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Integrated cytokin-auxin signaling: A critical loophole in architectural pattern during plant development

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Abstract
Higher plants display a variety of architectures that are defined by the degree of branching, intermodal elongation and shoot determination. Studies on a number of nodal plants including *Arabidopsis thaliana* have greatly strengthened our understanding on the molecular genetic basis of plant architecture; Cytokinins (CK) and auxins are the plant specific chemical messengers (hormones), yet they elicits a diverse array of responses and is involved in the regulation of growth and development throughout the plant life cycle. The recent identification of mutants that are defective in plant architecture and characterization of the corresponding and related genes will eventually enable us to elucidate the molecular mechanisms underlying CK signaling and plant architecture. Forward and reverse genetics strategies have identification important molecular components in auxins-CK perception, signaling and transport. These advances resulted in the identification of some of the underlying regulatory mechanisms as well as the emergence of functional frameworks for auxins-CK action. Furthermore, one prominent mechanism for auxins-CK signal transduction involves degradation of the targeted members of a family of transcriptional regulators participate in complex and competing dimerization networks to modulate the expression of a wide range of genes. These achievements allowed us to improve the plants architecture by molecular design to enhancing crop productivity.

Keywords: MDR, PGP proteins, PIN proteins and nutrient partitioning

Introduction
Phytohormones are important plant growth regulators that control many developmental processes, such as cell division, cell differentiation, organogenesis and morphogenesis. They regulate a multitude of apparently unrelated physiological processes, often with overlapping roles and they mutually modulate their effects. These features imply important synergistic and antagonistic interactions between the various plant hormones (Bielach *et al*., 2012; Mazid, 2014) [18, 105]. Auxin and cytokinins (CK) are central hormones involved in the regulation of plant growth and development, including processes determining root architecture, such as root pole establishment during early embryogenesis, root meristem maintenance and lateral root organogenesis. Thus, to control root development both pathways put special demands on the mechanisms that balance their activities and mediate their interactions. Bielach *et al*., (2012) [18] summarize recent knowledge on the role of auxin and CK in the regulation of root architecture with special focus on lateral root organogenesis, molecular mechanisms of their interactions and proposed forward genetic screen as a tool to identify novel molecular components of the auxin and CK crosstalk. In addition, Zhang *et al*., (2012) [178] reported that CKs, and IAA levels also high, and their contents in attached haustoria increased than in non-attached haustoria. A high auxin-to-CK ratio contributed to haustorial development of *Santalum album*. Endogenous hormones are involved in the haustorial development and in water and nutrient transport in the host-parasite association (Khan *et al*., 2011a; Mazid *et al*., 2011) [63, 90].

CKs and auxins are signaling hormonal molecules that may play an essential role in regulating cytokinesis, growth and development in plants. Today, the major challenge for plant biologists is to understand the mechanisms that control patterned development of capable systems. In addition, Kushwah *et al*., (2011) [76] results show that asymmetrical exposure of CK at the root tip in Arabidopsis (*Arabidopsis thaliana*) promotes cell elongation that is potentiated by glucose in a hexokinase-influenced, G-protein-independent manner (Khan *et al.*, 2014; Quddusi *et al.*, 2014) [63, 127].

The present review article focuses on hormonal molecules that shape growth, differentiation and architecture of the plant systems. The role of the main root signal (CK) is analysed, together with the role of the shoot signal, auxins (particularly Indole acetic acid (IAA)) and their interactions (Coenen and Lomex, 1997; Woodward and Bartel, 2005) [30, 173]. Auxin, predominantly represented by IAA is involved in the regulation of plant growth and development. Although IAA was the first plant hormone identified, the biosynthetic pathway at the genetic level has remained unclear. Two major pathways for IAA biosynthesis have been proposed: the tryptophan (Trp)-independent and Trp-dependent pathways. In

Trp-dependent IAA biosynthesis, four pathways have been postulated in plants: (i) the indole-3-acetamide (IAM) pathway; (ii) the indole-3-pyruvic acid (IPA) pathway; (iii) the tryptamine (TAM) pathway; and (iv) the indole-3-acetaldoxime (IAOX) pathway. Although different plant species may have unique strategies and modifications to optimize their metabolic pathways, plants would be expected to share evolutionarily conserved core mechanisms for auxin biosynthesis because IAA is a fundamental substance in the plant life cycle. The genes known to be involved in auxin biosynthesis are summarized and the major IAA biosynthetic pathway distributed widely in the plant kingdom is discussed on the basis of biochemical and molecular biological findings and bioinformatics studies. Based on evolutionarily conserved core mechanisms, it is thought that the pathway via IAM or IPA is the major routes to IAA in plants (Mano *et al*., 2012; Khan *et al.*, 2011b & c; Mazid *et al.*, 2011) [85, 59, 94].

In an intact plant, the living cells in both the root and the shoot are capable of producing CKs (Aloni *et al*., 2005; Khan *et al.*, 2013; Naz *et al.*, 2018) [62]. Although, both CK and IAA can be produced in roots and shoots (Ljung *et al*., 2005; Khan and Mazid., 2018) [62, 93], but however, the young shoot organs are the major sites of IAA production (Aloni *et al*., 2006) [7] while root tips are the major sites of CK productions. Auxin has a fundamental role throughout the life cycle of land plants. Lavy *et al*., (2012) [78] sequenced candidate genes in auxin-insensitive mutants of *Physcomitrella patens* and identified mutations in highly conserved regions of the moss ortholog of tomato DGT and did not observe a clear effect of the Ppdgt mutation on the degradation of Aux/IAA proteins. However, the induction of several auxin-regulated genes was reduced. Genetic analysis revealed that dgt can suppress the phenotype conferred by overexpression of an AFB auxin receptor. Lavy *et al*., (2012) [78] results indicate that the DGT protein affects auxin-induced transcription and has a conserved function in auxin regulation in land plants (Mazid *et al.*, 2014a; Mazid and Roychowdhury, 2014) [105, 106].

The productions of both CK and IAA does not occur randomly but is regulated by the locations of the synthesizing cells in the plant body and their developmental stages and in influenced by environmental conditions that although their signal move in specific structural pathways and by different mechanisms (Aloni *et al*., 2005; Mazid, 2014b) [114] to regulate plant development and differentiation. CK and IAA are key hormones that regulate the root development; its vascular differentiation and root gravitropism and along with enzyme ethylene, they maintain aregulate lateral root initiation (Aloni *et al*., 2006). Atta *et al*., (2012) [9] report a novel technique for controlled release of PGRs by sunlight using photoremovable protecting group (PRPG) as a delivery device. Carboxyl-containing PGRs of the auxin chemically caged using PRPGs of coumarin derivatives which exhibited good fluorescence properties. Bioactivity experiments indicated that caged PGRs showed better enhancement in the root and shoot length growth of *Cicer arietinum* compared to PGRs after sunlight exposure. The use of PRPG as a delivery device for controlled release of PGRs by sunlight in soil holds great interest for field application since it can overcome the rapid loss of PGRs in environmental conditions (Mazid and Naqvi, 2014 a) [104].

To understand the control of plant architecture and the sites of lateral shoot and root initiations, there are a need to understand the hormonal mechanisms that suggests the features of the radical pattern of the primary vascular systems in the shoot and roots. Most of the studies on vascular differentiation and vascular patterning were made a shoot of soil-grown plants (Aloni, 2001; Turner and Seiburth, 2002; Naqvi *et al.*, 2014) [116, 115]. The embryogenesis of higher plants establishes the plant body plan including the SAM shoot apical meristem and root apical meristem (RAM), which determine the architecture fit he aerial and underground parts of a plant, respectively. The SAM generates all above-ground components of a plant, including leaves, stems and auxiliary branches, under the dynamic balance of indeterminate growth and differentiation. Plant vascular tissues, xylem and phloem, evolved as early as the Silurian period some 430 million years ago. Evolution of vascular tissues solved the problems of long-distance transport of water and foods, thus enabling early vascular plants to gradually colonise in land (Raven *et al*., 1999; Mazid and Naqvi, 2014 b) [130, 114]. In primitive vascular plants, vascular tissues are organized in a simple pattern in which xylem located at the later and phloem surroundings xylem. With the evolution of diverse vascular plants, vascular tissue also evolved to have a variety of organization. The main site of CK synthesis in the root tip, specifically the root cap cells, was confirmed by Aloni *et al*., (2005) [61], showing that in seedlings as well as in plants grown under conditions of almost no transcription, the highest concentration of true occurs in the root cap statocysts. From the root cap, the CK is transported upward through plasmodesmata, which provide symplastic continuity in the meristematic and elongation zones and from the differentiation zone through vessels of the xylem by the transportation stream to developing shoot organs and with high transcription rates (Aloni *et al*., 2005; Mazid and Khan, 2015 a; Jafri *et al.*, 2015) [61, 104].

CKs have opposing roles in shoot and roots; in young shoot organs the CK positively regulate the development and promote shoot growth (Rahayu *et al*., 2005) [129], but in root they are negative regulators of growth and development (Werner *et al*., 2003) [170]. The absence of isopentenyl transferase (IPT) expression in SAMs is content with the absence of free CK in SAMs is context with the absence of free CK in SAM of *Arabidopsis thaliana* seed levels (Aloni *et al*., 2004) [114], and its absence in lateral buds of older plants grown under conditions of almost no transcription (Aloni *et al*., 2005) [116] indicates that the SAM are apparently no to the primary structures for CK synthesis. On the hand, other once i.e. auxin is known to be involved in virtually every aspect of plant growth and development. In recently, the exact position of auxins synthesis are identified, but the identification of molecular aspects of its biosynthesis revealed the existence of at least two separate biosynthesis modes (Cheng *et al*., 2006; Mazid and Khan, 2015) [60, 115]. The synthesized auxins are transported to specific tissues where it triggers a signaling cascade that causes developmental responses.

Metabolism and long-distance translocation

During plant development, distantly-located organs must communicate in order to adopt morphological and physiological features in response to environmental and physiological features in response to environmental inputs

(Kudo *et al*., 2010) [73]. CK and IAA have antagonistic roles in root development; auxin promotes the formation of lateral root (Woodward and Bartel, 2005) [173] and adventitious roots, whereas CK reverse the IAA effect. Auxin is mainly transported along the shoot-root axis from cell to cell in a polar manners, namely polar auxin transport (PAT) which requires both influx and efflux carriers. Similarly, Zheng *et al*., (2011) [179] identify the AUXIN UP-REGULATED F-BOX PROTEIN1 (AUF1) and its potential paralog AUF2 as important positive modifiers of root elongation that tether auxin movements to CK signaling in Arabidopsis (*Arabidopsis thaliana*). *auf1* roots are also hypersensitive to CK and have increased expression of several components of CK signaling. Kinematic analyses of root growth and localization of the cyclin B mitotic marker showed that AUF1 does not affect root cell division but promotes CK-mediated cell expansion in the elongation/differentiation zone (Fig. 1).

