereas subject B increased 668.8 nmol/L (272% above base line) at 48 h. This figure also shows the marked decline in lycopene, no change in α -carotene, and a moderate increase in lutein as noted in the placebo group (Fig 2).

Table 3 gives the absolute changes in plasma β -carotene concentration at peak absorption time, the percentage increase over base line, and AUC for the six groups given β -carotene capsules, carrots, broccoli, tomato juice, and the placebo capsule. No significant change occurred in the placebo group. Thirty milligrams of pure β -carotene produced a peak response only 1.6 times greater than the response of 12 mg, which suggests a reduced absorption efficiency at the higher intake. The 29 mg of β -carotene in carrots produced a plasma response that was only about one-fifth to one-seventh that produced by 30 mg pure β -carotene. All pair-wise comparisons of the three β -carotene response variables were statistically significant ($p < 0.01$, unadjusted for multiple comparisons). Not shown in Table 3 is the change in α -carotene in the carrot group in which a plasma increase of 61.5 nmol/L \pm 7.4 ($\bar{x} \pm SEM$; 101% \pm 24.5 above base line) occurred, which was statistically significant ($p < 0.001$).

There was a wide variation in plasma response within each group as shown by the range (Table 3). In both β -carotene-capsule groups there was a three- to fourfold difference between the lowest and highest responses and a similar difference existed in the carrot group. No relationship between base-line concentration and maximum change was apparent.

It is of interest that of the three poorest responders for carrots (absolute increase), two also had the poorest response to the 30-mg β -carotene capsule when they

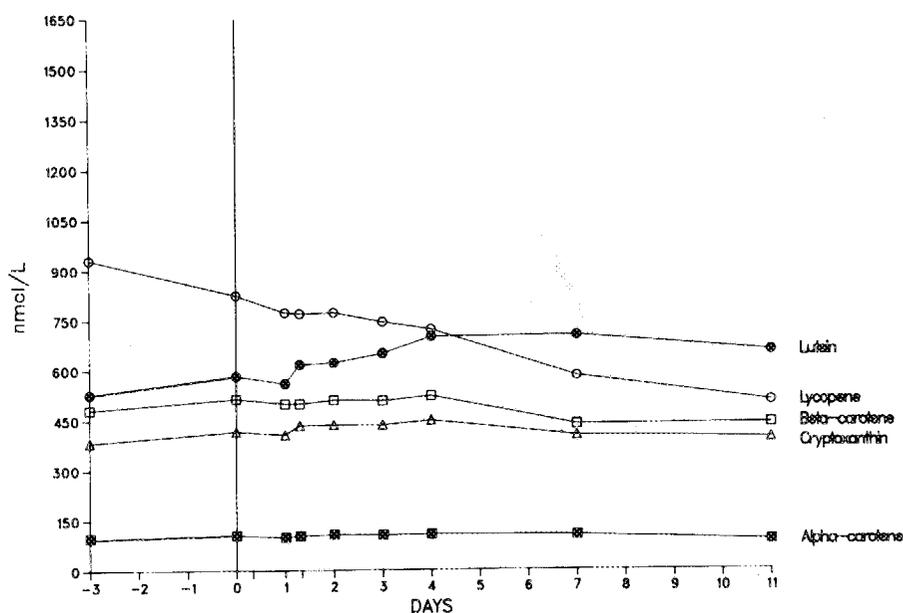


FIG 2. Profile of plasma carotenoids with time for a representative subject in the placebo group. For clarity, the two unidentified peaks are not shown.

