Dietary Factors That Affect the Bioavailability of Carotenoids[1]

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ABSTRACT Carotenoids are thought to contribute to the beneficial effects of increased vegetable consumption. Various dietary factors have an effect on the bioavailability of carotenoids. The type of food matrix in which carotenoids are located is a major factor. The bioavailability of β-carotene from vegetables in particular has been shown to be low (14% from mixed vegetables) compared with that of purified β-carotene added to a simple matrix (e.g., salad dressing), whereas for lutein, the difference is much smaller (relative bioavailability of 67% from mixed vegetables). Processing, such as mechanical homogenization or heat treatment, has the potential to enhance the bioavailability of carotenoids from vegetables (from 18% to a sixfold increase). The amount of dietary fat required to ensure carotenoid absorption seems low (~3–5 g per meal), although it depends on the physicochemical characteristics of the carotenoids ingested. Unabsorbable, fat-soluble compounds reduce carotenoid absorption, and interaction among carotenoids may also result in a reduced carotenoid bioavailability. Research into the functional benefits of carotenoids should consider the fact that the bioavailability of β-carotene in particular is one order of magnitude higher when provided as a pure compound added to foods than when it is present naturally in foods. J. Nutr. 130: 503–506, 2000.

KEY WORDS: • carotenoids • bioavailability • vegetables • processing • fat • interaction

Carotenoids are thought to contribute to the inverse relationship between fruit and vegetable consumption and the risk of coronary heart disease and some types of cancer (1). To increase our understanding of the potential benefits of carotenoids, it is important to obtain more insight into their bioavailability from foods and the factors that determine this bioavailability. The absorption of carotenoids includes several steps, as described previously (2). Factors that may interfere with the rate of each of these steps will affect the overall bioavailability of the carotenoids ingested. The mnemonic "SLAMENGHI" describes these factors: *S*pecies of carotenoids, *L*inkages at molecular level, *A*mount of carotenoid, *M*atrix, *E*ffectors, *N*utrient status, *G*enetics, *H*ost-related factors and *I*nteractions among these variables (3). This review focuses on the effect of dietary factors, i.e., *M*atrix and *E*ffectors, on the bioavailability of carotenoids. **Figure 1** shows an overview of these factors and the steps at which they may interfere with carotenoid absorption.

Bioavailability of carotenoids from different food matrices. Disruption of the food matrix and release of carotenoids constitute the first step in carotenoid absorption. Most of the studies on the effect of the food matrix on carotenoid bioavailability have determined the plasma response of carotenoids after supplementation with vegetables or fruits and compared that with the response to supplementation with pure carotenoids. A measure of "relative carotenoid bioavailability" can be obtained by dividing the plasma responses that are induced by vegetables or fruit consumption and corrected for differences in intake by those induced by pure carotenoid supplementation. This assumes a proportional linear relationship between the extent of carotenoid absorption and the plasma response. It should be noted, however, that the plasma response may also be affected by the duration of supplementation, the rate and extent of tissue uptake and release, and metabolism of carotenoids.

The relative bioavailability of β-carotene from vegetables compared with purified β-carotene ranges between 3 and 6% for green leafy vegetables, 19 and 34% for carrots and 22 and 24% for broccoli (4–9). In one study, broccoli and green peas induced a larger β-carotene response in plasma than whole-leaf and chopped spinach, despite a 10 times lower β-carotene content in the former vegetables (9). In another study, β-carotene from fruits was found to be 2.6–6 times as effective in increasing plasma concentrations of retinol and β-carotene than green leafy vegetables (10). These differences may result from differences in intracellular location of carotenoids. In leaves, they are present in chloroplasts, whereas in fruits, and possibly also other parts of the plant, carotenoids are located in chromoplasts. This has led to the speculation (10) that chloroplasts may be less efficiently disrupted in the intestinal tract than chromoplasts.

