Carotenoids are a diverse group of colourful pigments naturally found in plants, algae, fungi and bacteria. They play essential roles in development, photosynthesis, root-mycorrhizal interactions and the production of phytohormones, such as abscisic acid and strigolactone. Carotenoid biosynthesis is regulated throughout the life cycle of a plant with dynamic changes in composition matched to prevailing developmental requirements and in response to external environmental stimuli. There are key regulatory nodes in the pathway that control the flux of metabolites into the pathway and alter flux through the pathway. The molecular nature of the mechanisms regulating carotenoid biosynthesis, including evidence for metabolite feedback, transcription and epigenetic control as well as their accumulation, storage and degradation will be the focus of this review.

Carotenoids: everywhere in nature and essential for life
Carotenoids comprise many of the yellow, orange and red pigments of nature, including many fruits, vegetables, flowers, butterflies and crayfish. Animals are unable to synthesize carotenoids; however, they can accumulate carotenoids where they contribute to health and behaviour. For example, fish and birds accumulate dietary carotenoids, which boost their immune system and advertise health, often leading to preferential selection by the sexual partner [1,2]. The human health benefits associated with carotenoids have been extensively reviewed [3-7]. In brief, carotenoids promote antioxidant activity, reduce age-related macular degeneration of the eye and can be precursors for vitamin A. Metabolic engineering of carotenogenesis has proven successful to enhance accumulation [8], develop novel compounds [9] and redirect flux [10-12], and this has been covered in a number of excellent reviews [8,13-19].

In addition to providing colour to flowers and fruits, carotenoids also contribute to the production of scents and flavours that attract insects and animals for pollination and seed dispersal. All photosynthetic organisms accumulate carotenoids where they play crucial roles in photosystem assembly, light-harvesting and photoprotection [20,21]. Typically, leaf tissues accumulate lutein, β-carotene, violaxanthin and neoxanthin (Figure 1), with changes in this profile altering photosynthesis, antenna assembly and photoprotection [21-23]. Carotenoids also serve as precursors for plant hormones, abscisic acid (ABA) and strigolactones (Figure 1) [6,24-27]. The strigolactone class of carotenoid metabolites inhibit shoot branching and stimulate a symbiotic relationship with fungi in the rhizosphere [25,26,28]. Consequently, carotenoid biosynthesis is regulated throughout the life cycle of a plant, with dynamic changes in composition matched to prevailing developmental requirements during germination, photomorphogenesis, photosynthesis, fruit development and in response to external environmental stimuli [29-32]. There are many reports describing altered carotenoid gene transcript abundance during fruit ripening, flower development or stress, which coincide with changes in carotenoid content. This review highlights recent insights into epigenetic, post-transcriptional and metabolite feedback regulation of carotenoid accumulation.

Carotenoid biosynthesis depends upon the availability of isoprenoid substrates
Carotenoids are derived from the plastid-localized 2-C-methyl-D-erythritol 4-phosphate (MEP) pathway (Figure 1) [33] for which glyceraldehyde-3-phosphate and pyruvate act as initial substrates leading to the synthesis of geranylgeranyl diphasate (GGPP) (Figure 1) [34-36]. The condensation of two GGPPs by phytoene synthase (PSY) forms phytoene, the first carotenoid (Figure 1).

The first steps in the MEP pathway are regulated by 1-deoxyxylulose-5-phosphate synthase (DXS) and 1-deoxy-D-xylulose 5-phosphate reductoisomerase (DXR) (Figure 1). Expression analysis of DXS showed organ specific expression and developmental regulation during tomato fruit ripening, which correlated with an increase in PSY mRNA transcripts and carotenoid accumulation [37]. The overexpression of DXS and DXR is sufficient to enhance total carotenoid content by >12% in Arabidopsis (Arabidopsis thaliana) seedlings, while antisense silencing of DXS reduced carotenoid levels by 13% [38,39]. The second key regulatory step is 1-hydroxy-2-methyl-2-(E)-butenyl 4-diphosphate reductase (HDR), which catalyzes the production of IPP (isopentenyl diphasate) and DMAPP (dimethylallyl diphasate) (Figure 1). A correlation between HDR mRNA abundance and carotenoid accumulation has been observed in ripening tomato fruits and greening Arabidopsis seedlings [40]. Furthermore, overexpression of tomato (Solanum lycopersicum) LeHDR in Arabidopsis increased β-carotene and lutein in chloroplasts, but not etioplasts [40].