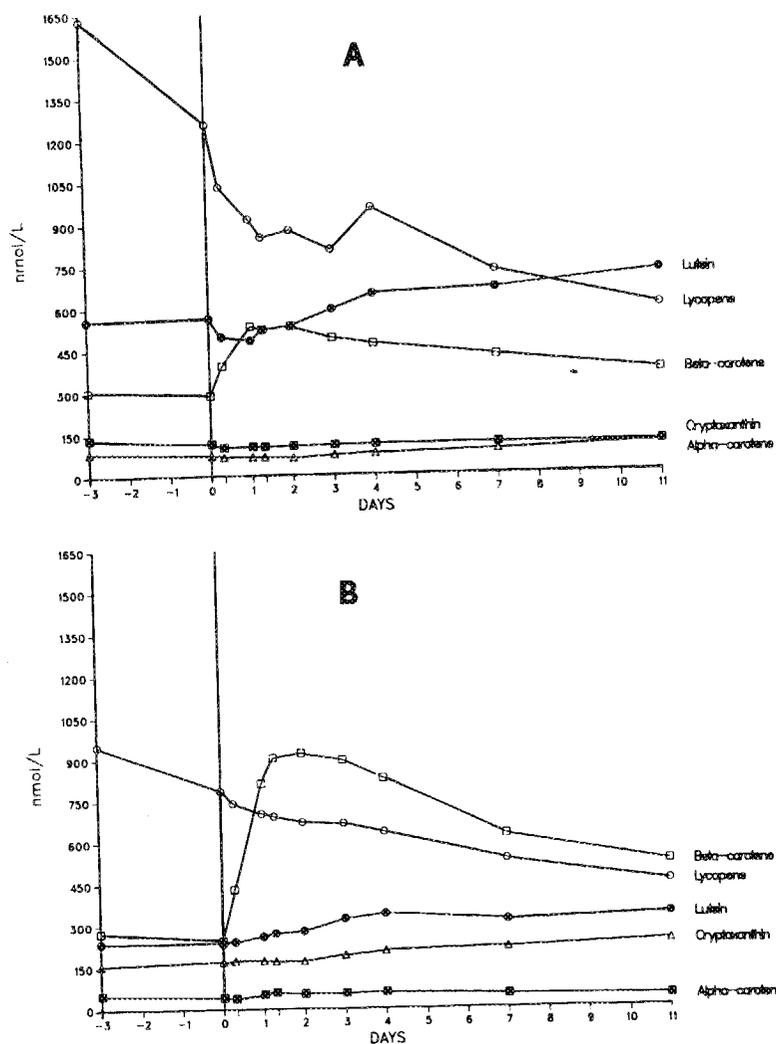


FIG 3. Profile of plasma carotenoids for two subjects (A and B) after ingesting 30 mg pure β -carotene.

crossed over. Their base-line values were in the intermediate range. These limited data suggest not only a large variation between individual responses to a β -carotene load but also a consistency in the magnitude of an individual's response.

The 600-g intake of broccoli, providing 6 mg β -carotene and 5 mg lutein, had no significant effect on the plasma β -carotene concentration (Table 3). The maximum change in lutein concentration in this group, however, was 160.0 ± 19.3 nmol/L, a highly significant ($p < 0.001$) change from base line. This change was not significantly different, however, from that in the placebo group (172.2 ± 33.4 nmol/L) (data not shown in Table 3).

A graphic presentation of the mean changes in plasma β -carotene during the 11-d experimental period, shown in Figure 4, illustrates the peak absorption for the β -carotene capsule groups over the first 24–48 h. Carrots produced a prolonged maximum from 24–96 h, suggesting slower absorption than pure β -carotene. The 30-mg dose

of β -carotene gave a considerably higher sustained plasma level than did the 12-mg dose.

Tomato juice containing 12 mg lycopene had no effect on the progressive decline in concentration of this carotenoid observed uniformly in all groups (data not shown), as illustrated in Figure 2. Three other carotenoid peaks in the HPLC chromatograms were also determined: β -cryptoxanthin and two unidentified peaks, one termed pre- β -cryptoxanthin (2). These carotenoids either declined gradually, were stable, or showed a moderate non-significant change throughout the 11-d period regardless of group treatment.

Studies in which the absorption of a compound is measured by the plasma response often use the area under the concentration-time curve to compare efficiencies. Even though our carotenoid response curves did not return to base line in the 11-d period (Fig 2), we estimated the areas by using a trapezoidal rule (see Statistical Methods). The results (Table 3) gave relationships among the groups that were reasonably similar to those

TABLE 3
Changes in plasma concentrations of β -carotene after ingesting a single dose of pure β -carotene or vegetables

Treatment	Number of subjects	Maximum change from base line* nmol/L	Percent maximum change	AUC†
Placebo	8	-31.7 ± 24.2 (-143.5, 50.3)	-5.6 ± 7.6 (-27.8, 23.1)	-343 ± 199 (-1480, 210)
30 mg β -carotene	15	538.4 ± 42.9 ‡ (247.8, 862.6)	219.7 ± 27.2 (43.3, 406.1)	4843 ± 474 (1723, 7610)
Carrots§	7	113.6 ± 18.6 ‡ (52.2, 180.7)	35.5 ± 4.1 (15.2, 45.5)	697 ± 156 (189, 1182)
12 mg β -carotene	15	329.8 ± 29.8 ‡ (130.4, 529.1)	110.4 ± 18.8 (26.3, 290.0)	2550 ± 384 (-474, 4712)
Broccoli	7	1.8 ± 18.6 (-61.5, 46.6)	0.6 ± 5.9 (-20.5, 22.6)	11 ± 109 (-390, 407)
Tomato juice	8	-44.7 ± 28.0 (-177.0, 48.4)	-4.5 ± 8.8 (-37.7, 36.0)	-369 ± 218 (-1380, 190)

* $\bar{x} \pm \text{SEM}$; ranges in parentheses.