Few data are available on the relative bioavailability of carotenoids other than β-carotene from vegetables. We recently showed (11) that the relative bioavailability of lutein from a diet supplemented with a variety of vegetables is much greater than that of β-carotene (i.e., 67 and 14%, respectively). The same was found (8) for the relative bioavailability of lutein and β-carotene from spinach (i.e., 45 and 5.1%, respectively). The release of lutein into an aqueous environment is probably higher than that of β-carotene because of its lower lipophilicity compared with β-carotene. In addition, the bioavailability of lutein appears to be lower from green leafy vegetables than from other vegetables, although the differences are less pronounced than those of β-carotene (9).

The presence of dietary fiber in vegetables and fruits may explain in part the lower bioavailability of carotenoids from plant foods. It has been suggested that fiber interferes with micelle formation by partitioning bile salts and fat in the gel phase of dietary fiber. However, results to date are contradictory (8,12,13).

Disruption of the food matrix. Not only the intracellular location, but also the intactness of the cellular matrix may be a determinant of carotenoid bioavailability from vegetables and fruits. As early as 1948, Van Zeben and Hendriks (14) reported that homogenization improved the bioavailability of

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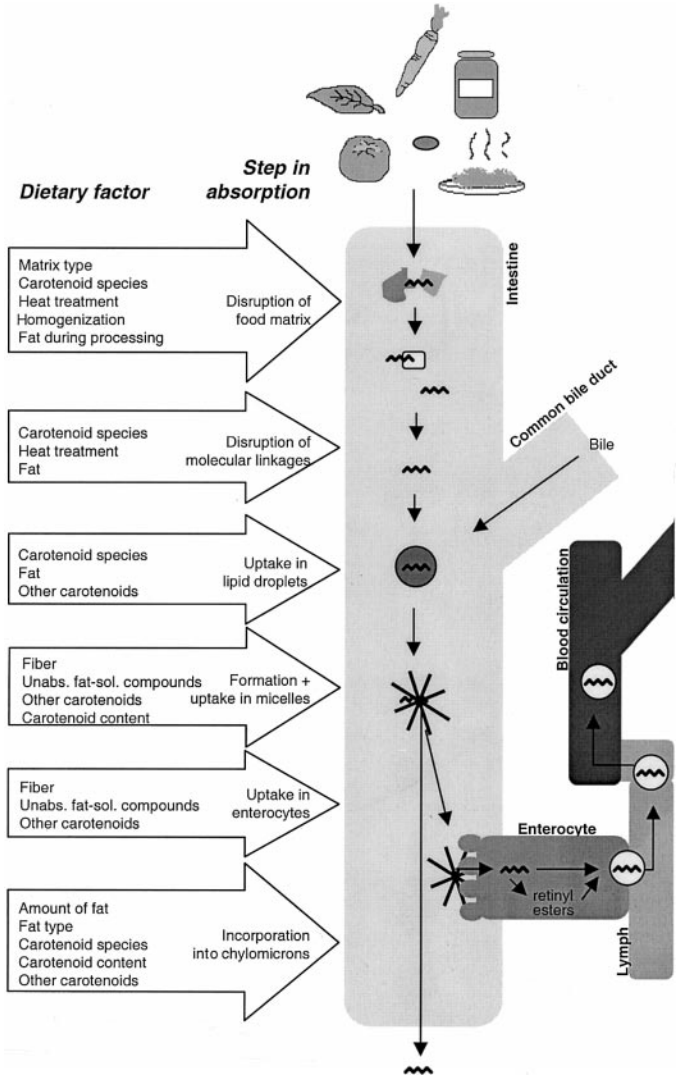

FIGURE 1 Steps of carotenoid absorption and dietary factors that affect carotenoid absorption.

β-carotene from carrots in humans. In a later study with vitamin A–depleted boys who consumed raw grated carrots or carrot juice for 2 wk, plasma concentrations of retinol and β-carotene were found to be slightly higher in the group consuming carrot juice (15). However, no statistical evaluation was presented in that study. Törrönen et al. (7) showed a 70% difference in bioavailability of β-carotene from raw carrots vs. carrot juice consumed by well-nourished adult females for 6 wk, although the difference was not significant.