Abiotic and biotic factors may influence the availability of isoprenoid precursors. Light and circadian oscillations...
Figure 1. Major reactions in the higher plant carotenoid biosynthetic pathway showing enzymes, carotenoids and their precursors (pipes), carotenoid sinks (barrels), carotenoid-derived signalling hormones (green signs) and other MEP isoprenoid-derived metabolites (blue sign). The windows displayed within the chrome pipes indicate abundant carotenoid pigments found in photosynthetic tissues and also represent key nodes for regulation in the pathway. Carotenoid biosynthesis is modulated by environmental factors (light), chromatin modification and metabolic feedback regulation. The side funnels represent examples of metabolic feedback control mechanisms acting upon biosynthetic gene expression as a result of altered PSY and CRTISO enzymatic activity, respectively. First, the bottleneck in phytoene biosynthesis is regulated by PSY and its overexpression increased DXS and DXR mRNA levels post-transcriptionally in etiolated tissues. Second, loss-of-function CRTISO mutants show reduced eLCY transcript levels in etiolated tissues. Abbreviations: βLCY, β-cyclase; βOHase, β-hydroxylase; CCD, carotenoid cleavage dioxygenase; CRTISO, carotenoid isomerase; DXR, 1-deoxy-D-xylulose 5-phosphate reductoisomerase; DXS, 1-deoxyxylulose-5-phosphate synthase; LCY, α-cyclase; αOHase, α-hydroxylase; GGPP, geranylgeranyl diphosphate; HDR, 1-hydroxy-2-methyl-2-(E)-butenyl 4-diphosphate reductase; NCED, 9-cis-epoxycarotenoid dioxygenase; NXS, neoxanthin synthase; PDS, phytoene desaturase; PSY, phytoene synthase; SDG8, histone methyltransferase; VDE, violaxanthin de-epoxidase; ZDS, ω-carotene desaturase; and ZE, zeaxanthin epoxidase.
can alter the expression of nearly all MEP genes and several carotenoid genes. The colonization of plant root arbuscular mycorrhizal fungi activates the MEP pathway by increasing transcript levels of MEP, carotenoid biosynthetic and cleavage genes [41]. This stimulates production of carotenoids and carotenoid cleavage products, such as C_{13} cyclohexenone derivatives (e.g. blumenol), C_{14} apocarotenoids (e.g. mycorradicin) and strigolactones in root plastids. These compounds trigger hyphal branching and a symbiotic association between the fungi and roots [41,42]. Post-transcriptional regulation of DXS and HDR at the level of protein accumulation has also been demonstrated and this has the potential to regulate carotenoid accumulation (see below).

**Phytoene biosynthesis is a rate-limiting step in carotenogenesis**

PSY is generally accepted as being the most important regulatory enzyme in the pathway. Transcriptionally, PSY genes respond to ABA, high light, salt, drought, temperature, photoperiod, development cues and post-transcriptional feedback regulation. While there is only one PSY gene in Arabidopsis, there are two or more homologues in tomato, rice (Oryza sativa), poplar (Populus trichocarpa), bread wheat (Triticum aestivum) and maize (Zea mays) [29,43–46]. The activity of the multiple PSY enzymes appear redundant, but their expression is tissue-specific and shows unique responses to environmental stimuli in cereal roots [29,43,46]. Indeed, salt and drought induced PSY3 transcript abundance and this correlated with increased carotenoid flux and ABA in maize roots [46]. A rapid disappearance of PSY2 and PSY3 mRNA after re-watering suggests tightly controlled mRNA stability or transcription [46]. A similar finding for OsPSY3 in rice roots subjected to salt stress was found [43]. However, stress-induced changes in PSY mRNA do not always result in changes in carotenoid flux. For example, elevated temperatures decreased PSY1 mRNA abundance in maize leaves and unexpectedly there was a concurrent increase in carotenoids [29]. The different expression profiles of the rice PSYs correlate strongly with the presence of promoter regulatory cis-elements that mediate light (PSY1 and PSY2) and ABA responses (PSY3) [43].

