Review

Intestinal absorption of dietary carotenoids

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The assessment of carotenoid bioavailability has long been hampered by the limited knowledge of their absorption mechanisms. However, recent reports have elucidated important aspects of carotenoid digestion and absorption. Disruption of food matrix and increasing amounts of fat seem to enhance the absorption of carotenes to a larger extent than that of xanthophylls. Comparing different carotenoid species, xanthophylls seem to be more easily released from the food matrix and more efficiently micellized than the carotenes. On the other hand, carotenes are more efficiently taken up by the enterocytes. However, carotenoid emulsification and micellization steps are largely affected by the food matrix and dietary components, being the main determinant of carotenoid bioavailability from foodstuffs. Although the intestinal uptake of carotenoids has been thought to occur by simple diffusion, recent studies reported the existence of receptor-mediated transport of carotenoids in enterocytes. Comparisons between the intestinal absorption of a wide array of carotenoids would be useful to elucidate the absorption mechanism of each carotenoid species, in view of the recent indications that intestinal carotenoid uptake may involve the scavenger receptor class B type I and possibly other epithelial transporters. The unraveling of the whole mechanism underlying the absorption of carotenoids will be the challenge for future studies.

Keywords: Bioavailability / Carotenoids / Digestion / Intestinal absorption

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1 Introduction

Carotenoids are pigments responsible for the yellow, orange, and red colors of many fruits and vegetables, although in photosynthetic tissues their color may be masked by the presence of chlorophylls. Some foodstuffs from animal origin such as eggs, shrimp, lobster, and salmon also contain carotenoids; however, those are derived from dietary sources, as carotenoid biosynthesis occurs only in plants, algae, and some bacteria and fungi. Apart from the carotenoids present in major foodstuffs, the human diet includes carotenoids from spices such as saffron, paprika, and annatto, which have been used since ancient times imparting color (from carotenoids) and flavor (from carotenoid-derived compounds such as β-ionone and safraonal) to many dishes. Due to their intense orange to red colors, carotenoids are also widely used as colorants in the food processing industry.

The structure of carotenoids is based on a C_{40} isoprenoid backbone that may be acyclic or have one or both ends modified into rings. The hydrocarbon carotenoids are classified as carotenes, and those containing at least one oxygen atom are the xanthophylls (Fig. 1). The extended system of conjugated double bonds in the carotenoid backbone is largely responsible for their color and antioxidant properties. Till date, approximately 600 carotenoids have been identified, with ~60 being found in the human diet and a few of those being present in the blood in measurable amounts (Fig. 1). Owing to its two unsubstituted β-ionone rings at the ends of the isoprenoid chain, β-carotene is the carotenoid with the highest provitamin A activity, while other carotenoids such as α-carotene, γ-carotene, and β-cryptoxanthin have lower provitamin A activities. The relative abundance and the function as a vitamin A precursor have centered most of the carotenoid research on β-carotene. The other hand, the interest on the absorption and the function of other carotenoids arose from epidemiological data supporting the protective effects of carotenoid-rich fruits and vegetables against many degenerative diseases, including cardiovascular diseases, age-related macular degeneration, and some types of cancer. The health-promoting effects of carote-
The antioxidant effects of carotenoids are based on the physical quenching of singlet oxygen [1, 2] and the scavenging of peroxyl radicals, particularly at low oxygen tension. In addition, the anticancer effects of carotenoids may be explained by the modulation of various transcription systems, changing the expression of many proteins participating in cell proliferation, growth factor signaling, and gap junctional intercellular communication [2, 3].

The bioavailability of carotenoids is extremely variable, being influenced by many dietary and physiological factors. Castenmiller and West [4] have developed the mnemonic SLAMENGHI, which describes all factors that are likely to influence the carotenoid bioavailability, as follows: species of carotenoids, molecular linkage, amount of carotenoids consumed in a meal, matrix in which the carotenoid is incorporated, effectors of absorption and bioconversion, nutrient status of the host, genetic factors, host-related factors, and interactions. Many studies have addressed the influence of dietary factors on carotenoid bioavailability from food sources, as reviewed by van het Hof et al. [5].