Auxin is a mobile signal which affects nuclear transcription by regulating the stability of AUX/IAA repressor proteins. Auxin is polarly transported from cell to cell by auxin efflux proteins of the PIN family, but it is not as yet clear how auxin levels are regulated within cells and how auxin access to the nucleus may be controlled. The Arabidopsis genome contains eight PINs, encoding proteins with a similar membrane topology. While five of the PINs are typically targeted polarly to the plasma membranes, the smallest members of the family, PIN5 and PIN8, seem to be located not at the plasma membrane, but in endomembranes. Bosco *et al*., (2012) [23] demonstrate by electron-microscopy analysis that PIN8, which is specifically expressed in pollen, resides in the endoplasmic reticulum and that it remains internally localized during pollen tube growth show a functional role for endoplasmic reticulum-localized PIN8 and suggests a mechanism whereby PIN8 controls auxin thresholds and auxin access to the nucleus, thereby regulating auxin-dependent transcriptional activity. Transgenic Arabidopsis and tobacco plants generated over expressing or ectopically expressing functional PIN8 and its role in control of auxin homeostasis studied. PIN8 ectopic expression resulted in strong auxin related phenotypes. The severity of phenotypes depended on PIN8 protein levels, suggesting a rate-limiting activity for PIN8. The observed phenotypes correlated with elevated levels of free IAA and ester-conjugated IAA. Activation of the auxin-regulated synthetic DR5 promoter and of auxin response genes strongly repressed in seedlings over-expressing PIN8 when exposed to 1-NAA. Beside this, polar transport of auxin is controlled by PIN- and ABCB/PGP-efflux catalysts. PIN polarity is regulated by the AGC protein kinase, PINOID (PID), while ABCB activity shown to be dependent on interaction with the FKBP42, Twisted Dwarf1 (TWD1) and provide evidence that PID phosphorylation has a dual, counter-active impact on ABCB1 activity that is coordinated by TWD1-PID interaction. Using co-immunoprecipitation (co-IP) and shotgun LC-MS/MS analysis, Henrichs *et al*., (2012) [49] identified PID as a valid partner in the interaction with TWD1. *In-vitro* and yeast expression analyses indicated that PID specifically modulates ABCB1-mediated auxin efflux in an action that is dependent on its kinase activity and that is reverted by quercetin binding and thus inhibition of PID autophosphorylation. Triple ABCB1/PID/TWD1 co-transfection in tobacco revealed that PID enhances ABCB1-mediated auxin efflux but blocks ABCB1 in the presence of TWD1.

To date, a variety of natural CK species, including tZ (ribozeatin), cis-zeatin (cZ) and their conjugates have been identified; the active CK species are the free base type (Mok and Mok, 2001) [112]. CK activity in an organ is regulated at diverse steps, including de novo synthesis, activation, conjugation and degradation. Local and long-distance transport system is involved in regulation of CK action. In addition, spatial distribution of CK signaling systems (i.e., receptors and response regulators) specifies the domain in which a CK response can occur. Today, our understanding of CK biosynthesis has greatly progressed due in large part to the identification of key pathway genes endocding adensine pyrophosphate-IPT (Sakamoto *et al*. 2006; Mazid and Jafri, 2015) [102], t-RNA-IPT (Sakamoto *et al*. 2006), CK-transhydroxylase, and the CK nucleoside 5^1-monophosphate phosphoribo hydrolase, LONELY GUY (LOG) (Kurakawa *et al*., 2007; Mazid and Khan., 2015 b) [59, 96].

Auxin plays a central role in the regulation of plant growth and development, as well as in responses to environmental stimuli. Narciclasine (NCS), an alkaloid, isolated from Narcissus tazetta bulbs has a broad range of inhibitory effects on plants. Hu *et al*., (2012) demonstrated the inhibitory effects of NCS on auxin-inducible lateral root formation, root hair formation, primary root growth, and the expression of primary auxin-inducible genes in Arabidopsis roots using DR5::GUS reporter gene, native auxin promoters (IAA12::GUS, IAA13::GUS), and quantitative reverse transcription PCR analysis. Results also showed that NCS did not affect the expression of CK-inducible ARR5::GUS reporter gene. NCS relieved the auxin-enhanced degradation of the Aux/IAA repressor modulated by the SCF (TIR1) ubiquitin-proteasome pathway. In addition, NCS did not alter the auxin-stimulated interaction between IAA7/AXR2 (Aux/IAA proteins) and the F-box protein TIR1 activity of the proteasome. These results suggest that NCS acts on the auxin signaling pathway upstream of TIR1, which modulates Aux/IAA protein degradation, and thereby affects the auxin-mediated responses in Arabidopsis roots.

Traditionally, it was thought that CK were synthesized in the root and transported to the shoot through the xylem (Letham and Palni, 1993; Beveridge *et al*., 1997; Hassanpourdgham *et al.*, 2015). However, in Arabidopsis, the initial step of iP and TZ biosynthesis is catalyzed by IPT using dimethylallyl diphosphate (DMAPP) and adenosine5^1-diphosphate (ADP) or adenosine-5^1-tri phosphate (ATP), to gunrate; P-ribotides. These i-P-ribotides are then hydroxylated to tZ-ribotide by CYP735A1 of cZ is initiated by tRNA-IPT that catalyze the prenylation of t-RNA using DMAPP; however, the enzyme for cis-hydroxylation has yet to be identified in plants. In addition, CK-riboside-5 ^{1}-monophosphate is directly converted to free base-CKs by LOG (Kurakawas *et al*., 2007). Inactivation of CKs in carried out by degradation or conjugation. Degradation is catalysed by CK-oxidase/dehydrogenase. Recent studies have demonstrated that the degradation step plays an important role in regulating CK activity (Ashikari *et al*., 2005) [8]. Recent studies on the LOG family genes in rice and Arabidopsis suggests that activation of CK occurs in nearly all parts of the plant (Kuroha

et al., 2009). However, few studies on the spatial distribution of CK metabolism in roots, but also in various sites within the aerial parts of the plant. Such differential distribution of these CK biosynthesis genes might be important to produce the various CK species in underground and above ground organs.

However, recent findings of Choi *et al*., (2011) [29] have shed light on a distinct role of CKs in plant immune responses. They suggest that plant-borne CKs systemically induce resistance against pathogen infection which is orchestrated by endogenous CK and salicylic acid (SA) signaling. Numerous reports ascribe a stimulatory or inhibitory function to CK in different developmental processes such as root growth and branching, control of apical dominance in the shoot, chloroplast development, and leaf senescence. Conclusions about the biological functions of CK have mainly been derived from studies on the consequences of exogenous CK application or endogenously enhanced CK levels, up to now, it has not been possible to address the reverse question: what are the consequences for plant growth and development if the endogenous CK concentration is decreased.

The rate of de novo synthesis metabolic inter-conversion and breakdown are together with transport processes relevant to the regulation of CK homeostasis in cells. CK metabolism includes mainly conversions among CK bases, a side chain modification and conjugate-hydrolysing reactions and CK-degradation. CKs are irreversively degraded in a single enzymatic step by oxidative cleavage of the N^6-side chain. The reaction is catalyzed by the CK oxidase/dehydrogenase (CKX), which contain FAD as a cofactor. The reaction products are adenine and an aldehyde. The preferred substrate of CKX is isopentenyladenine, zeatin and their corresponding ribosides. Ribotides, O-glucosides, dehydro-zeatine and aromatic CKs are not degraded by CKX. The Arabidopsis genome contains several At CKX genes which are preferentially expressed in zones of active cell division and growth. The corresponding enzymes are located in the ER, in the apoplast and in the vacuoles. Root cap predominantly produces CK by expressing the IPT genes (Takei *et al*., 2004) and that the highest concentration of free CK in a roots is found in the root tip (Aloni *et al*., 2005; Mazid and Naz, 2017a) [94] justify a more precise term for the this instance of apical dominance, namely CK-dependent root apical dominance.

CK regulates root architecture by balancing the promoting role of IAA in lateral root development. CK produced in the active cap of a primarily root as the hormonal signal which enables maximum development of an actively growing primary root by retarding lateral root initiation. Tree bioactive CK can be visualized by the expression of ARR5:GUS (a CK-activated promoter sequence of a response regulator fused to β-glucuronidase), which reflects the transcriptional activation of a CK-sensitive promoter fused to the GUS receptor gene. It is likely that higher plants have import and export systems to mobilize the CK across the plasma-membrane (Hirose *et al*., 2008; Mazid and Khan, 2017a) [65]. Till date, the Purina permease (PUP) family and the equlibrative nucleoside transporter (ENT) family have been proposed as candidates for CK transporters. Although results suggest that plant ENT proteins are involved in the transport of CK ribosides, but however, definitive evidence has not been provided. Although, further genetic studies on PUPs, ENTs, and other transporters are needed to fully understood such putative CK transport systems establishing concentration gradients with in specific tissues/organs. Among this family members, the ability of at PUP1 and ZAt PUP2 to transport tZ and iP was shown using a yeast system; however, genetic studies on plant PUPs using loss-of function or gam-of-function mutants have not been reported.

In higher plants, long-distance translocation of CK is mediated by the xylem, an acropetal transport system that occurs by transpiration flow, and the phloem translocation systems that delivers photosynthates throughout the body of the plant. This hypothesis is supported by an experiment (grafting experiment) using an atiPt1;3;5;7 mutant, in which the content of both iP type and tZ type CK decreased in comparison with wild type plants (Miyawaki *et al*., 2006) [109]. It is conceivable that plants might use tZR as an acropetal messenger and iP-type CKs as systemic or basipetal messengers. In the xylem sap the major form of CK is TZR and in phloem sap, the major forms are iP-type CKs, such as iPR and iP-ribotides. Systemic translocation of CKs was implied by early tracer experiments. Although radioactive CKs applied tolerance is strongly retained at the treated site, a small proportion of the labelled CKs are translocated to other plant parts (Letham, 1994) [81].

Wild type root-stocks recovered the tZ-type CKs in the mutant shoot-scions but not the iP-type CKs (Matsumoto-Kitano *et al*., 2008; Mazid and Naz, 2017b) [86]. Wild type shoot-scions recovered the iP-type CKs in the mutant root-stocks to normal levels, whereas the tZ-type CKs were only partially recovered (Matsumoto-Kitano *et al*., 2008) [86]. Reciprocal grafting experiments also restored visible mutant phenotypes, such as defects in the thickening growth of roots and inflorescence stems (Matsumoto-Kitano *et al*., 2008) [86]. Over the past decade, identification and characterization of CK related genes has greatly advanced our understanding of CK metabolism and transport; however, to fully elucidate the metabolism and transport for CKs, a large array of tissues need to be resolved. Auxin biosynthesis in plants is extremely complex. Multiple pathways likely contribute to de novo auxin production. IAA can also be reduced from IAA conjugates by hydrolytic cleavage of IAA-amino acids, IAA-sugar, and IAA-methyl ester (Woodward and Bartel, 2005; Yang *et al*., 2008) [173].