Allelic variation in PSY is a mechanism that may change PSY enzymatic activity as alternative splicing of PSY-A1 allele appeared to be a major QTL determinant of flour colour in bread wheat [45]. The alternative splicing results in the generation of four different transcripts, of which one is functional, thereby titrating the level of functional PSY [45].

PSY transcript abundance is upregulated during photomorphogenesis via a phytochrome-mediated (red-light) pathway [30,43]. Both red and far-red light treatments increase PSY mRNA and this is abolished in the phyA mutant [30]. Interestingly, photoinduction of ZmPSY2 transcription in maize leaf tissues can also be mediated by blue photoreceptors (phototropins and cryptochromes) in addition to phytochrome [29]. There is strong evidence to show diurnal oscillations of PSY mRNA and MEP genes, which is consistent with their phytochrome-mediated activities [34,47–50].

A promoter study identified a cis-acting motif (ATCTA), which was present in the promoter region of other photosynthesis-related genes and is believed to be important in mediating the transcriptional regulation of PSY [51]. However, modulating mRNA levels of RAP2.2, an APETALA2 transcription factor that binds to the PSY promoter, resulted in only small pigment alterations in root calli revealing it is just one constituent of a significantly more complex regulatory network involved in carotenogenesis [52]. Finally, there are post-translational effects of photomorphogenesis as it results in activation and relocation of PSY from the prolamellar body to the newly developing thylakoid membranes [30].

There is substantial evidence to support metabolite feedback regulation that modulates the supply of isoprenoid substrates and the accumulation of carotenoids and ABA. This includes feedback within the carotenoid pathway and between carotenoids, MEP and ABA [53–57]. A positive feedback regulation mediated by ABA affects PSY3 gene expression in rice and may play a specialized role in abiotic stress-induced ABA formation [43]. Elevated expression of PSY by a transgene resulted in increased carotenoid levels in etiolated Arabidopsis seedlings and this was via a concomitant post-transcriptional accumulation of DXS mRNA which reveals a feedback mechanism initiated by PSY that stimulated the supply of MEP substrates (Figure 1) [32,58]. However, the overexpression of just DXS in dark-grown seedlings does not increase carotenoid accumulation [32]. Therefore, regulation of the first committed step in carotenogenesis by PSY is tightly coordinated and controlled by source and sink metabolites (Figure 1).

**Regulation of lycopene biosynthesis by desaturases, isomerases and chromatin modifiers**

The production of all trans-lycopene from phytoene requires a complex set of four reactions requiring phytoene desaturase (PDS), \( \zeta \)-carotene isomerase (Z-ISO), \( \zeta \)-carotene desaturase (ZDS) and carotenoid isomerase (CRTISO), as well as a light-mediated photoisomerization (Figure 1) [57,59–65]. PDS may play a rate-limiting role in the generation of 9,15,9\'-tri-cis-\( \zeta \)-carotene as transcript abundance is slightly upregulated during photomorphogenesis via a phytochrome-mediated pathway [30]. The Arabidopsis variegated mutant, IMMUTANS contains lesions in a plastid-targeted alternative oxidase (PTOX) required for phytoene desaturase (PDS) activity, thereby links desaturation to chloroplast electron transport [66]. The accumulation of phytoene has been postulated to involve negative feedback regulation [56]. For example, in the pds3 mutant, genes encoding downstream enzymes such as ZDS and lycopene cyclase (LCY), were downregulated, as were upstream genes such as IP1, GGPS and PSY [56]. Alternatively, the absence of downstream carotenoids in pds3 mutants could modulate the signal. Evidence for this is an analysis of a tomato PDS promoter–GUS fusion that demonstrated end-product regulation in photosynthetic tissues [67]. The regulatory roles for ZDS and Z-ISO in the catalysis of \( \zeta \)-carotene, the product of PDS, to tetra-cis-lycopene, the substrate for CRTISO, as well as control
by photoisomerization under day- length- limiting conditions, have not yet been described.