The absorption of dietary carotenoids involves several steps starting with the mechanical and enzymatic disruption of the food matrix, release of the carotenoids, followed by their incorporation into lipid droplets of the gastric emulsions. The carotenoids are then transferred from the lipid droplets to mixed micelles produced by the action of bile salts, biliary phospholipids, dietary lipids, and their hydrolysis products. After the solubilization in mixed micelles, carotenoids are absorbed by the intestinal cells, packed into chylomicrons and secreted to the lymphatic system. Each step of the carotenoid absorption may be influenced by multiple factors, thus making difficult the task of assessing the effects of each factor on the overall carotenoid bioavailability [6, 7]. This review describes the dietary factors that affect carotenoid absorption as well as the mechanisms

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**Figure 1.** Structures of major carotenoids found in human plasma.
involved in the uptake of carotenoids by the intestinal epithelium, focusing on the recent advances in this field.

2 Effects of food matrix

The bioavailability of carotenoids from supplements is usually higher than that of those embedded in the matrix of fruits and vegetables. One of the factors accounting for that is the degree of food matrix disruption. Food processing, including heat, mechanical and enzymatic treatments, facilitates the disruption of the cell wall and organelles, releasing the carotenoids from the food matrix and promoting their dispersion in the gastrointestinal tract.

Carotenoid mass-balance calculations after the ingestion of a single meal by ileostomy volunteers indicated a higher absorption and plasma response of \( \beta \)-carotene from cooked (65.1 \( \pm \) 7.4\%) than from raw ground carrots (41.4 \( \pm \) 7.4\%) [8]. The thermal and mechanical processing of vegetables were also reported to enhance plasma \( \beta \)-carotene concentration in humans after a 4-wk intervention with cooked/pureed versus raw/sliced carrots and spinach [9], and in preruminal calves after a 7-day feeding period with steamed versus raw carrot slurries [10]. Similarly, the chylomicron lycopene postprandial response (0–12 h, area under the curve, AUC) was significantly higher after consumption of tomato paste versus fresh tomatoes [11]. In another trial, only the subjects consuming the thermally processed tomato juice showed an increase of serum lycopene levels, as opposed to those who received the unprocessed juice [12]. Furthermore, homogenization of canned tomatoes stimulated \( \beta \)-carotene and lycopene responses (AUC) in the plasma triacylglycerol-rich lipoprotein (TRL) following a single meal; increasing carotenoid AUC values were observed after the ingestion of canned tomatoes with none (54.9 \( \pm \) 11.0 nmol \( \cdot \) h \( \cdot \) L\(^{-1} \) \( \beta \)-carotene; 2.51 \( \pm \) 6.5 nmol \( \cdot \) h \( \cdot \) L\(^{-1} \) lycopene), mild (72.2 \( \pm \) 11.0 nmol \( \cdot \) h \( \cdot \) L\(^{-1} \) \( \beta \)-carotene; 16.6 \( \pm \) 6.5 nmol \( \cdot \) h \( \cdot \) L\(^{-1} \) lycopene), and severe (88.7 \( \pm \) 11.0 nmol \( \cdot \) h \( \cdot \) L\(^{-1} \) \( \beta \)-carotene; 23.0 \( \pm \) 6.5 nmol \( \cdot \) h \( \cdot \) L\(^{-1} \) lycopene) homogenization treatments [13]. In addition, the dose-normalized increment in the plasma lycopene AUC, following the ingestion of tomato soup was found to be greater than that after drinking tomato juice [14].

Castenmiller et al. [15] determined the serum \( \beta \)-carotene response in volunteers during a 3-wk dietary intervention with whole, minced, or enzymatically liquefied spinach. The enzymatic disruption of cell walls in the liquefied spinach noticeably increased the serum \( \beta \)-carotene response, compared to that of whole and minced spinach preparations. Serum lutein responses were higher than that of \( \beta \)-carotene and were little influenced by the changes in the vegetable matrix [15]. The lower plasma appearance of \( \beta \)-carotene compared to that of lutein could be due to its partial cleavage into retinol. However, by using intrinsically labeled carotenoids in kale, it was shown that even considering the conversion to retinol, the bioavailability of kale \( \beta \)-carotene was still lower than that of lutein [16]. A detailed in vitro simulation of the solubilization of \( \beta \)-carotene and lutein (from carrot juice and homogenized spinach) to the oil phase during the gastric digestion was reported by Rich et al. [17]. The solubilization of \( \beta \)-carotene from raw carrot juice was above 40\% (of the initial amount), while that from raw spinach was below 10\%. This reflects a different microenvironment surrounding the carotenoid molecules in the raw spinach chloroplasts compared to that of carotene crystals (free and membrane-bound in carotene bodies and chromoplasts) in carrot juice [17]. The process of blanching, which disrupted most of the organelles, had little influence on the solubilization of \( \beta \)-carotene from carrot juice, but it enhanced the solubilization of both \( \beta \)-carotene and lutein from homogenized spinach by more than 60\%. However, van het Hof et al. [18] reported similar dose-normalized plasma responses of \( \beta \)-carotene and lutein in subjects consuming diets containing a variety of cooked vegetables. Therefore, the consumption of assorted vegetables, in addition to the cooking step, may reduce the overall inhibitory effects of the food matrix on carotenoid bioavailability, especially on the highly lipophilic \( \beta \)-carotene and lycopene.