Cerny *et al*., (2010) [26] indicate novel links between temperature and CK signaling, and an involvement of calcium ions in CK signaling. Most of the differentially regulated proteins and phosphoproteins are located in chloroplasts, suggesting an as yet uncharacterized direct signaling chain responsible for CK action in chloroplasts. Finally, first insights into the degree of specificity of CK receptors on phosphor proteomic effects are obtained from analyses of CK action in a set of CK receptor double mutants. In contrast to the great progress made in understanding about signaling and transport, much less is known about how auxins is produced in plants (Mockaitis and Estelle, 2008; Vanneste and Friml, 2009). [111, 168] The exact mechanisms and cellular locations of auxins biosynthesis remains relatively unknown, but the identification of molecular components of auxin biosynthesis revealed the existence of at least two separate biosynthesis

pathways (Barlier *et al*., 2000; Cheng *et al*., 2006) [12, 27]. The synthesized auxin is transported to specific tissues where it triggers a signaling cascade that causes developmental responses. The transport of auxin is unique because it displays directionality, which is provided through the specific sub-cellular localization of auxin efflux and auxin influx machineries (Benjamins *et al*., 2005) [14]. The sub cellular targeting of the constituent protein is regulated by components involved in endosomal trafficking, and also depends on the phosphorylation status of the proteins, as well as components that determine membrane composition (Friml *et al*., 2004; Mazid *et al.,* 2017) [89]. Auxin is perceived by auxin receptors, represented by members of the Transport Inhibitor Response 1 (Tir 1) Family, Which Results In The Proteolysis Of Auxin/Indole-3-Acetic Acid (Auxiiaa) Proteins, thereby releasing their inhibitory effect on AAUXIN RESPONSE FACTORS (ARFs) transcription factors that regulate auxin responsive gene expression (Quint and Gray, 2006). Polar transport of auxin mediated by carrier proteins is a unique mechanisms resulting in a controlled distribution of auxin that generates higher auxin concentrations in specific cell/and tissues. The model of Rubery and Sheldrake (1974) [134] states that auxin is an acid, can freely enter the cell but is subsequent trapped inside the apoplast. An important aspect of this model is that the specific membrane localization of the auxin efflux carriers provides directionality to auxin transport. Various molecular and structural components are described below that actively involved in the transport of auxin.

Role of PIN proteins

The PIN protein family consists of eight members, most of which mediate auxin efflux. Two important characteristics provided the first hints of their proposed function in the efflux of auxin. Firstly, PIN expressed in auxin-transporting tissues and cells and secondary, it is asymmetrically localized in the plasma-membranes of these cells (Galweiler *et al*., 1998) [40]. This gene family named after the pin formed 1 mutant, white almost entirely lacks flowers or lateral organs along its inflorescence, a phenotype that can be copied by growing plants on auxin transport inhibitor (Okada *et al*., 1991; Mazid and Khan., 2017 b) [64, 88]. This fully of proteins is centrally localized in stele cells of stem and roots, while are believed to be important conductors of auxin. The first PIN family members to be cloned was Ethylene Insensitive Root1 (Eir1; Later Called As PIN2, AGR1 or WAV6); moreover mutants in EIR1/PIN2 display a gravitropic root growth. PIN2 is localized to the upper side of epidermal and lateral root cap cells and to the lower side of cortex cells. The stability of the PIN2 protein is important for its function during the root gravitropic response (Sieberer *et al*., 2000) [147]. The other PIN proteins (e.g., PIN3, OPIN4, PIN4 and PIN7) have been linked to active auxin transport as well (Friml *et al*., 2002) [168]. PIN3 expressed in the root pericycle, the columella, the hypocotyls endodermis and the apical hook and involved in the control of the tropic growth responses (Friml *et al*., 2002) [168] as well as PIN4 involved in stabilization of a local auxin maximum in the root meristems (Friml *et al*., 2002) [168]. On the other hand, PIN7 is expressed from early embryogenesis onward and appears to be involved in the auxin-mediate control of embryonic axis formation but functional analysis of the PIN5, 7, 6 and 8 genes has not yet been reported (Friml *et al*., 2003). Although, indications towards the exact molecular functions of PIN proteins are provided but however, several aspects remain unclear, such as how do PIN proteins transport auxin?, Yet, hints towards the exact molecular functions of PIN proteins came from experiments in which they respects the rate-limiting factors in the efflux of auxin as well as supreme determinants in providing directionality to polar auxin flow (Petrasek *et al*., 2006; Wisniewska *et al*., 2006) [124]. Resolving crystal structure of PIN proteins would be a critical step in elucidations their exact molecular function (Fig. 1).

Interestingly, the expression of *PLT* genes in auxin inducible and largely overlap with the auxin maximum in the root (Aida *et al*., 2004; Sabatini *et al*., 1999) [136]. *PLT* genes are necessary for the expression of *PIN* genes in the embryonic root pole and root meristem, which in part expression the serve mutant phenotype. In the turn the *PIN* genes are needed to position the auxin maximum in the root meristem, thereby restricting the expression of the *PLT* genes. This suggests that the auxin maximum is not only read out by *PLT* genes, which are they likely activated by ARF-dependent transcription, but also that the *PLT* genes subsequently regulate auxin distribution. The genes intriguing aspects of PIN auxin efflux facilities and the AUX1 influx carriers that fascinated researchers for almost a decade in their polar localization.

MDR and PGP proteins

Another class of auxin efflux, in addition to PIN, facilities is represented by the MULTIDRUG RESISTANCE (MDR); p-glycoprotein (PGP) family containing MDR1, PGP1, PGP2 and PGP4 and PGP19 (Murphy *et al*., 2002) [114]. MDR display auxin-transporting activity when expressed in hetrologous hosts (Blakeslee *et al*., 2005) [19]. PIN and PGP protein co localize and interact with each other (Blakeslee *et al*., 2007) [20]. Among the PIN family, only PIN1 co-immuno precipitates with PGP1 and PGP19 and therefore suggesting specificity of interactions. A triple mutant (pin2pgpppgp19) display synergistic interaction among these genes interaction that PIN and PGP protein function in distinct auxins transport pathway that partially overlap (Blakeslee *et al*., 2007) [20]. The exact functional relationship between PINs and PGPs remain unclear. An interacting hypothesis is that PIN proteins act to guide the action of several MDR transporters, which may provide an alternate explanation for the capacity of PIN protein to transport auxins in heterogonous systems (Petrasek *et al*., 2006).

AUX 1: Auxin resistant 1

In plants, formation of local gradients of auxin is critical for plant developmental processes exhibiting polarity. The auxin efflux carriers, PINs, localize asymmetrically in the plasma membrane and cause the formation of local auxin gradients throughout the plant and a role in phosphorylation is important for polar PIN trafficking. The asymmetry of PIN distribution in the plasma membrane is determined by phosphorylation mediated polar trafficking of PIN proteins (Ganguly *et al*., 2012). Auxin acts as a prominent signal, providing, by its local accumulation or depletion in selected cells, a spatial and temporal reference for changes in the developmental program.

The distribution of auxin depends on both auxin metabolism (biosynthesis, conjugation and degradation) and cellular auxin transport. Barbez *et al*., (2012) [11] identified a novel putative auxin transport facilitator family, called PIN-LIKES (PILS), and illustrate that PILS proteins are required for auxin-dependent regulation of plant growth by determining the cellular sensitivity to auxin. PILS proteins regulate intracellular auxin accumulation at the endoplasmic reticulum and thus auxin availability for nuclear auxin signalling. PILS activity affects the level of endogenous auxin IAA, presumably via intracellular accumulation and metabolism.

AUX1 is the only well-described auxin import (auxin-influx) carrier. Before auxin can be transported out of the cell it first needs to get in, it occurs mainly via diffusion but active transport also takes place carrier mediated auxin import has been proposed to be required for transport against a diffusion gradient or to prevent diffusion into neighbouring cells, and therefore would provide an effective mean to establish and maintain optimal auxin conditions and distributions with in cells and tissues (Reinhardt *et al*., 2003) [131]. Membrane-associated AUX1 expression in root tropic root cap and root epidermis cells further links the gravitropic root phenotype of aux1 of defects in polar auxin transport. Together the available data suggest a function of Aux1 to support polar auxin delivery to the root apex. Aux1 is the capable of transporting auxin even in a heterologous system. As in the case of the PIN proteins, the specific localization of Aux1 and Aux-1 like protein can be anticipated to be very important for its function. A wealth of information is available on functional aspects of AUX1; however, the biochemical characterization of Aux1; as well as the crystal structure, is still lacking. The analysis of the AUX1 familial members might also provide information on their role in auxin transport. An interesting transport is low Aux1 activity is regulated during a root gravitropic response components involved in auxin perception and transport, the existence of two regulatory loops, rather than a strictly hierarchical system, can be involves auxin, *Aux/IAA* and ARF proteins as well as loops represent extensive feedbacks as well as feed forward regulation. Auxin induces expression of *Aux/IAA* proteins which in many cases reduces the sensitivity of cells towards auxin (Kim *et al*., 1997). The auxin-dependent degradation of *Aux/IAA* proteins mediated by SCF^{TIR1} releases ARF/IAA proteins to transcribe auxin-regulated genes, including the *Aux/IAA* genes (Gray *et al*., 2001). Auxin transport components were also found to be under the control of ARF proteins (Okushima *et al*., 2005). This suggest a regulatory mechanisms in which high concentrations are sensed by SCF^{TIR1}, which results in breakdown of *Aux/IAA* and the subsequent release of ARF transcription factors that up regulate the expression of auxin transport components which transport auxin out of the cell. The involvement of auxin transport in Arabidopsis vascular pattering was first studied by pharmacological inhibition of polar auxin transport (Mattsson *et al*., 1999; Sieburth, 1999). PIN1 is the major auxin transport component in vascular development, because it is the only family member expressed during early stages of procambium formation (Scarpella *et al*., 2006) [144].

An important aspect of on auxin is ARFs, which activate or repress the auxin response genes by binding to auxin response elements (AuxREs) on their promoters. Liu *et al*., (2011) focused on molecular biological advances of plant ARF families, and discussed ARF structures, regulation of ARF gene expression, the roles of ARFs in regulating the development of plants and in signal transduction and the mechanisms involved in the target gene regulation by ARFs. The phylogenetic relationships of ARFs in plants are close and most of them have 4 domains. ARFs are expressed in various tissues. Their expressions are regulated at both transcriptional and post-transcriptional levels. They play important roles in the interactions between auxin and other hormones. Auxin plays an essential role in embryonic root initiation, in part through the action of the ARF5/MP transcription factor and its auxin-labile inhibitor IAA12/BDL. MP and BDL function in embryonic cells but promote auxin transport to adjacent extraembryonic suspensor cells, including the quiescent center (QC) precursor (hypophysis).