CRTISO, which catalyses cis-trans reactions to isomer- ase the four cis-bonds introduced by the desaturases, has emerged as a regulatory node in the pathway [61,68]. CRTISO mutants, such as ccr2 and tangerine, result in accumulation of cis-carotenones, such as 7,7,9,9-tetra-cis-lycopenone, in the etioplasts (dark-grown plastids) of seedlings and chromoplasts of fruit [63,64]. Despite this block in etioplasts and chromoplasts, the biosynthetic pathway proceeds in chloroplasts via photoisomerization, but there is delayed greening and substantial reduction in lutein in Arabidopsis and varying degrees of chlorosis in tomato and rice [63,64,69].

Interestingly, a chromatin-modifying histone methyl- transferase enzyme (SET DOMAIN GROUP 8, SDG8) was shown to be required for CRTISO expression (Box 1) [68]. The absence of SDG8 alters the methylation of chromatin associated with the CRTISO gene, thereby reducing gene expression, impairing lutein biosynthesis and increasing shoot branching, in part by possibly limiting strigolactone biosynthesis [68]. This was the first report implicating epigenetic regulatory mechanisms in the control of carotenoid composition [68]. SDG8 is required to maintain expression of CRTISO in seedlings, leaves, shoot apices, anthers and pollen [70]. The CRTISO and SDG8 promoters show overlapping tissue-specific expression patterns in many tissues essential for defining plant architecture and development, including germinating seedlings, meristems, shoot apices, floral anthers and pollen (Box 1) [70].

Regulation of lutein and other xanthophylls

Carotenoid biosynthesis bifurcates after lycopene to produce epsilon- and beta-carotenoids by enzymatic activity of the two lycopene cyclases, εLCY and βLCY, and this branch point has a major regulatory role in modulating the ratio of the most abundant carotenoid, lutein, to the beta-carotenoids [55,68] (Figure 1). Investigations into lutein biosynthesis have yielded lut1, ε-hydroxylase [71]; lut2, εLCY [72,73]; ccr2, CRTISO [63]; and lut5, an additional β-hydroxylase [74] as well as the SDG8 chromatin regulatory mutant, ccr1 [68].

Intriguingly, lutein levels can be altered by producing lycopene via an alternate pathway that does not require the formation of cis-carotenones [75]. Furthermore, the absence of CRTISO or specific carotene isomers results in less lutein [63,64]. The question is whether this reflects altered lycopene substrate preference by the cyclases or metabolite feedback regulation. Flux through the two branches can be controlled at the level of εLCY mRNA [55,73,76] and recent experiments indicate that both CRTISO (ccr2) and SDG8 (ccr1) mutants have some effect on ε-cyclase (εLCY) transcript levels (Figure 1), suggesting feedback may account for at least part of the reduction in lutein (Figure 1) [55,68].

Natural genetic variation in maize was found to be regulated by εLCY, for which four natural dcy polymorphisms explained 58% of the variation in lutein and beta-carotenoids [77] while cosuppression of εLCY in Arabidopsis also altered the ratio of lutein to beta-carotenoid [76].

In Brassica napus the downregulation of lycopene εLCY by RNAi in seeds showed a higher total carotenoid content, specifically increased levels of β-carotene, zeaxanthin, violaxanthin and, unexpectedly, lutein [78]. This unexpected increase in lutein was inconsistent with another report that showed tuber specific silencing of βLCY increased β-carotene levels in potato (Solanum tuberosum) [79]. Clearly, the evidence supports a hypothesis that lutein composition is largely rate-determined by εLCY expression, but feedback regulation can reveal complex regulatory mechanisms, such as that in B. napus.

A molecular synergism between εLCY and βLCY activi- ties is an overall major determinant of flux through the branch leading to production of lutein, β-carotene and the xanthophyll cycle (XC) carotenoids [53,78]. The βLCY gene from the eubacterium Erwinia herbicola and daffodil (Nar- cissus pseudonarcissus) flowers were introduced into the tomato plastid genome and lycopene was channeled into the beta-branch, resulting in increased accumulation of XC carotenoids in leaves and predominantly β-carotene in fruits [12]. Unexpectedly, transplastomic tomatoes again showed a >50% increase in total carotenoid accumulation [12]. Conversely, in the absence of βLCY, εLCY produces a number of unusual carotenes, including δ-carotene, ε-carotene and lactucaxanthin (ε,ε-carotene-3,3′-diol), in endosperm tissue. Several genes encoding enzymes in isoprenoid (DXR and DXS) and carotenoid biosynthesis (β-OHase and ZE) appear to be the subject of negative transcriptional regulation, mediated by a carotenoid or a molecule derived from a carotenoid [53] and these epsilon carotenoids are candidates.