3 Effects of dietary components

3.1 Amount of dietary fat

The variability of \( \beta \)-carotene and lutein bioavailability from different studies, may reside on the amount of fat used in the simulated digestions in vitro and in the meals used on the intervention trials. The amount of fat in the diet is known to improve carotenoid bioavailability. However, increasing amounts of dietary fat do not seem to influence all carotenoid species equally, enhancing the absorption of highly lipophilic carotenoids (e.g., \( \beta \)-carotene, \( \alpha \)-carotene, and lycopene) to a larger extent than that of less lipophilic xanthophylls (e.g., lutein and zeaxanthin). After a simulated digestion of spinach puree containing 10\% corn oil, the micellar fractions of lutein, and \( \beta \)-carotene tended to be equivalent [19]. However, at 2.1–3.5\% fat the micellization of \( \alpha \)-carotene and \( \beta \)-carotene after an in vitro digestion was shown to be approximately one-half of that of lutein [20–22]. With a limited oil phase, the micellization of the highly lipophilic carotenones is inhibited, while the relatively polar xanthophylls seem to travel more freely from the food matrix to the lipid and micellar phases of the digesta. Such effect is also reflected in the absorption of carotenoids by human subjects. The amount of dietary fat (3 or 36 g) in carotenoid-enriched spreads consumed with a meal did not affect plasma \( \beta \)-carotene and \( \alpha \)-carotene responses (nmol/L per \( \mu \)mol of added carotenoid); however, the plasma lutein response was higher when lutein esters were consumed with high-fat spreads [23], denoting a higher requirement of fat for the optimal
solubilization of the relatively lipophilic lutein esters. In addition, the limited amount of oil may imply a low accessibility of xanthophylls esters by the hydrolytic enzymes.

For a satisfactory absorption of carotenoids from relatively unprocessed vegetables, the requirement of fat seems to be higher than that for cooked or more homogenized meals [24, 25]. The addition of avocado or avocado oil to a very low-fat meal, based on fresh vegetables, significantly increased the absorption of carotenoids (lutein, \( \beta \)-carotene, and lycopene) in the order of their lipophilicity [24]. Also, lycopene, \( \beta \)-carotene, and lutein responses were higher in subjects that consumed salads with full-fat dressings (containing 28 g of oil) compared to those who consumed the same salad with a low-fat dressing (containing 6 g of oil), indicating that 6 g fat may not be enough for an adequate absorption of carotenoids from relatively low-processed vegetable matrices [25].

The amount of fat also affects the absorption of carotenoids in the absence of food matrix. Lymph duct-cannulated rats infused with canthaxanthin-containing emulsions showed a linear increase of canthaxanthin absorption as the amount of lipid in the emulsion increased [26]. In human, the dietary fat may also promote carotenoid absorption by stimulating bile secretion, raising the luminal concentration of bile salts that act as surfactants in the formation of mixed micelles [27]. Earlier reports on the effects of dietary fat on \( \beta \)-carotene absorption have been reviewed by Ribaya-Mercado [28].

### 3.2 Sucrose polyesters, plant sterols, and phospholipids

Sucrose polyesters possess organoleptic characteristics resembling the vegetable oils; however, they are not cleaved by gastric and pancreatic lipases (PLs), thus being used as nonabsorbable fat replacers. When the sucrose polyester come in contact with the carotenoids and other lipophilic substances in the lumen, a portion of the nutrient partitions into the sucrose polyester phase and becomes unavailable to solubilization by the mixed micelles [29, 30]. This portion is then excreted from the body together with the unabsorbed sucrose polyester, impairing the absorption of carotenoids and reducing their concentration in the serum [31–34]. The octanol–water partition coefficient of most carotenoids is in the same range as of other phytochemicals whose absorption rates are also affected by the ingestion of sucrose polyesters [30]. However, xanthophylls such as lutein, zeaxanthin, and \( \beta \)-cryptoxanthin are about 100 times less lipophilic than carotenones. In line with that, the inhibition of carotenoid absorption by sucrose polyesters is less severe for xanthophylls than for \( \beta \)-carotene, as demonstrated by serum carotenoid concentrations in humans consuming sucrose polyesters [31, 32].