† Area under the curve.

‡ Significantly greater than base-line values ($p < 0.001$).

§ Contained 29 mg β -carotene.

|| Contained 6 mg β -carotene.

for maximum concentration over base line. For example, the areas for β -carotene in the 30-mg and 12-mg capsule groups were 4843 and 2550, respectively, a ratio of 1.9. The ratio of incremental concentrations in Table 3 was 1.6 for these groups. Similarly, the β -carotene area for the carrot group was 697, which gave a ratio of 0.14 when compared with the 30-mg capsule group (4843). This compares with a ratio of 0.21 from the maximum increases in Table 3 (113.6:538.4).

Discussion

The preexperiment (initial) range of most carotenoid concentrations of our study population were similar to those reported recently by Stacewicz-Sapuntzakis et al (16) for a group of 55 middle aged men in the United States. The notable exception was lycopene, which in our younger population was more than double that of the middle aged men. This may be due to a high consump-

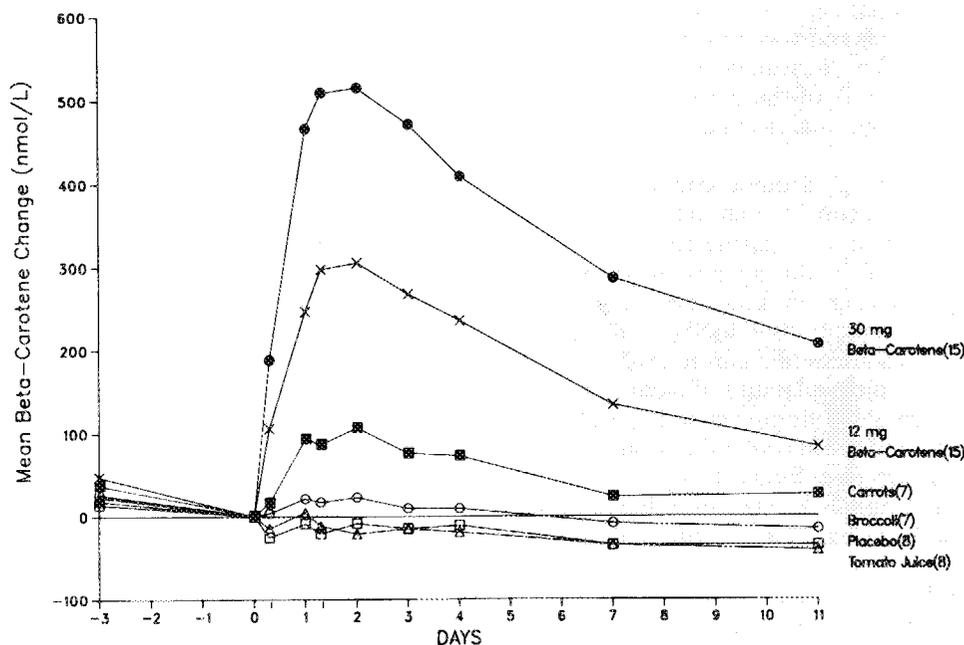


FIG 4. Mean plasma concentration of β -carotene for each treatment group during the 14-d period (3 d before and 11 d after dosing).

tion of tomato-containing foods, such as pizza, by our population. Of particular interest were the high correlations between the initial concentrations of individual carotenoids for the two treatment periods, which indicated that individuals under free-living conditions appear to maintain a characteristic carotenoid profile. The relatively poor plasma responses to a single, large portion of either cooked carrots, broccoli, or canned tomato juice suggest that an individual's steady state carotenoid profile is a result of long-term dietary pattern and is little influenced by occasional large intakes of one food high in a particular carotenoid pigment.