With respect to accumulation of the β-xanthophylls, light stress results in synthesis of zeaxanthin from β- carotene [80] and it is worth noting that beta-hydroxylase (β-OHase) and violaxanthin de-epoxidase (VDE) mRNA are high-light inducible and repressible, respectively [81]. Post-translation modulation of VDE activity by the luminal pH and ascorbate content are also critical for determining the levels of zeaxanthin during high light [82,83].

Carotenoid degradation and turnover

A long-standing question for carotenoid accumulation in photosynthetic tissues has been the rate of synthesis and presumed slow rate of turnover implied by the persistent yellow of senescing leaves. However, recent data using 14CO2 uptake demonstrates that synthesis, and by inference turnover, is much greater than expected [84]. Furthermore, the incorporation of 14C in different carotenoids was not uniform and varied in different mutants and under high light [84]. Given the continued synthesis in mature leaves is much greater than expected, then there must be active degradation. A mechanism for enzymatic turnover in addition to that due to oxidative damage has now been provided.

Studies in Arabidopsis seeds, strawberry (Fragar- ia × ananassa), grape (Vitis vinifera L.) and citrus fruits (Citrus unshiu, Citrus sinensis and Citrus limon) as well as chrysanthemum (Chrysanthemum morifolium) petals, have all demonstrated that the pool of carotenoids is determined in part by the rate of degradation by carotenoid cleavage dioxygenases, which appear to have different
Box 1. Epigenetic implications for carotenoid regulation during root, shoot and flower development

SDG8 is a histone lysine methyltransferase (HKMT) that was discovered as a lutein regulatory mutant, ccr1 (carotenoid and chloroplast regulation). SDG8 trimethylates lysine 4 and/or lysine 36 on the tails of histone proteins contained within the nucleosome, and in so doing promotes an open chromatin configuration and permissive gene expression. ccr1 displayed pleiotropic phenotypes such as reduced root branching (Figure 1a), increased shoot branching (Figure 1b), early flowering and partial male sterility (Figure 1c). SDG8 targets CRTISO and both proteins are encoded by genes that are expressed in similar tissues, specifically in the apical meristem (Figure 1c) and anthers (Figure 1d). These tissues are sites of rapid cell division as well as differentiation and this raises important questions.

Q1. What are the molecular mechanisms by which SDG8 recognizes and targets CRTISO for permissive gene expression? To date, both CRTISO promoter and gene sequences are necessary for recruiting SDG8.

Q2. Are there novel roles for SDG8 and CRTISO in fine-tuning plant development and architecture? One of these roles might be to provide precursors for phytohormone biosynthesis, such as ABA, strigolactones and BYPASS.

Q3. Are carotenoids regulated in response to an epigenetic event? While there is no conclusive evidence to date, there are implications that SDG8 is involved in the epigenetic regulation of flowering time (vernalization) and therefore it is plausible that SDG8 and CRTISO fine-tune carotenoid composition during plant development.

Figure 1. (a, b) SDG8 regulates root and shoot development. (c, d) SDG8 affects flowering time and anther development.

Source verses sink regulation

Organelle biogenesis is one determinant of the storage compartment size of plastids and can affect carotenoid accumulation by providing a larger sink. The hp-2 tomato mutant (caused by lesions in the gene encoding DEETIOLATED1, a negative regulator of light signalling) contains higher fruit pigmentation and a larger plastid compartment size [90]. Similarly, analysis of the high-pigment 3 (hp-3) tomato mutant (lesion in the gene coding for zeaxanthin epoxidase, ZE, which converts zeaxanthin to violaxanthin) revealed an ABA deficiency that led to enlargement of the plastid compartment size, probably by increasing plastid division, and in so doing enabled greater pigment biosynthesis and 30% more storage capacity for carotenoids in mature fruit [91].