Direct auxin measurements using sorted cell populations should now validate this theoretical framework. According to this model, popularly localized PIN proteins are the main players to set up the auxin maximum and gradient for auxin distribution in the root. Many more predictions of the model were validated expectedly or explained by previous observations. In accordance with model, settling up the auxin maximum is fast, which is in the line with earlier experimental data in which the auxin maximum is rapidly re-established after laser ablation of the QC (Xu *et al*., 2006). The precise auxin gradient likely depends on auxin influx as well (Swarup *et al*., 2001) [156].

Application experiments using auxin and auxin transport inhibitors provided an excellent correlation between the accumulation of auxin in convergence points and PIN1 expression determining the site of vein formation. Auxin is transported through the epidermis of the new primordial and accumulation at the top of the primordium; this determines the formation of the convergence points for PIN1 expression which leads to the formation of a mid vein from the top towards the base (Scarpella *et al*., 2006) [144]. Another model produced a mathematical transport of auxin varies as a function of the flux of auxin (Mitchison, 1980; 1981). This model suggested that into the young primordial, vascular stand formation is sink driven; the sink is the base of the leaf and the source is at the margin of a leaf. In this model, the localized auxin sources also provide an explanation for the formation of discontinuous veins, something that is observed in vesicles trafficking mutants such as '*gnan*' and '*van3*' (Geldner *et al*., 2001) [42]. The model by Rolland-Lagan and Prusinkiewicz (2005) [135] shows that when multiple auxin sources as well as a high auxin flux from both source and sink exist in the leaf the canalization process can be stopped and discontinuous strands can be formed. This model would allow a cell to share its auxin flux with two neighbouring cells. Discontinuous are then readily explained by setting low values for the flux-bifurcator. PIN expression is crucial to the localization of the auxin maximum, which in turn induces the expression of stem cell-specifying *PLT* gene (Aida *et al*., 2004). The family of *PLT* genes which are also present in a gradient that nicely overlaps with the anticipated auxin gradient are good candidates for performing the readout. Taking into account the interdependence of *PIN* and *PLT* genes. This model implies

the existence of a regulatory loop that setup an auxin gradient, which is self-regulating because PLT transcription factors subsequently regulate the expression of PINs (Aida *et al*., 2004; Grieneisen *et al*., 2007) [46]. The theoretical model predicted an auxin gradient inside the individual cells, instructive for the retention of PIN proteins at the membrane as well as for the or polar localization (Paciorek *et al*., 2005). This intracellular auxin gradient might also regulate cell polarity in general. However, high auxin was reported to retain PIN protein at the membrane, whereas in the model. Low intracellular auxin correlates with the position of PIN protein at the membrane (Grieneisen *et al*., 2007; Paciorek *et al*., 2005) [46]. This raises the concept that auxin has emerged as a star player in almost every aspect of plant development. It is exactly this pervasive role that makes auxin research about its transport so complex.

Auxins and CKs under pathogen infection

Auxin is a major growth hormone in plants, and recent studies have elucidated many of the molecular mechanisms underlying its action, including transport, perception and signal transduction. However, major gaps remain in our knowledge of auxin biosynthetic control, partly due to the complexity and probable redundancy of multiple pathways that involve the YUCCA family of flavin-dependent mono-oxygenases. Differential localization of YUCCA4 alternative splice variants to the endoplasmic reticulum and the cytosol, which depends on tissue-specific splicing. One isoform is restricted to flowers, and is anchored to the cytosolic face of the endoplasmic reticulum membrane via a hydrophobic C-terminal transmembrane domain. The other isoform is present in all tissues and is distributed throughout the cytosol and play a role for intracellular compartmentation in auxin biosynthesis (Kriechbaumer *et al*., 2012) [70].

Auxins and CK are both produced by several plant pathogenic bacteria and play a role in pathogencity. Plant pathogens such as Agrobacteria produce auxin to high CK plant cells for nutrient production. *Agrobacterium tumifaciens*, the causative agent of the crown gall disease and induced CK production by plants. During the infection process, it transfers a small stretch of DNA (T-DNA) to the host plant which has been integrated with nuclear genome of host. The T-DNA harbours an *IPT* gene, which is expressed in the host cell and causes CK over production. In case of auxin production, the other pathogens like *Pseudomonas* and *Agrobacterium* use atryptophan-2-mnoxygenase called iaaM to convert tryptophan to indole-3-acetamide (IAM), which is subsequently hydrolysed into IAA by the hydrolase iaaH (Dharmasiri *et al*., 2005) [33]. The iiaM/iiaH pathway is the only complete try-dependent IAA biosynthesis pathway known to date. It is generally believed that plants do not use the iiaM/iiaH pathway to make IAA. However, IAM exists in plant extract and has been suggested as a key intermediate in converting indole-3-acetal-doxime (IAOx) to IAA (Sugawara *et al*., 2009). However, the biochemical reactions for IAM production in plants have been not solved. The bacterial iiaM gene alone in petunia, tobacco and Arabidopsis lead to auxin overproduction phenotypes, suggesting that plant have enzymes for the hydrolysis of IAM (Sitbon *et al*. 1992) [150]. Under influence of over expression of CK, this together with an enhanced auxin content cause cell proliferation leads to tumour formation. Other CK-synthesis pathways are *Pseudomonas syringe* causes gall formation and *Rhodococeus fascians* causes fasciations and a growth abnormality called witches Broom disease.

He root-nodule forming and N-fixing plant symbiotic *Rhizobium* species as also known to produce CK. Recently enzymes involved in converting Trp to IPA have been isolated in Arabidopsis. The iaaM over expression lines define the characteristics of auxin overproduction in Arabidopsis thus providing important traits for identifying plant auxin biosynthetic genes. IAA can also be produces from Trp through the indole-3-pyruvate pathway (IPA) found in some microorganisms. The IPA decorboxylase, which catalyses the conversion of IPA to indole-3-acetaldehyde,has been cloned from *Enterobacter cloacae* and *Azospirillum brasilense*, but enzymes for catalysing indle-3-acetic acetaldehyde to IAA have not been conclusively identified in microorganisms. The microorganisms system may be useful for identifying the other genes involved in the IAA pathway in plants.

Nutrient partitioning under auxins and CK effect

CK are important regulators of development and environmental responses of plants that execute their action via the molecular machinery of signal perception and transduction. The characterization of the molecular mechanisms regulating hormone synthesis, signaling, and action are facilitating the modification of CK biosynthetic pathways for the generation of transgenic crop plants with enhanced abiotic stress tolerance (Peleg and Blumwald, 2011) [123]. Since plant hormones generally are assumed to interact with specific receptors that reside either on the cell surface or within the cytoplasm. Two candidates for a CK receptor have recently been identified. One of which tends to fit the steroid hormone receptor model while the other fits the membrane receptor model. It is possible, although unlikely, that both of these are CK receptors. Until recently, our knowledge of how CK works at the cellular and molecular levels is still quite fragmentary, significant progress has been achieved in regard to biosynthesis, metabolism, perception, and signal transduction (Fig. 1 and 2).

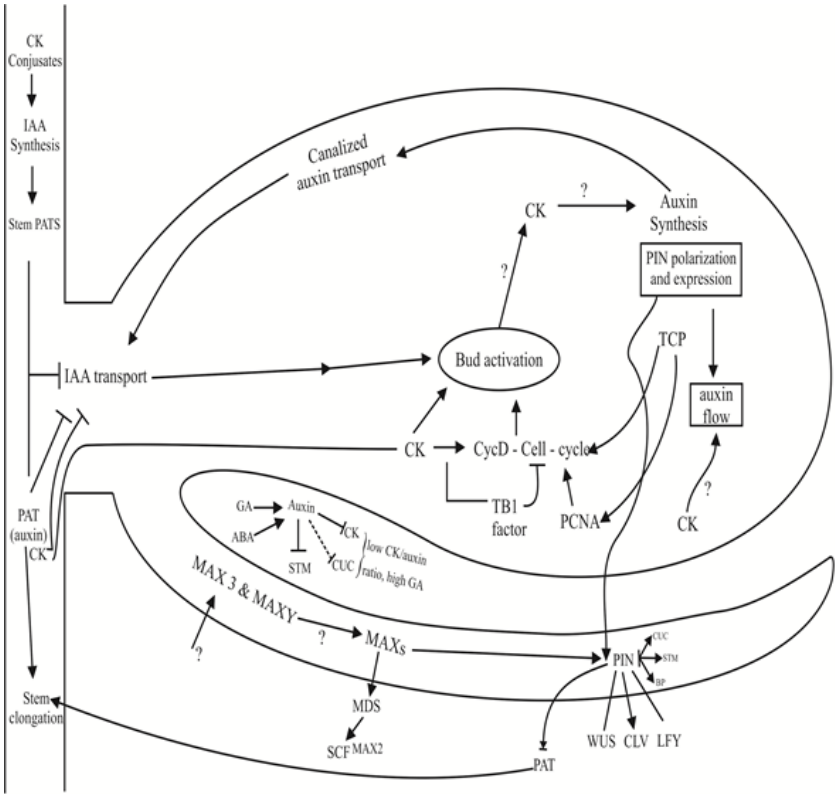

Fig 1: Cytokinin interaction during growth and development. A model in which Ck-AUX complex regulates but out-growth. An initial auxin flow towards an auxin sink promotes auxin transport canalization. The polarization and up-regulation of auxin transport feedback to promote flow of auxin repeatedly. In addition, the figure also demonstrate that Cks regulates TCR transcription factors and in turn regulate cell-cycle to control bud activation but it oppositely regulated buy auxin in the stem PATS and by CK (PCNA = Proliferating cell nuclear antigen; TB1-like = Teosinte Branched 1-like; TCP = Teosinte branched cycleoidea-PCF)

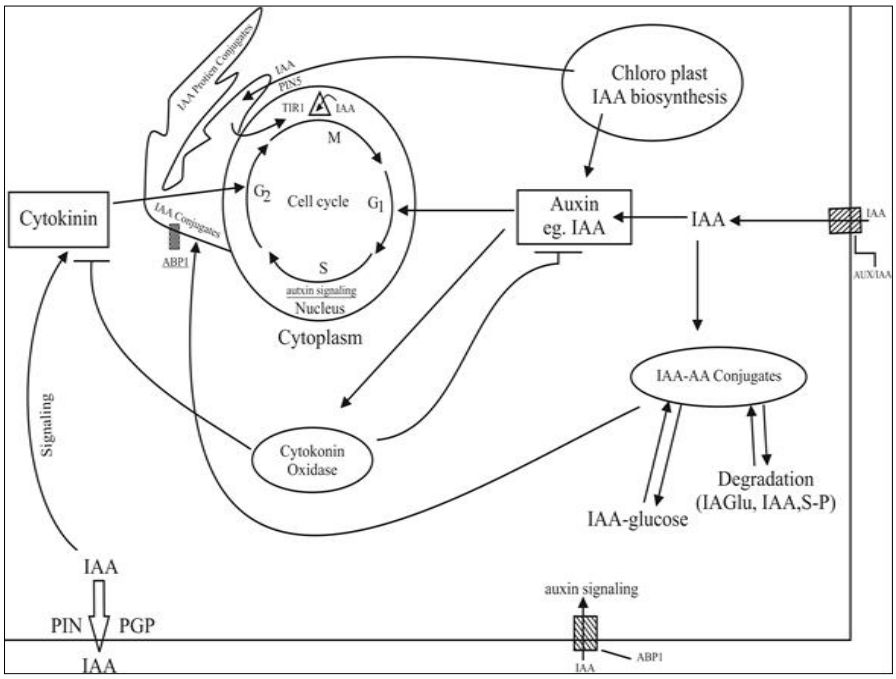

Fig 2: Role of cytokinin oxidase in degradation of auxin-cytokinin conjugates in plant system during cell division and metabolism.