Plant sterols and stanols are effective in lowering plasma cholesterol levels in humans [35]. However, due to the lipophilic nature of carotenoids, they may compete with the plant sterols and stanols for the solubilization in mixed micelles. The absorption of \( \beta \)-carotene was reduced to \(~50\%\) in Caco-2 cells by the presence of 20 \( \mu \)M \( \beta \)-sitosterol in the medium [36], where both \( \beta \)-carotene and \( \beta \)-sitosterol were completely dissolved. Therefore, plant sterols may compete with carotenoids also in their site of absorption [36]. Likewise, \(~50\%\) reductions in the plasma TRL \( \beta \)-carotene were observed in humans after the addition of 2.2 g of free or esterified plant sterols to a single meal [37]. These effects also seem to be dose-dependent [38] and partially linked to the simultaneous decrease of cholesterol in LDL [35, 39, 40] which are the major carriers of carotenoids in the human serum.

Phospholipids, especially phosphatidylcholine (PC), are present in the diet either as a natural component of the food matrix or as an emulsifier/stabilizer in processed foods [41, 42]. Additionally, the bile constitutes a large physiological pool of phospholipids (mainly PC) [43]. Although both dietary and biliary PC are important for the emulsification of dietary lipids in the digestive tract, the presence of PC in mixed micelles inhibits the absorption of carotenoids by human intestinal Caco-2 cells [44, 45] and mice [46], most likely by shifting the carotenoid partition into the micellar phase. However, during the normal digestive process, most of the dietary and biliary PCs are hydrolyzed by phospholipases, producing lysophosphatidylcholines (LysoPCs) which restore or even enhance \( \beta \)-carotene and lutein absorption by Caco-2 cells and experimental animals [44, 46, 47]. Thus, under normal dietary conditions, PCs are not likely to interfere in the carotenoid absorption. In agreement with that, the TRL lutein response from eggs, which naturally contain high amounts of PC, was found to be higher than that from supplements or spinach [48].

### 3.3 Dietary fiber

Dietary fibers have been considered as a factor contributing to the low bioavailability of carotenoid from fruits and vegetables. However, the complex and uneven structures of the fibers and the presence of other phytochemicals have hampered the elucidation of the whole mechanism involved in the fiber effect on the carotenoid absorption.

The intake of dietary fiber, especially those classified as soluble, has been associated with cholesterol-lowering effects [49]. Similar to cholesterol, carotenoids are of lipophilic nature, thus their absorption can also be affected by some types of fibers, as observed in humans [50, 51], rats [52], chicks [53], and Mongolian gerbils [54]. Among those studies, citrus pectin has been suggested as the fiber with the strongest inhibitory effects on \( \beta \)-carotene absorption [53, 55]; however, the effects of fibers on the absorption of other carotenoids are less known. Although various fibers suppressed postprandial responses of \( \beta \)-carotene, lycopene, lutein, and canthaxanthin in humans, how the interaction
between the measured effects of various fiber types on the bioavailability of different carotenoids proceeded remained unclear [50]. On the other hand, the degree of inhibition of β-carotene absorption by pectins seems to be related to their structure, more specifically to their methoxyl content, in line with the lower utilization of β-carotene in chicks consuming high and medium methoxyl pectins, compared to those receiving a low methoxyl pectin [53].

4 Carotenoid species

As mentioned in Section 3, food matrix and dietary components affect the bioavailability of each carotenoid species at a different degree, with stronger effects on the more lipophilic carotenes. Similarly, the transfer of less lipophilic xanthophylls from the lipid emulsion droplets to micelles seems to occur with a better efficiency compared to the transfer of more lipophilic carotenes. The transfer of carotenoids from emulsions to micelles in vitro was found to be inversely correlated to the carotenoid partition coefficient in octanol/water, which is an index of lipophilicity [56]. Lutein from processed vegetables was also found to be more efficiently micellized than β-carotene under conditions mimicking the duodenal environment [57]. The more efficient micellization of xanthophylls, compared to that of carotenes, may explain the discrepancy between the higher bioavailability of xanthophylls from foodstuffs, and their lower absorption by Caco-2 cells when presented as mixed micelles in the apical medium [44], as described in the Section 5.