The targeting, storage and sequestration of carotenoids within the various plastid types is another important regulator mechanism providing a sink for carotenoid accumulation (see Figure 2) [55,92,93]. The precise localization of the biosynthesis of carotenoids within chloroplasts appears to be largely localized to the envelope and in some cases the thylakoid membrane [94]. The chloroplast signal recognition particle heterodimer (scSRP54; ffc) and (scSRP43; chaos) and its receptor (cpFtsY) are involved in targeting light-harvesting proteins to the thylakoids of chloroplasts. In ffc/chaos and cpFtsY mutants total carotenoid levels are reduced. More specifically, there is a 67% reduction of xanthophylls and a >80% reduction of β-carotene and chlorophyll [93]. It is conceivable that the signal recognition pathway could perform such a role directly, although it is equally likely that chloroplast to nuclear signalling as a result of the reduced LHCs, could cause an indirect effect upon carotenoid biosynthesis [95].

The identification and characterization of a mutation in the Brassica oleracea Orange (Or) gene that creates a metabolic sink to accumulate β-carotene in the chloroplast of cauliflower reveals plastid differentiation is an important mechanism by which to control carotenoid
accumulation, specifically key steps in the MEP pathway, phytoene synthases and the branch point enzymes. Identification of transcription factors that bind PSY is, and will be important breakthroughs, but the lack of change of PSY in root calli expressing a transcription factor [92] indicate complexity and the need for further research to provide a deeper understanding of the most studied enzyme in the pathway. Sequences within the CRTISO gene have been shown to be necessary for SDG8-mediated expression of CRTISO [70], but exactly how SDG8 interacts with and regulates CRTISO by modifying chromatin is of interest. A deeper understanding of the regulation of carotenoid composition by SDG8 may reveal important implications for programming and fine tuning of metabolic flux in response to epigenetic stimuli [68,102].

Carotenoids within the white leucoplasts of roots play essential roles in plant development, including production of strigolactone and ABA. Furthermore, there are indications of yet-to-be defined roles and BYPASS may prove to be one such novel root–shoot β-carotene-derived signal that is required for development (Figure 1) [103]. Also, disrupting carotenoid biosynthesis in ccr1 alters root growth [102] and the co-regulation of CRTISO with developmental regulators, such as flowering locus C (FLC) suggests yet other novel interactions between carotenoids and development. Thus, it is likely more new functions for carotenoids and their derivatives will be found.

Although progress on targeting of carotenoid enzymes within the plastid has been made [94,104], in general we have little understanding of regulation at the level of the protein. How carotenoid enzymes are targeted to the thy-lakoids remains elusive and it may be that the chloroplast signal-recognition proteins are involved in the import of carotenoid enzymes. Consequently, targeting of carotenoid enzymes, such as PSY to the thy-lakoids or regulation by chloroplast–nuclear retrograde signalling may represent novel mechanisms of controlling carotenoid accumulation.

The cross-talk between and within the MEP and carotenogenic pathways is intriguing and the identification of key regulators of the pathways that coordinate carotenoid accumulation and flux is among the foremost challenges to unravel. While the evidence for metabolite feedback as a mechanism to control carotenoid accumulation has been extensively reported, the molecular nature remains unknown. Alternatives for metabolite feedback range from a signalling protein bound to specific carotenoids or their isomers, or we might speculate that soluble or volatile carotenoid cleavage products participate directly in signalling and transcriptional control. For now, the nature of the different forms of metabolic feedback regulation remains the subject of current research and indeed it promises to be one of the most interesting areas of study.

One final consideration is that carotenoid-related research has mostly focused on model species such as Arabidopsis, maize, rice and tomato. Examples of non-model work on regulation include the cauliflower Or gene [92] and other interesting reports have emerged from vegetables and fruits such as orange [105]. In the future more in-depth research on the genotypes enriched with special carotenoids from other crops, especially vegetable and fruit crops, will not only have a larger impact on...
carotenoid genetic improvement in economically important crops, but will also strengthen knowledge of carotenoid regulation [13]. The elucidation of all these elusive regulatory mechanisms continues to excite carotenoid researchers and recent progress indicates fascinating discoveries are waiting to be made.

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