Vercruyssen *et al*., (2011) [169] results show new interactions and contribute to the molecular and physiological understanding of biomass production at the whole plant level. In addition, they also help in delay senescence or the ageing of tissues are responsible for mediating auxin transport throughout the plant and affect internodal length and leaf growth. They have a highly synergistic effect in concert with auxins and the ratios of the two groups of plant hormones

affect most major growth periods during a plant's life time. Over the past few years, exciting progress has been made to reveal the molecular mechanisms underlying the auxin-CK action and interaction.

CK are plant specific chemical messenger that play a central role in the regulation of the plants cell cycle and numerous developmental programs. The first common natural CK was identified was purified from immature maize kernels and named zeatin. Several others CKs with related structures are known today. They are abundant in the root tip, the shoot apex and immature seeds. Aromatic CKs have an aromatic benzyl group at N6. They occur more rarely and much less is known about them. Because of their greater stability aromatic CKs are often used in tissue culture, an example is benzyl adenine. There are several reports suggesting that the accumulation of CK is closely correlated with the nitrogen status of the plants, such as *Urtica dioica* and maize (Takei *et al*., 2001). These studies suggest that CK metabolism and translocation could be modulated by the N-nutrition status. Remarkable findings from change of status from deficient to sufficient (Takei *et al*., 2001). Namely, CK accumulation and translocation occurred after sensing a change in N-availability. In maize roots following the addition of nitrate to nitrogen-depleted maize plants,isopentenyladenine-5^1-monophosphate (iPMP) started to accumulate in roots within 1 hr, preceding accumulation of trans-zeatin riboside-5^1-monophospate (ZMP), trans-zeatin riboside (ZR) and trans-zeatin (Z) (Takei *et al*., 2001). iPMP is the first molecule to be synthesized in CK metabolism, suggesting that CK was synthesized de novo in response to NO_3 supply. When changes in Z-O-glucoside (ZOG), an inactive and storage form of CK decreased after replenishment of nitrate in the maize roots. This reciprocal pattern against the accumulation of an active species implies that, in addition to biosynthesis, conversion from a storage form to an active form, also contribute to the accumulation of CK in roots.

To confirm the root-to shoot signal translocation, the xylem sap was collected after nitrate resupply and then applied to detached maize leaves. Treatments with xylem sap from N-resupplied maize plants resulted in the accumulation of the ZmRR1 transcript, whereas that from nitrogen-starved plants did not. It should be noted that ZmRR1 is a CK-responsive genes that functions as a response regulator involving N-signaling in the maize leaf (Sakakibara *et al*., 1999) [139]. This result suggests that the root-to-shoot xylem flow contain a signaling substance to induce the CK-response gene. In the previous studies, when CK species were removed from the xylem sap by the treatment with a polygonal antibody against CK, the inducible effect was greatly diminished (Takei *et al*., 2002) [150]. This is constant with the hypothesis that CK synthesized in the roots is the signaling substances communicating N-availability to the leaves. In barely roots, N-dependent accumulation of CK was inhibited by treatment with methionine sulphoximine, a potent inhibitor of glutamine synthetase, suggesting that the metabolic flow of N via glutamine is involved in the early step of the response. IPT, a CK biosynthesis enzyme, genes for IPT have been identified in *A. thaliana* (Takei *et al*., 2001) [152]. The enzyme is encoded by a small multigene family that includes both IPT and tRNA (tRNA-IPT). It is assumed that the expression of each gene in differently regulated in terms of the genes might be regulated by the nitrogen availability.

In maize and *A. thaliana*, some response regulators have been found to be up regulated by the both CK and nitrate (Imamura *et al*., 1999). The signal transduction systems referred to as the two-component system or His-Asp phosphorelay system has recently been uncovered in the plant kingdom (Sakakibara *et al*., 2000). It is widely used as a microbial signaling pathway, and recently, it has been revealed that some eukaryote such as yeast, and plants are equipped with this communication system. The findings of (Takei *et al*., 2002) [153] strongly suggest that the His-Asp phosphorelay operates in an inorganic N-signaling pathway mediated by CK in plants. In detached leaves, the effect can be replaced by treatment with CK, but not by inorganic N-sources (Sakakibara *et al*., 1998) [26], suggesting that the actual signal of the N-availability is CK. In the whole plants supplement of ammonium ions to the N-depleted maize also induced the Zm RR1 transcript, as is the case with NO_3-ions (Sakakibara *et al*., 1998) [26]. Although the changes in the amount of CK species depending NH_4^+ administration have not been determined, this result suggests that supplement of NH_4^+ also increased the translocation of CK to the leaf. Active CKs species that are newly synthesized from a stage form are translocated from the root to the shoot with further convenient to various active species, and Z is a possible signal substance communicating N-availability from the roots to the shoots. In the leaf cell, the His-Asp phosphorelay system transduces the signal to the target genes(s) or proteins(s). Although some of the key factor(s) involving the signal transduction process have emerged the whole body of the communicating systems is still unclear at present. It will be necessary to reveal the downstream target genes of the His-Asp phosphorelay system in order to understand the physiological function in the N-signaling pathway mediated by CK.

On the other hand, CK translocation via xylem is controlled by both environmental and endogenous signals. The tZR content and flow rate of the xylem sap are significantly increased by NO_3 supplement in barley (Samuelson *et al*., 1992) and maize (Takei *et al*., 2001b), implying the tZR act as a messenger for NO- signaling. Xylem CKs up regulated by NO_3^{2-} supplement induced by accumulation of CK responsive gene transcripts in leaves (Sakakibara *et al*., 1998; Takei *et al*., 2001b) [141]. In Arabidopsis roots, the accumulation of AtIPT3 transcripts is induced by NO_3^{2-} followed by that of tZ-ribotides and tZR (Takei *et al*., 2002; 2004a).

Furthermore, in an AtIPT3-deficient mutant, the nitrate-dependent accumulation of CKs was markedly reduced or diminished (Takei *et al*., 2004a), indicating that AtIPT3 in a key gene for the nitrate-dependent de novo biosynthesis of CKs. Promoter-reporter analysis with transgenic Arabidopsis plants showed that the AtIPT3 promoter is active in phloem companion cells rather than xylem tissues (Takei *et al*., 2004a) [52]. Thus, there may well be a CK translocation system operating between the phloem and xylem tissues. Studies on the increased-branching mutants of Pee (e.g., *rms 4*) (Beveridge *et al*., 1996) and Arabidopsis (*max 2*) (Stirnberg *et al*., 2002), in which the CK content of the xylem sap is dramatically reduced; imply a novel control mechanism for CK delivery via the xylem translocation stream. A scion-dependent reduction in xylem CK content was also observed

by reciprocal grafting experiments between *max 2* and wild type plants (Foo *et al*., 2007). Currently, in addition to CK and auxin, strigolactone is proposed as a novel phytohormones involved in branching (Gomez-Roldan *et al*., 2008) [44], but the identity of the signals regulating xylem CK levels remains to be elucidated.

Development of meristem versus CK and Auxins

Moreover, Su *et al*., (2011) briefly discuss the major progress made in CK transport and signaling. Further, this study also suggest the complicated interaction of these two hormones in the control of shoot apical meristem and root apical meristem formation as well as their roles in vitro organ regeneration. It has been known for many decades that auxin inhibits the activation of axillary buds, and hence shoot branching, while CK has the opposite effect.

The direct evidence that CK are involved in maintain SAM activity comes from the experiments that revealed the relationship between CK and the home box gene STM. Besides CK, other PGRS phytohormones have been regarded as indispensible players in plant developmental biology. Among, CK and auxins are plant specific hormones that regulate cell division and are the best known hormones involved in maintain meristem activity (Riou-Khamlichi *et al*., 1999) [132]. Recently, two independent laboratories presented further evidence to clarify the relationship between CKs and the homeobox genes drawing an identical conclusion that are up-regulated expression of STM will sufficiently induce the expression of CK biosynthesis genes. AtIPT and AtIPT7, which ultimately elevate the CK content. The application of CKs or expression of IPT driven by the STM promoter can rescue the stem mutant phenotype. On the other hand, auxin has a broad effect on plant development, particularly on AM initiation and developments; various AMs for leaves branches and flowers are derived from the PZ (Palcobutrazol) of the vegetative or reproductive SAMs (Fig. 1).

Auxins have been proposed as an essential regulator in this process based on a series of studies on auxin transport and distribution. In Arabidopsis, auxin carriers, especially the efflux carriers, have been identified and well characterized; 8 members of the PIN protein family have been demarcated to facilitate auxin efflux (Petrasek *et al*., 2006) [124]. PIN1, the most well-characterized auxin efflux carrier mediates auxin distribution and this triggers AM initiation (Benkova *et al*., 2003). In a vegetative SAM, PIN 1 localizes in the epidermis, the vasculature of developing leaf primordial, and the L1 and L2 layers of the SAM (Reinhardt *et al*., 2003) [131]. This is also the case when the L1 layer is distributed by microsurgical removal (Reinhardt *et al*., 2003) [131] (Fig. 2).