The breakfast given with the treatment was relatively high in fat to facilitate absorption. The vegetable doses were intended to be large amounts that might be expected, on the basis of the literature, to produce measurable plasma increases but as noted above, the servings of tomato juice and broccoli were insufficient. Witschi et al (17) calculated that a group of American men consumed 0.5–6.5 mg β -carotene/d, thus, our 30-mg dose of β -carotene was probably more than a reasonable dietary intake as were also the very large servings of cooked carrots and broccoli. Of particular interest was the large variation in response within each treatment group. This variation is most likely because of absorption differences among the subjects. Earlier studies (3, 17) have also noted this irregularity in plasma response to β -carotene dosing.

The relatively slow rate of appearance of β -carotene in the blood, whether from pure β -carotene or from carrots (Fig 3), has been observed by others (3, 4, 18). Although there was a clear response at 8 h, the mean maximum concentration did not occur until 24–48 h after dosing. This response time is much longer than that of other fat-soluble nutrients such as retinol or tocopherol. Other studies (5, 19) noted shorter response times and it is not evident why this discrepancy occurs. Cornwell et al (4) showed that chylomicrons and very-low-density lipoproteins peaked ~ 7 h after β -carotene dosing but that LDLs, which carry the bulk of the plasma carotenoids, peaked at 24–40 h. The gradual decline indicates a slow turnover of β -carotene.

The 29 mg of natural β -carotene contained in the cooked carrots was nearly equal to that in the 30-mg capsules. However, the maximum plasma response of the carrot group was 21% of the 30-mg capsule group and 34% of the 12-mg capsule group. Likewise, previous investigators noted that carotenoids in foods are much less available than purified sources of β -carotene (20, 21). In the case of carrots, the bioavailability of β -carotene can be increased by mechanical homogenization (22). Although our data suggest that bioavailability from purified β -carotene is greater than from foods, dark green and yellow vegetables may contain numerous other components that have a beneficial impact on maintaining health (23).

We were unable to identify plasma lipid components that might predict subject response to a dose of β -carotene. Initial concentrations of retinol, tocopherol, triglycerides, and cholesterol were not good indicators for plasma β -carotene response for these normolipemic sub-

jects. None of these indices showed a strong correlation with the plasma β -carotene increase after ingesting pure β -carotene.

Differences in responses between subjects are most probably due to varying degrees of absorption and transport and to a lesser degree to the efficiency in converting β -carotene to retinol. The conditions of this study do not allow us to distinguish between these variables.

The controlled diet, which had no tomato products, was a definite change from the tomato-containing, self-selected diets the subjects normally consumed as indicated by the declining lycopene concentrations throughout the study for all subjects. There were no significant differences between placebo and tomato juice groups. The very high initial lycopene concentrations (932–1863 nmol/L) in seven of the subjects in these two groups decreased almost linearly (Fig 2) and permitted an estimation of the apparent half-life. At the end of each 14-d experimental period during which a low-carotenoid diet was ingested, these high lycopene concentrations had declined 51% (range 43–63) indicating a half-life of ~ 14 d. In contrast, lycopopenia as manifested by orange-yellow skin has been described in a subject who consumed ~ 2 L tomato juice for several years (24). This quantity is more than 10-fold the amount of the single ingestion by subjects in this study.

The placebo group demonstrated an increase in plasma lutein beginning 4–5 d after starting the control diet. This was probably due to the daily serving of peas during this period. Subsequent analysis in our laboratory of peas from a different source found 3.2 mg lutein per serving, which was considerably higher than the value from food composition tables. In contrast, the 5 mg lutein in one large serving of broccoli did not give a response significantly different from the placebo response.

This study has shown that normal men under carefully controlled dietary conditions show a wide variation in carotenoid utilization as indicated by the change in plasma concentration after a single ingestion of a carotenoid source. We confirmed the findings of previous studies by showing that purified β -carotene is more efficiently absorbed than is a similar amount present in vegetables. The data also show that the steady state plasma concentration of individual carotenoids is changed only slightly by a single, large intake of a green leafy or yellow vegetable. Comparison of the plasma carotenoid profiles separated by 6 wk of ad libitum conditions showed that each individual had a relatively stable, characteristic plasma profile. The identification of several men who were consistently poor absorbers suggests that plasma carotenoid levels may not accurately indicate dietary intake. Our current analysis of a long-term study of plasma carotenoid response with daily carotenoid ingestion, in which carotenoderma has been reported (25), should provide quantitative information on the diet-plasma carotenoid relationship. 

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