Xanthophylls are detected in the human plasma in the free form, even after being ingested in the esterified form [23, 58, 59]. Carotenoid esters are cleaved during the digestive process, most likely by the action of the pancreatic cholesterol esterase (EC 3.1.1.13) [60] which is also responsible for the hydrolysis of dietary cholesterol esters, esters of fat-soluble vitamins, phospholipids, and mono-, di- as well as triglycerides. Except for low-fat dietary conditions, the serum response to free or esterified xanthophylls is fairly equivalent [58, 59, 61], supporting the existence of an effective enzymatic cleavage system for xanthophylls esters in the gastrointestinal tract [58]. The uptake of xanthophylls in the esterified form by Caco-2 cells is minimal, while the free xanthophylls are largely absorbed indicating the necessity of the ester cleavage step for an efficient absorption of xanthophylls [62].

5 Mechanisms of absorption by the intestinal cells

5.1 Simple diffusion

Once incorporated into mixed micelles in the intestinal lumen, the carotenoids are “ready” to be absorbed by the small intestinal epithelium (enterocytes). Mixed micelles are formed in the duodenum by the detergent action of bile salts and the hydrolysis of the emulsified lipid (triaclyglycerols) by PLs and colipase, yielding monoacylglycerols and free fatty acids. Phospholipids (mainly PCs) from biliary and dietary sources, also present in the lipid emulsion droplets, are cleaved by the pancreatic phospholipase A2 (PLA2), producing lysophospholipids (mainly LysoPCs). The micellar structure has a disk-like shape with an approximate diameter of 4–60 nm, consisting of an outer shell of bile salts surrounding a core formed by more hydrophobic lipids (Fig. 2). The LysoPCs, monoacylglycerols, and free fatty acids resulting from lipid hydrolysis are located in the micelle core, probably with their polar heads oriented to the aqueous interface. The carotenoids are also accommodated in the micelle core, where they remain until their uptake by the enterocytes. The steps involved in carotenoid transfer from mixed micelles to the enterocytes are not completely understood. However, an important step for the absorption of carotenoids [44] and other lipophilic compounds [63, 64] is the cleavage of phospholipids by PLA2. Although the cleavage of phospholipids is not a prerequisite for the formation of mixed micelles, the presence of PC in the mixed micelle inhibits carotenoid absorption by rats [46] and Caco-2 cells [44, 45]. The action of PLA2 can restore the absorption of lipophilic compounds that were inhibited by PC [44, 63], indicating the importance of PLA2 on the absorptive process of carotenoids, along with other lipophilic compounds such as cholesterol, vitamin A, and vitamin E.

The uptake of carotenoids by the enterocytes has been thought to occur by simple diffusion, similarly to many other dietary lipids. Experimental evidences for the simple diffusion mechanism were provided by linear responses to increasing the carotenoid concentrations in perfused rat intestines [65] and in rat small intestinal cells [66]. Additionally, only a small inhibition of 14C-labeled carotenoid uptake was observed when those cells were incubated at 4°C (compared to their incubation at 37°C), or when an excess of unlabeled carotenoid was added to the medium [66]. According to the simple diffusion mechanism, the micelles migrate through the unstirred water layer to the brush border membrane, the carotenoid then leaves the micellar structure and diffuses through the membrane into the cytoplasm of enterocytes (Fig. 2). Cell membranes are basically formed by lipid bilayers, thus in the absence of a specific transporter, lipophilic substances can diffuse more easily through the membrane than the hydrophilic ones. Similarly, the uptake of the relatively lipophilic carotenes by Caco-2 cells is higher than that of the less lipophilic xanthophylls [44, 67, 68]. Moreover, a strong linear relationship between carotenoid uptake and their lipophilicity (defined as their partition coefficient in 1-octanol/water) was obtained from the uptake rates of 15 different carotenoids [44].
5.2 Receptor-mediated transport

Recent studies reported the existence of receptor-mediated transport of β-carotene and lutein in the apical membrane of enterocytes, with strong indications for the involvement of the scavenger receptor class B type I (SR-BI) in this transport [69–71]. SR-BI, a member of the ATP-binding cassette (ABC) transporter super-family, mediates the selective uptake of cholesterol and cholesteryl esters by the liver and other steroidogenic tissues from HDL particles, therefore exerting an important role in the reverse cholesterol transport [72, 73]. The SR-BI was also found to be expressed in the murine and the human small intestinal epithelium [72, 74], where it mediates part of the choles-
terol uptake [75]. The first evidence that SR-BI was important for carotenoid transport was observed in Drosophila, whose gene encoding a SR-BI-homologous protein was essential for the cellular uptake of carotenoids in this species [76]. The absorption of dietary β-carotene and α-tocopherol by mice [70, 77], and the uptake and transport of β-carotene [71] and lutein [69] by Caco-2 cells seem to be at least partly mediated by SR-BI.