Because of the significance and complexity of the SAM in plant development, the maintenance and differentiation of the SAM in postembryonic development have drawn the interest of biologists for centuries. CK also controls the SAM size by participating in the CLV-WUS loop sustains the dynamic balance of SAM activities; there should be some other molecules that determine the position and timing of a primordium. These findings suggest that *ARR* genes might negatively influence meristem size and that their repression by WUS might be necessary for the proper function of meristems (Leibfried *et al*., 2005). Consistent with this hypothesis is the observation that overexpresssion of an active form of ARR7 produces an aborting SAM in Arabidopsis (Leibfried *et al*., 2005). Primary functions of AHK genes, and those of endogenous CKs, are triggering cell division and maintenance of the meristematic competence of cells to prevent subsequent differentiation until a sufficient number of cells has accumulated during organogenesis (Nishimura *et al*., 2004). Earlier observations that the shoot meristems overproducing CKs showed a similar phenotype to that of the transgenic plants over expressing KNAT1 or KN1 suggested that CK and homeobox genes may be involved in regulating SAM function in the same pathway and that CKs may act upstream of KNAT1 and STM (Rupp *et al*., 1999). Conversely, a loss of-function mutation in a maize AR homolog has an enlarged SAM (Giulini *et al*., 2004) [23]. Beyond the CLV-WUS feedback regulatory loop, WUS appears to be central players that integrate the regulatory information from several pathways to govern SAM activities. Another positive regulator of SAM maintenance is STM, which encode a homeodomin protein of the KN1 family. It has been shown that STM acts as an antagonist of CLV but synergistically sustain the balance of stem cell regeneration and differentiation (Lenhard *et al*., 2002). Consistent with its central position, the WUS promoter contain distinct regulatory region that control tissue specificity and levels of transcription in a combinatorial manner (Baurle and Laux, 2005).

Auxin was first regarded as a direct controller in the process of apical dominance (Thimann and Skoog, 1934). In many plant species, axillary buds became dormant owing to the inhibitors effects of the primary shoot apex on the outgrowth of AMs. CKs have been proposed as a second messenger that mediates the action of auxin in controlling apical dominance. CK are synthesized in roots and transported through xylem in to axillary buds to break the dormancy of arrested buds, whereas auxin modulates the CK concentration, this repressing AM out growth (Nordstrom *et al*., 2004). However, auxin has been found incapable of a accumulation in the inhibited AMs, suggesting an indirect suppression effect of auxin on AM out growth (Booker *et al*., 2003) [165]. Although the outgrowth behaviour between dicot and monocot AMs are apparently different but they appear to share a conserved MAX-involved carotenoid-derived branching signal pathway because orthologs/homologs of Max 2 to Max 4 have also been identified in rice.

The number of branches is an essential detriment of plant architecture. However, for monocotyledons crops the angle between the shoot branches and the main culms, termed the tiller angle in gross species, is also an important agronomic trait that should be considered. Dicotyledons roots have few primary xylem strands and their lateral roots initiation typically occurs in pericycle cells located immediately outside the protoxylem point (Casimiro *et al*., 2001) [24]. However, lateral roots may also develop in various positions between the protoxylem strands, e.g., near the phloem in polyarch roots. In all cases, the vascular tissues of the main root are continuous with those of a lateral root because the root tip is a sink for the IAA-inducing signal. Naturally occurring xylem regeneration around adventitious roots in hypocotyls of *Luffa cylindrica* seedlings marks the sites where the polar IAA transport is interrupted inside the vascular bundles by a naturally

occurring signal. The combination of both adventitious root ignition and xylem regeneration at the same site of a vascular bundle can be promoted by main root decapitation (Aloni and Baun, 1991), or by external ethylene application indicating that wound-induced ethylene or elevated ethylene concentration might interrupt the polar IAA transport along two naturally occurring pathways inside a vascular bundle. The ethylene is released from the differentiating-protoxylem vessels elements and diffuses to the neighbouring tissues; in the centrifugal direction; it locally inhibits IAA movement in the pericycle. Therefore, immediately above this IAA inhibition site, newly arriving IAA from young leaves is locally accumulated in the pericycle; this fact IAA build-up stimulation cell divisions in the pericycle founder cells (Casimiro *et al*., 2003) [25] and the initiation of a lateral roots primordial in the xylem pole. The high CK concentration at the root cap antagonizes IAA and inhibits lateral root initiation in the vicinity of the tip, which is crucial for CK enabling uninterrupted elongation of the root tip. Therefore, central roots initiate further away from the CK-synthesizing cap, occurring above the elongation zone (Van Staden and Ntigane, 1996), and this ensuring the elongation of a shoot primary root tip from lateral roots. Above the elongation zone where concentrations of CK decrease lateral roots can initiate. As auxin and CK concentration along the parent root are thought directly to control lateral root initiation and emergence, they could regulate root apical dominance (Lloret and Casero, 2002).

CKs signaling and phosphorelay

Higher plants are composed of multiple organ system that is functionally differentiated, such as photosynthetic and non-photosynthetic organs and vegetative and reproductive organs. Plants organs interact with each other to optimize both metabolic and developmental processes to allow the organisms to accommodate to environmental inputs. For these mutual interactions, local and long distance communication among cells and organs are essential. CKs, a class of phytohormones are one of these long distance messenger transported thorough the plant vascular system. The past decade has seen substantial advances in know-lodge of molecular mechanisms and activities of plant hormone, but only in the past few years has research on CK began to hit stride. Recent studies have demonstrated that CK signaling involves a multistep two component signaling pathway, resulting in the development of a canonical model of CK signaling that is likely representative in plants (Sheen, 2002). CKs are master regulator of a large number of processes in plant development, which is known to be unusual plastic and adoptive, as well as resilient and perpetual. These characteristic allow plants to respond sensitivity and quickly to their environment. Together with auxin, CKs can reprogram terminally differentiated leaf cells into stem cells and support shoot regeneration indefinitely in plant tissues culture (Mok and Mok, 2001) [112]. Research interest in the signaling pathways activated by CKs has increased recently because of new information arising from studies of Arabidopsis and the completion of its genome sequence. In the Arabidopsis CK signal transduction pathway, hybrid histidine protein kinase (AHKS) serve as CK receptor and histidine phosphotransfer protein (AHPs) transmit the signal from AHKs to ARRs which can activate or repress transcription (Kakimoto, 1996; Inoue *et al*., 2001) [65, 55]. There are four major steps to CK signaling: AHK sensing and signaling, AHP nuclear translocation, ARR transcription activities, and a negative feedback loop through CK-inducible ARR gene products. The multistep two-component phosphorelay mechanism found in Arabidopsis in reminiscent of the bacterial two-component signaling system (Stock *et al*., 2000) [152], but it is linked by AHPs, which shuttle from the cytoplasm to the nucleus in a CK-dependent manner (Hwang and Sheen, 2001). Functional analysis of AHKs, AHPs and ARRs in *E.coli*, yeasts plants and a leaf protoplast systems, and protein-protein interactions in yeast two hybrid assays have providing compelling evidence for the importance of multistep two component phosphorelay in CK signaling (Haberer and Kieber, 2002; Lohrmann and Harter, 2002). In Arabidopsis, at least three genes encode CK receptors: AHK4 (CRE1), and WOODEN LEG (WOL), AHK2 and AHK3 (Inoue *et al*., 2001; Ueguchi *et al*., 2001). Quantitative transcription analysis based on CK-inducible ARR6-LUC receptor gene activity suggests that *CKI1* and AHKs act through different CK perception mechanisms. The function of AHK4 has been thoroughly demonstrated by direct CK binding and by the isolation of *cre1* and *wol* mutants that exhibits defect in CK-mediated shoot induction from callus and root vascular morphogenesis, respectively (Inoue *et al*., 2001; Mahonen *et al*., 2000) [84]. Further analysis of cellular expression pattern, CK binding and chimeric AHKs with swapped domains showed clarify the underlying mechanism of each AHK action in CK signaling. In Arabidopsis, there are more AHKs, ARRs and related proteins than there are AHPs (Hwang and Sheen, 2001). The B-type ARR transcription activators (ARR1, ARR2 and ARR10) carry MYB-like domains for DNA binding and a glutamine (Q)-rich domain for transcriptional activation and they activate CK-responsive ARR6 transcription (Hwang and Sheen, 2001; Sakai *et al*., 2000). Mutation in the conserved Asp residue of ARR2 does not abolish its function as transcription activation for a CK-phosphorylation may not ARR6 promoter, suggesting that phosphorylation may not intrinsically activate the transcription factor (Hwang and Sheen, 2001). Thus, phosphorylation likely eliminates negative regulation of ARR1, ARR2 and ARR6 etc. Ectopic expression in transgenic Arabidopsis of ARR2, one of the rate-limiting transcription factors in the response to CK, is sufficient to mimic CK in promoting shoot meristem proliferation and leaf differentiation and in delaying leaf senescence (Hwang and Sheen, 2001). Although, the B-type ARRs with transcriptional activation activities are likely the major regulators of a broad spectrum of CK target genes, the A type, ARRs could also contribute to the output of CK signaling through protein-protein interactions (Haberer and Kieber, 2002; Lormann and Harter, 2002). ARR4 also interacts with phytochrome B and modulates light signaling. Thus, two-component elements could serve as the molecular links in a complex plant growth signals such as plant hormone, sugars light and other environmental cues. However, CK response can also occur in other cell types (D Agostino, 2000) [31]. This broad cellular competence to CK responses may explain the plasticity of plant development. In

present scenario, the major challenge is to determine how a conserved CK signal transduction pathway influences cell-cycle, leaf senescence, shoot initiation, and leaf patterning in different cell types at various developmental stages.

Basis of plant architecture

The molecular basis of cellular auxin transport is still not fully understood. Although a number of carriers have been identified and proved to be involved in auxin transport, their regulation and possible activity of as yet unknown transporters remain unclear. Nevertheless, using single-cell-based systems it is possible to track the course of auxin accumulation inside cells and to specify and quantify some auxin transport parameters. The synthetic auxins 2,4-D and NAA are generally considered to be suitable tools for auxin transport studies because they are transported specifically via either auxin influx or efflux carriers, respectively. Research indicates that NAA can be metabolized rapidly in tobacco BY-2 cells. This implies that the transport efficiency of auxin efflux transporters is higher than previously assumed. Moreover, using data on the accumulation of 2,4-D measured in the presence of auxin transport inhibitors, it is shown that 2,4-D is also transported by efflux carriers. Based on the accumulation data, a mathematical model of 2,4-D transport at a single-cell level is proposed. Optimization of the model provides estimates of crucial transport parameters and, together with its validation by successfully predicting the course of 2,4-D accumulation, it confirms the consistency of the present concept of cellular auxin transport (Hosek *et al*., 2012) [51].

Crop plant architecture determines planting density in the field and thus influences, to a large degree, the light harvest, disease resistance, and lodging. Rice is one of the most important staples and feeds more than half of the world population of world. And therefore rice attracts tremendous attention in crop breeding. Therefore, rice plant architecture is regarded as one of the most important factors that affect rice yield (Khush, 2003) [66]. Conventional breeding has been playing an essential role in rice cultivar innovation for centuries. However, compared with the current population explosion and crop land reduction, progress is rather slow owing to several barriers such as the time-consuming cross/selection process and limited appropriate selection for desired genotypes. Best example is the Green Revolution, the innovation of semi dwarf wheat and rice cultivars that has important crop production increasingly since 1960. The ability to produce more food in the same acreage is crucial to feeding an increasing world population and also important for curbing deforestation and dedicating more land to biofuels. Molecular design refers to pyramiding one or more genes into a plant for modifying one or more target traits via genetic engineering.