There are indications that disruption of the matrix affects the bioavailability of various carotenoids differentially. The plasma response of lutein was significantly increased by ~14% when spinach was consumed as chopped spinach instead of as whole leaf spinach, whereas the plasma response of β-carotene was not affected (9). There are several explanations for this finding. First, the different lipophilic character of the two carotenoids results in a greater release of lutein in response to homogenization. Second, it may well be that homogenization releases both carotenoids to the same extent but that lutein inhibits β-carotene absorption (16,17). In contrast, however, Castenmiller et al. (8) found that disruption of the matrix of spinach by enzymatic treatment enhanced the plasma response of β-carotene (by 60–70%) but not that of lutein. Because the bioavailability of lutein from spinach, relative to a supplement, is higher than that of β-carotene (8), it can be speculated that the vegetable matrix is a less important determinant of the bioavailability of the less lipophilic lutein than that of β-carotene.

Some studies have found that cooking enhances the carotenoid content measured in vegetables, possibly due to increased extractability of carotenoids from the vegetable matrix (18,19). This increased extractability due to heat treatment may be associated with improved bioavailability of carotenoids from the vegetable matrix. Homogenized carrots, carrot juice and carrot chromoplasts were fed to preruminant calves and ferrets to test this hypothesis (20,21). However, steaming of the carrot products did not result in increased levels of α- or β-carotene in serum, adrenals or liver. Because homogenized carrots were used as starting material, heat treatment may have had no further destructive effect on the matrix in which the carotenoids were located.

Processing of tomatoes into tomato paste includes both mechanical homogenization and heat treatment. There is evidence that this process is very effective in increasing lycopene bioavailability. The lycopene response in plasma or triglyceride-rich lipoproteins was 22–380% greater after consumption of tomato paste than that for the same amount of lycopene consumed as fresh tomatoes (22,23). These results support the suggestion of Giovannucci et al. (24) that the association between consumption of various tomato products and risk of prostate cancer depends on the bioavailability of lycopene. Rock et al. (25) reported recently that the plasma response of β-carotene was enhanced after consumption of pureed, cooked carrots and spinach compared with that after consumption of the vegetables in their raw, unhomogenized form (twofold higher increase in plasma β-carotene).

Amount of dietary fat present. A second step in the absorption process of carotenoids that may affect their bioavailability involves the incorporation of released carotenoids into mixed micelles. Among other factors, formation of these micelles is dependent on the presence of fat in the intestine. Therefore, ingestion of fat along with carotenoids is thought to be crucial.

Various studies assessed the importance of dietary fat in comparison with its complete absence at the moment of ingestion of β-carotene (26–28). Under these circumstances, absorption of β-carotene seems to be suboptimal because the increases in plasma concentrations improved substantially when fat was added to the test meals. However, from the findings of Jayarajan et al. (26), it appears that 5 g of fat in a meal is already sufficient to ensure carotenoid uptake. They found no difference in improvement of the vitamin A status when 5 or 10 g of dietary fat was added to spinach, whereas 0 g fat resulted in less improvement. A recently reported study of Jalal et al. (29) indicated that the cut-off point lies between 3 and 5 g of fat. They observed a significantly smaller increase in serum retinol if 3 g of fat was added to a sweet potato snack than if 18 g of fat was added. This is in line with our own results (unpublished data), which show that a very low fat carotenoid–enriched spread added to a meal (3 g fat/meal) was as effective in enhancing plasma α-carotene and β-carotene concentrations in plasma as a full-fat carotenoid–enriched spread (35 g fat/meal). However, for lutein, which was added as lutein esters, the plasma response was ~100% higher after consumption of the full-fat spread. The low amount of fat may have limited the solubilization of lutein esters in the fat phase and/or the release and activity of esterases and lipase. These enzymes are required for the hydrolysis of lutein esters, a step very likely to be crucial for their absorption.