In contrast with most of the protein-mediated transport, the carotenoid absorption via SR-BI seems to occur without energy expenditure. The hairpin-like conformation of SR-BI external domain forms a hydrophobic channel that may facilitate a bidirectional flux of lipophilic substances, and similar to the simple diffusion mechanism, the net flux via SR-BI will depend on the direction of the concentration gradient [73, 78] (Fig. 2). Thus, an efficient solubilization of carotenoids into mixed micelles and their release to the aqueous phase would favor carotenoid uptake by both simple diffusion and SR-BI mediated transport. The plasma response after the ingestion of nutritional doses of carotenoids as well as the carotenoid uptake by Caco-2 from physiological concentrations of carotenoids (up to ca. 2 μM) respond linearly to the carotenoid dose [67, 68, 79]. Therefore, with carotenoid intakes at nutritional levels the luminal carotenoid concentration falls in the linear response range, well below the onset of carotenoid transport saturation which occurs at 15–20 μM [67, 68]. Such high concentrations of carotenoids in the lumen can only be attained at pharmacological doses of carotenoids (ca. 100 mg β-carotene per day) [80]. At apical concentrations of carotenoids above the level that would be reached by a nutritional dose, the uptake of carotenoids by Caco-2 cells does not respond linearly to the initial concentration at the apical side [67]. However, carotenoid secretion to the basolateral side correlates linearly to the amount absorbed by the cell monolayer regardless of the initial carotenoid concentration at the apical side [67], indicating that the regulation of carotenoid absorption occurs at the apical side, where the SR-BI is expressed.

The SR-BI seems to have low substrate specificity, mediating the transport of many lipophilic substances such as cholesteryl esters, phospholipids, triglycerides, and plant sterols and stanols [81, 82]. However, rate constant analyses of the lipid transfer from HDL particles to SR-BI-expressing cells have indicated that the more lipophilic substances (e.g., cholesterol, cholesteryl esters, plant sterols, and triglycerides) may be transported more readily than the phospholipid molecules which have a bulky polar head [81]. As already demonstrated for β-carotene, lutein, and vitamin E, other lipophilic substances may also be transported by SR-BI. This shared mechanism of transport for carotenoids and vitamin E is consistent with the inhibitory effects of pharmacological levels of vitamin E on canthaxanthin absorption [83]. However, the unraveling of the whole mechanism and the transport efficiency of different carotenoid species awaits further investigations. Furthermore, other transporters may also be involved in carotenoid absorption, as the treatment of the apical membrane of Caco-2 cells with antibodies raised against SR-BI do not inhibit carotenoid uptake as much as the treatment with proteases or substances blocking lipid transport (e.g., Ezetimibe, BLT1) [71, 84]. A recent publication has suggested the involvement of the ABCG5 transporter (known to mediate, together with ABCG8, the efflux of cholesterol and plant sterols from the intestinal epithelial cells) gene polymorphisms in the individual responses to some carotenoids from eggs [85]. The unraveling of the network of transporters that may be involved in carotenoid uptake/efflux and secretion may provide the answers to the old question on the large interindividual variations in carotenoid response.

6 Conclusion

The assessment of carotenoid bioavailability has long been hampered by the limited knowledge on their absorption mechanisms as well as by the limitations presented by the experimental approaches involving laboratory animals and humans. Recently, with the use of in vitro cell culture systems and molecular biology techniques, the mechanisms of carotenoid absorption at cellular level have started to be unveiled. Such systems have also enabled the investigation of factors involved in each step of carotenoid absorption, identifying which steps are influenced by each factor. Detailed in vitro approaches modeling the gastrointestinal environment have characterized the emulsification and micellization steps occurring prior to carotenoid uptake by the intestinal cells. These steps may be largely affected by the food matrix and other dietary components, being the main determinants of carotenoid bioavailability from foodstuffs.

Although the investigations on the intestinal absorption of carotenoids other than β-carotene are still limited, they would be useful to elucidate the differential absorption of individual carotenoids, in view of the recent indications that carotenoid uptake may involve specific epithelial transporters such as SR-BI. Moreover, some studies have already indicated the possibility of other transporters being involved in carotenoid absorption. The challenge of future studies will be the identification of these transporters and the elucidation of the related mechanisms.

7 References