Moreover, Hartmann *et al*., (2010) [47] established an *in vitro* assay using excised tuber buds to study the dormancy-releasing capacity of GA and CK and show that application of GA_3 is sufficient to induce sprouting. GA_3-treated wild-type and CKX-expressing tuber buds were subjected to a transcriptome analysis that revealed transcriptional changes in several functional groups, including cell wall metabolism, cell cycle, and auxin and ET signaling, denoting events associated with the reactivation of dormant meristems.

Moreover, scientists from the International Rice Research Institute have proposed a model of the real rice plant architecture, which should have a low tiller number (9-10 tillers for transplanted conditions); a high number of productive tillers; 2000-2500 grains per panicle; dark-green, thick and erect leaves; and vigorous and deep root systems. The tiller number or the tiller angle of rice can be manipulated by altering the expression level of MOc1 or LAI and TAC1 (Xing and Zhang, 2010) [174]. Besides this, the presently available genome sequences and mature techniques for genetic manipulation will facilities and energetically allow plant breeders to modify and design elite cultivars that have ideal plant architecture, inbreed quality, efficient use of nutrients, and strong resistance to pest and disease. Therefore all their achievements indicate that agronomically important plant architecture traits can be improved at the molecular level through genetic engineering. However, unlike GM cotton and maize that are mainly for industrial and feed uses, GM rice will be the first commercial staple crop in the world. Studies assessing productivity and health effects of insect-resistant GM rice varieties have provided positive evidence that farmers will benefit from adopting the GM rice with higher crop yields and reduced use of pesticide.

Axiallary meristems are major determinants of plant architecture. A key component of architectural variations in the degree and pattern of shoot later organs, by which man can distinguish different kinds of plants in the natural world even without advanced technologies or knowledge. Although, the pattern of lateral organs is flexible, to some extents, in response to environmental conditions, it is essentially determined by the genetic makeup. Therefore, so far, a number of mutants defective in lateral organs pattern have been found in nature or generated by mutagenesis or T-DNA insertion. These mutants can be classified into three classes on the basis of whether they affect meristem initiation, meristem outgrowth or both. Elucidation of the molecular mechanism underlying rice plant architecture provides a solid idea for modifying the plant architectural pattern of food crops. Recent transgenic studies showed that some genes affecting plant architecture have potential applications in genetically engineering the ideal plant architecture of crops. For instance, the Green Revolution rice sd1 cultivars are GA biosynthesis mutant indicating that plant height can be manipulated through modifying the GA biosynthesis or signaling pathway at the molecular level (Neeraja *et al*., 2009) [117].

In addition, over-expression of EUI in rice results in dwarf or EUI-like transgenic plants, and the gai protein is used in a switchable expression system in which the *gai* gene can be induced by ethanol to inhibit growth at the various stages in AlcA;gai transgenic plants demonstrating the possibility that the *gai* gene can be used to provide the dwarf phenotype in a range of crop species. The sub cellular targeting of the constituent proteins is regenerated by components involved in endosomal trafficking and also depends on the phosphorylation status of the proteins as well as components that determine membrane composition (Kleine-Vehn and Friml, 2008) [68]. Feedback a forward loops represents ethanol mechanism in developmental biology and there are strong indication that such loops play a critical role in auxin role in developmental systems biology of plants such as phyllotaxis, vascular patterning and root pattering.

Synergistic or Antagonistic approach of CK-auxins for root development

Auxin is the main regulator of root apical meristem (RAM) functioning, and auxin maxima coincide with the sites of RAM initiation and maintenance. Auxin gradients are formed due to local auxin biosynthesis and polar auxin transport. The PIN family of auxin transporters plays a critical role in polar auxin transport, and two mechanisms of auxin maxima formation in the RAM based on PIN-mediated auxin transport, have been proposed to date: the reverse fountain and the reflected flow mechanisms. Regeneration of the RAM in decapitated roots is provided by the reflected flow mechanism. The dual-mechanism model proposed can be a powerful tool for the study of several different aspects of auxin function in root. Moreover, it has been shown that the transcriptional regulator phytochrome-interacting factor 4 (PIF4) and the auxin are involved in the regulation of high temperature-induced hypocotyl elongation in Arabidopsis. PIF4 regulates high temperature-induced hypocotyl elongation through direct activation of the auxin biosynthetic gene YUCCA8 (YUC8), and also that high temperature also co-upregulates the transcript abundance of PIF4 and YUC8. PIF4-dependency of high temperature-mediated induction of YUC8 expression as well as auxin biosynthesis, together with the finding that over expression of PIF4 leads to increased expression of YUC8 and elevated free IAA levels in planta, suggests a possibility that PIF4 directly activates YUC8 expression. Indeed, gel shift and chromatin immune-precipitation experiments demonstrate that PIF4 associates with the G-box-containing promoter region of YUC8. A molecular framework by which the PIF4 transcriptional regulator integrates its action into the auxin pathway through activating the expression of specific auxin biosynthetic gene. These studies advance our understanding on the molecular mechanism underlying high temperature-induced adaptation in plant architecture (Sun *et al*., 2012) [155].

Mutant analyses suggest that auxin transported from the shoot represses root greening via the function of INDOLE-3-ACETIC ACID14, AUXIN RESPONSE FACTOR7 (ARF7), and ARF19. The regulation by auxin/CK is dependent on the transcription factor LONG HYPOCOTYL5 (HY5), which is required for the expression of key chlorophyll biosynthesis genes in roots. The expression of yet another root greening transcription factor, GOLDEN2-LIKE2 (GLK2) found to be regulated in opposing directions by auxin and CK. Furthermore, both the hormone signaling and the GLK transcription factors modified the accumulation of HY5 in roots. Over expression of GLKs in the hy5 mutant provided evidence that GLKs require HY5 to maximize their activities in root greening. The combination of HY5 and GLKs, functioning downstream of light and auxin/CK signaling pathways, is responsible for coordinated expression of the key genes in chloroplast biogenesis (Kobayashi *et al*., 2012) [69].

Hormonal regulation of root development is along known phenomenon. Plant root development is ensured by activity of primary and lateral root meristems. Primary root (PR) meristem forms during embryogenesis and it acquires its final size soon after germination. In contrast, lateral root (LR) meristem is formed post-embryonic cells. In the past decades, the molecular mechanisms of included hormonal pathways and their impact on root development have been studied. Recent genetic and molecular studies suggest importance of interactions of the individual hormone pathways and their components. Studies of Kuderova and Hejatko (2009) show impact of endogenous CK on the root architecture and its interaction with auxin in *Arabidopsis thaliana* interactions of plant hormone that act in defined cells at specific time-points are thought to be crucial for meristems formation. The CK has been shown to affect the correct stem-cell organization of PR meristem in embryo (Muller and Sheen, 2008) [113], root meristem size (Kuderova *et al*., 2008; Nishimura *et al*., 2004) [72], lateral root formation (Kuderova *et al*., 2008) [72]. All these CK controlled processes are also affected by auxins (Blilou *et al*., 2005) [21]. But, however recent data that provide first insights into molecular mechanisms and spatiotemporal specificity of CK-auxin interactions during not growth and development.

Auxin antagonizes CK signaling during formation of stem cell niche of the root meristem early embryogenesis. The core of the PR meristem consists of highly organized etc; cells termed the stem cell niche (Sabatini *et al*., 2003) [137]. Formation of local auxin gradients and the activity of genes regulating auxin efflux were shown to be critical for its proper formation (Aida *et al*., 2004; Sabatini *et al*., 1999) [137]. Recently, the construction of receptor genes expressed under control of a novel CK signaling-responsive, TCs (two-complex-output-sensor) promoter allowed visualizing CK signaling response in this process. Interestingly, TCs activity was counteracted by auxin via auxin-induced expression of type A, ARR7 and ARR15, the CK primary response genes and negative regulator of CK signaling (Muller and Sheen, 2008) [113]. Thus, auxin inhibits CK signaling in specific embryonic cell lineages. Root growth is ensured by the activity of meristematic cells. While auxin increases the size of the cell-division zone by promoting cell division (Dello Loio *et al*., 2008; Blilou *et al*., 2005) [21]. CK acts as a negative regulator of the root meristem size (Werner *et al*., 2003; Laplaze *et al*., 2007) [77, 170].

Recently, CK has been shown to control the meristem size by promoting cell differentiation specifically in the vascular tissue at the transition zone located between the cell-division and the cell-elongation/differentiation zone. It is mediated by CK perception via AHK3 and B-tpye ARR, ARR1 and ARR12 which act as downstream of AHK3. This site specific CK action is sufficient to affect the rate of cell differentiation of all other tissue of the transition zone by antagonizing a non-cell autonomous signal, suggested to be auxin. Similarily, Kuderova and Hejatko (2009) have shown that endogenous CK levels change during early stages of the PR development and that there are a developmental window specifying the increasing sensitivity of the root towards endogenous CK (Kuderova *et al*., 2008) [72]. Recently, CK has been proved to activate SHORT HYPOCOTYL2 (SHY2/IAA3), a repressor of auxin signaling and negative regulator of auxin efflux carrier genes from the PIN-FORMED family (PIN) via ARR1. On other hand, auxin mediates decrease in the activity of SHY2/IAA3 (Dello Ioio *et al*., 2008) [32]. Root meristem is a dynamic structure which forms and retains its activity as a result of consecutive steps of a developmental program

including cell division and cell differentiation. CK and auxins are up regulators of these two processes. All the spatiotemporal specificity. All these findings suggest spatiotemporal specificity of the CK and auxin interaction during post embryonic maturation of PR meristem. On the other hand, root growth is ensured by the activity of meristemtic cells. While auxin increases the size of the cell-division zone by promoting cell division, CK act as a negative regulator of the root meristem size (Werner *et al*., 2003) [170]. This site specific CK action is sufficient to affect the rate of cell differentiation of all the other tissues of the TZ by antagonizing a non-cell autonomous signal, suggested to be auxin. Similarly, it has shown that endogenous CK levels change during early stages of the PR (plant root) development and that there are a developmental window specifying the increases sensitivity of the root towards endogenous CK (Kuderova *et al*., 2008) [72]. All these above findings suggest spatiotemporal specificity of the CK and auxin interaction during post-embryonic maturation of PR meristem. In addition, Laplaze *et al*., (2007) [77] have further shown that endogenous CK inhibits LR formation by directly affecting LR initiation and that application of exogenous CK correlation with miss expression of genes of auxin-efflux carriers for the PIN finally in initiating and develops LRP.