Type of fat and digestibility of fat-soluble components present in the diet. It has been shown that if subjects consume fat-soluble components that are not absorbable or absorbable only to a limited extent, their plasma carotenoid concentrations may

TABLE 1

Estimation of the quantitative effects of various dietary factors on the bioavailability of carotenoids[1]

Dietary factor	n[2]	Carotenoid		
		β-Carotene	Lutein	Lycopene
Matrix type (carotenoids in oil = 1.0)				
Mixed vegetables (11)	10–22	0.14 ± 0.011	0.67 ± 0.08	NA
Green leafy vegetables (6)	56–62	0.04	NA	NA
Whole-leaf spinach (8)	10–12	0.04	0.45	NA
Whole-leaf spinach (9)	26–67	0.03 ± 0.5	NA	NA
Carrots (4)	7–15	0.19	NA	NA
Carrots (5)	5	0.19	NA	NA
Carrots (7)	12–13	0.26	NA	NA
Broccoli (4)	5	0.22	NA	NA
Broccoli (9)	26–67	0.74 ± 0.64	NA	NA
Green peas (9)	26–67	0.96 ± 0.71	NA	NA
Matrix disruption (undisrupted vegetables = 1.0)				
Chopped vs. whole-leaf spinach (9)	26	1.0	1.18	NA
Liquefied vs. whole-leaf spinach (8)	12	1.69	1.0	NA
Homogenized vs. whole carrots (7)	13	[1.7][3]	NA	NA
Homogenized vs. whole carrots (14)	7–10	5.9	NA	NA
Tomato paste vs. raw tomatoes (22)	5	NA	NA	4.8[4]
Tomato paste vs. raw tomatoes (23)	9	NA	NA	1.2–1.5
Homogenized and heated vs. raw carrots and spinach (25)	8	3.1	NA	NA
Amount of dietary fat (high amount of fat = 1.0)				
0 g fat vs. 5 g fat present in carotenoid-supplemented meal (26)	22–26	0.48[5]	NA	NA
0 g fat vs. 10 g fat present in carotenoid-supplemented meal (26)	22–26	0.48[5]	NA	NA
5 g fat vs. 10 g fat present in carotenoid-supplemented meal (26)	22	1.0	NA	NA
3 g fat vs. 18 g fat present in meal containing sweet potatoes (29)	41–43	0.63[5]	NA	NA
3 g fat vs. 36 g fat present in carotenoid-supplemented meal (unpublished data)	15	1.0	0.43 ± 0.062[6]	NA
Indigestible fat-soluble compounds (regular dietary fat = 1.0)				
3 g/d sucrose polyester vs. regular dietary fat with main meal (30)	26–27	0.80 ± 0.03	NA	0.62 ± 0.05
12.4 g/d sucrose polyester vs. regular dietary fat with main meal (30)	21	0.66 ± 0.02	0.80 ± 0.04	0.48 ± 0.05
18 g/d sucrose polyester vs. regular dietary fat at various times during day (31)	65–67	0.73	0.81	0.77
Dietary fiber (no dietary fiber = 1.0)				
12 g/d citrus pectin added to carotenoid-supplemented meal (12)	7	0.42	NA	NA
Beet root fiber added to liquefied spinach (8)	12	1.0	1.0	NA

[1] Values are presented as means ± SEM or as mean. The factors were calculated from changes in plasma or serum concentrations of carotenoids, unless otherwise stated. The plasma or serum carotenoid response after the treatment stated was divided by the plasma or serum carotenoid response after the treatment, which was taken as a reference at 1.0 (identified between brackets for each dietary factor), and corrected if necessary for differences in carotenoid intake. In case no change was expected from the reference treatment (e.g. in case of indigestible vs. regular fat), the factors were calculated as the percentage of change from baseline, corrected if necessary for the change in the control group. A factor <1.0 indicates that the bioavailability of carotenoids is reduced compared with the reference chosen; a factor >1.0 indicates an enhanced carotenoid bioavailability.

[2] Number of subjects per treatment.

[3] Value is not significantly different from 1.0 ($\alpha = 0.05$).

[4] Calculated from area under the curve of the carotenoid response in triglyceride-rich lipoproteins.

[5] Calculated from changes in serum concentrations of retinol.

[6] Lutein was present as lutein esters.

NA, not assessed.

decrease substantially. Sucrose polyester, a nonabsorbable fat replacer, decreased plasma levels of carotenoids by 20–120%, depending on the amount of sucrose polyester and the type of carotenoid (30–32). The largest decreases were found for the most lipophilic carotenoids (i.e., lycopene and β-carotene). Apparently, carotenoids released from the food matrix were incorporated into the nonabsorbable sucrose polyester rather than into the micelles that were formed from dietary fat. This is supported by the fact that the effect was less pronounced if participants were allowed to consume snacks containing sucrose polyester at will (31) rather than together with the major dietary sources of carotenoids, i.e., during the main meal (30,32).

Borel et al. (33) recently reported that the type of fat present in the diet also influences carotenoid bioavailability. This could not be explained by a reduced absorbability of the fat itself. Medium-chain triglycerides are absorbed primarily via the portal vein; thus the chylomicron formation is low after a meal containing only these types of triglycerides. Borel et al. (33) showed that if β-carotene was added to such a meal, the incorporation of β-carotene into chylomicrons was also low, compared with the addition of β-carotene to a meal containing long-chain triglycerides.

Interactions between carotenoids. Interactions at the intestinal level may reduce absorption of either of the carotenoids. Competition for absorption may occur at the level of micellar incorporation, intestinal uptake, lymphatic transport or at more than one level. On the other hand, simultaneous ingestion of various carotenoids may induce a antioxidant-sparing effect in the intestinal tract and thus result in increased levels of uptake of the protected carotenoids. A similar phenomenon may occur within the body, with respect to both sparing of antioxidant capacity and provitamin A activity, and thus result in an enhanced status of carotenoids. Van den Berg (34) recently reviewed this topic. It can be concluded that, in general, long-term β-carotene supplementation has limited or no effect on plasma or serum concentrations of other carotenoids. However, the supplements may have been ingested at times during the day other than those at which foods rich in carotenoids were consumed. Studies on simultaneous ingestion of carotenoids indicate that β-caro-

tene may interfere with absorption of lutein (16,17) and canthaxanthin (35,36), resulting in reduced bioavailability of lutein and canthaxanthin by β-carotene and of β-carotene by lutein. Further research is required to identify the mechanisms behind these interactions.

Conclusion and Implications. Estimates of the quantitative effect of each factor, as far as possible, on the bioavailability (i.e., the responses in plasma, serum or triglyceride-rich lipoproteins) of carotenoids are presented in **Table 1**. The type of food matrix in which carotenoids are located determines their bioavailability to a great extent. Processing, such as mechanical homogenization or heat treatment, has the potential to enhance the bioavailability of carotenoids from vegetables. However, as shown in particular for β-carotene, this will not result in bioavailability similar to that observed for the pure compound. The amount of dietary fat required to ensure carotenoid absorption seems to be low (3–5 g per meal), although it depends on the physicochemical characteristics of the carotenoids ingested. Unabsorbable, fat-soluble compounds reduce carotenoid absorption and interaction among carotenoids may also result in a reduced carotenoid bioavailability.

In the future, more research will be required to improve our understanding of the bioavailability and function of carotenoids. The development and use of carotenoids labeled with stable isotopes may contribute quantitative data on the extent to which carotenoids are absorbed from different food matrices. The studies presented in this review show that different types of vegetables may vary substantially with respect to the bioavailability of carotenoids. The underlying differences, e.g., cellular location of carotenoids, should be explored further. Research into the functional benefits of carotenoids should consider the fact that the bioavailability of β-carotene in particular is one order of magnitude higher when provided as a pure compound added to foods than when it is present naturally in foods. This should be taken into account when deciding on the amount of carotenoids to be provided, whether it is added to foods or used as a pharmaceutical preparation. Processing, such as mechanical homogenization or heat treatment, has the potential to enhance the bioavailability of carotenoids from vegetables. This may be applied in the development of foods with enhanced carotenoid bioavailability. A possible negative effect of such conditions on the content of other, more vulnerable, micronutrients should be taken into account. If novel food ingredients are developed, in particular if they are fat-soluble and absorbable only to a limited extent, attention should be paid to a possible negative effect on the bioavailability of carotenoids.

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