Auxins and CK: Root architecture and gravitropism

Rademacher *et al*., (2012) show that a cell-autonomous auxin response within this cell is required for root meristem initiation. ARF9 and redundant ARFs, and their inhibitor IAA10, act in suspensor cells to mediate hypophysis specification and, surprisingly, also to prevent transformation to embryo identity. ARF misexpression, and analysis of the short suspensor mutant, demonstrates that lineage-specific expression of these ARFs is required for normal embryo development. These results imply the existence of a prepattern for a cell-type-specific auxin response that underlies the auxin-dependent specification of embryonic cell types.

Auxin-related gene expression also altered both in the short term (TRPB) and in the long term (GH3, TIR1 and PIN1), indicating that auxin plays different roles during development and ripening processes (Torrigiani *et al*., 2012) [165]. GH3 proteins control auxin homeostasis by inactivating excess auxin as conjugates of amino acids and sugars and thereby controlling cellular bioactive auxin. Since auxin regulates many aspects of plant growth and development, regulated expression of these genes offers a mechanism to control the various developmental processes. OsMGH3/OsGH3-8 is expressed abundantly in rice florets and is regulated by two related and redundant transcription factors, OsMADS1 and OsMADS6, but its contribution to flower development is not known. Yadav *et al*., (2011) [176] functionally characterize OsMGH3 by over expression and knock-down analysis and show a partial overlap in these phenotypes with that of mutants in OsMADS1 and OsMADS6. The over expression of OsMGH3 during the vegetative phase affects the overall plant architecture, whereas its inflorescence-specific over expression creates short panicles with reduced branching, resembling in part the effects of OsMADS1 over expression (Yadav *et al*., 2011) [176].

Moreover, gravity profoundly influences plant growth and development. Plants respond to changes in orientation by using gravitropic responses to modify their growth. Cholodny and Went hypothesized over 80 years ago that plants bend in response to a gravity stimulus by generating a lateral gradient of a growth regulator at an organ's apex, later found to be auxin. Auxin regulates root growth by targeting Aux/IAA repressor proteins for degradation. Band *et al*., (2012) [10] used an Aux/IAA-based reporter, domain II (DII)-VENUS, in conjunction with a mathematical model to quantify auxin redistribution following a gravity stimulus. Multidisciplinary approach revealed that auxin is rapidly redistributed to the lower side of the root within minutes of a 90° gravity stimulus. Unexpectedly, auxin asymmetry was rapidly lost as bending root tips reached an angle of 40° to the horizontal.

From the sites of hormone production, the signals move in specific structural pathways and by different mechanisms to regulate plant development and differentiation (Aloni *et al*., 2005). In this context, CK and IAA are two key hormones that regulate root development, its vascular differentiation and root gravitropism; these two hormones, together with ethylene, regulate lateral root initiation (Aloni *et al*., 2006). CK synthesized in the root cap, promotes CK, vascular cambium sensitivity, vascular differentiation and root apical dominance. Auxin (IAA) produced in young shoot organs, promotes root development and induces vascular differentiation. In addition, both IAA and CK regulate root gravitropism (Aloni *et al*., 2006). The auxin hormone and its polar movement originating in young shoot organs (Aloni *et al*., 2006), play a crucial role in many aspects of root growth, development and differentiation. IAA regulates the development of the primary and lateral roots (Teale *et al*., 2005; Taiz and Zeiger, 2002) [163, 157], the quiescent centre, root apical meristem, root cap and root vascular differentiation (Jiang and Feldman, 2005; Ponce *et al*., 2005; Aloni *et al*., 2004) [58, 125].

It has been suggested that the differentiation of phloem strands and phloem anastomoses between the vascular strands are induced by streams of low IAA concentrations (Aloni, 1995). When a high auxin concentration was applied to decapitated *Luffa cylindrica* stems, it induced xylem differentiation in its phloem anastomoses (Aloni, 1995) [2], indicating the need for high auxin stimulation to induce xylem differentiation. In leaves, the proximity between the sites of IAA probably results in relatively high local IAA concentration at the differentiation sites (Aloni *et al*., 2004) [4] which may explain why in leaves xylem can differentiate in the absence of phloem at the freely ending vein lets (Horner *et al*., 1994) and hydathodes (Aloni *et al*., 2003) [2]. The primary vascular tissues and the pericycle are the main pathways of phloem IAA transport. In flowering plants, there are two common primary vascular morphogenesis, as exemplified by monocot roots and dicots roots. Xylem strands made up of vessels are either distributed around the periphery of the vascular cylinder and the centre of the cylinder in parenchymatous or the centre of the vascular cylinder is occupied mainly by vessels with xylem standards radiating out to the periphery of the cylinder. The high-concentration IAA streams induce the protoxylem vessels. The relatively concentration IAA flow may also occupy the centre of the vascular cylinder resulting in differentiation of metaxylem vessels. Because low-concentration streams are enough to induce protoxylem sieve

tubes, they are induced and mature first and closer to the root tip than the protoxylem vessels.

There are a linear co-relation between the number of protoxylem strands and circumference of the vascular cylinder (Fahn, 1990) [35]. An increase in surface circumference enable more free space for additional separate IAA streams, which results in an increase number of protoxylem strands and protoxylem strands among them. Pattern analysis of the phloem and xylem strands in adventitious monocots roots has revealed is induced by a separate independent IAA stream. The primary xylem in roots matures centripetally and characterized as exarch. It has been suggested that IAA flow in the pericycle influences the width of primary vessels in the roots vascular cylinder. By contrast, the metaxylem vessels, which differentiate away from the pericycle, have more time to expand before secondary wall deposition and therefore become wide vessels. The differences between the initiation of the two types f roots is that in the shoot the ethylene which induces adventitious root initiation, is revered from a vascular bundle; this ethylene which diffuses into the centrifugal direction, locally inhibits polar IAA.

Transport in the bundle sheath (Aloni, 2004) [3], whereas in the root the ethylene originates from a protoxylem strands and locally inhibits IAA movements in the pericycle. External ethylene applications that induce ethylene production (Mergemann and Sauter, 2000) [106] cause local inhibitions of IAA transport in root pericycle or in a stem bundle sheath; immediately above these IAA-inhbition sites, the IAA accumulation in these two IAA transporting tissues induce the initiation of lateral and adventitious roots respectively (Aloni, 2004) [3].

The phenomenon of root apical dominance can be cylindrical with the well-known shoot-apical dominance; in both causes the actively growing leader inhibits limited organ inhibition and development. Decapitation of a shoot results in a rapid growth of one or more axiallary buds below the cut, suggesting that the polar flow of IAA produced in the apical bud and young leaves inhibits the outgrowth of axiallary buds (Taiz and Zeiger, 2002) [157]. Root apical dominance may occur in wild type-plants with an actively growing primary root that inhibits lateral root initiation by the root cap synthesized CK, and their lateral root develops further away from the root tip. By contrast low CK content in CK-deficient transgenic plants (Werner *et al*., 2003) [170], or almost CK insensitivity results in the formation of lateral roots closer to the root tip and an increase in root branching (Schmuling, 2002).

In the root cap, sedimentation of starch grains (amyloplasts) in gravity-sensing cells (statocysts) enables gravity perception and response (Sievers and Volkmann, 1972). The root cap cells produce CKs, which appear to regulate growth and gravitropism (Aloni *et al*., 2004) [3]. The findings that there is no gravity response after root cap removal (Shaw and Wilkins, 1973) [146] and that CK production occurs in the root cap, which is detected by both IPT5 expression (Miyawaki *et al*., 2004) [109] and ARR5::GUS expression (Aloni *et al*., 2004) [3], suggest that it is likely that the CK produced in the cap is the primary signal of the root statocysts. Surgical removal of half of the cap from a vertical root results in root bending towards the side with the remaining half-cap. Similarly, lateral exogenous application of CK to a vertical root induced root bending towards the site of application, confirming the inhibitory effect of CK on root elongation during gravitropism (Aloni *et al*., 2004) [3]. Roots of the triple loss-of-function CK receptor, the *ahk* mutant, are almost insensitive to CK but respond to gravity, indicating that in CK-insensitive mutants the gravity response could be regulated only, or mainly, by IAA. Hydrotropism research suggests a possible role for abscisic acid (ABA) in controlling the direction of tropic root growth (Eapen *et al*., 2005) [34]. Detailed studies of the root's gravitropic curvature (Wolverton *et al*., 2002) [172] have revealed that the downward curvature is initiated in the region just behind the root meristem defined as the distal elongating zone (DEZ), and not in the central elongation zone (CEZ) of the root which is inhibited by external auxin application (Ishikawa and Evans 1993). Unlike the CEZ cells located further basal in the root, the cells in the DEZ are not susceptible to inhibition by elevated concentrations of auxin (Ishikawa and Evans, 1993), but they do respond to external CKs application (Aloni *et al*., 2004) [3]. The late, slower phase of gravity root bending occurs in the CEZ and is probably regulated by IAA (Wolverton *et al*., 2002) [172]. The downward curvature starts near the root apex during the early phase of gravity response by inhibiting growth at the lower root side and promoting elongation of the upper side at the distal elongation zone closely behind the root cap (Aloni *et al*., 2004) [3]. In CK-deficient transgenic Arabidopsis plants, which over express the CKX genes and have only 30–45% of the wild-type CK content (Werner *et al*., 2003), indicating that CK retards root elongation in wild-type plants. CKs produced in the shoot (Miyawaki *et al*., 2004; Takei *et al*., 2004) [109] are not involved in the regulation of root gravitropism. This was evident in horizontally orientated roots of young. Arabidopsis plants, in which the CKs in the vascular cylinder was retained by the endodermis and therefore was not involved in regulating root gravitropism (Aloni *et al*., 2004) [3]. In summary, the extreme developmental plasticity of roots is regulated by complexes of diverse external and internal signals, in which hormones may mediate the external stimulation and adapt the root to changing environment, e.g. response to gravity.

Conclusion

Plants display enormous variations during development of body plans, including both genetically determined differences between individuals and plastic responses of a single genotype to environmental conditions. The integration of the multiple inputs is likely to be mediated by a network of integrating hormonal signals that move systematically through the plant. Various studies revealed antagonistic interactions between auxin and CK in regulating bud outgrowth. Progress has been made in understanding how auxin-CK mediates a specific developmental output. In root meristem, CK affects depends on their antagonistic interaction with auxin. Auxin suppresses CK signaling at embryo stage to ensure the establishment of a proper embryonic root-stem cell niche whereas post-embryonically, CKs mediate cell-differentiation, suppressing auxin signaling and transport, thus controlling root meristem size and root growth. CKs output might depend on the differentiation context in which they are acting, as they

mediate cell differentiation in the root meristem but promote cell division in the shoot meristem.

Recent advances provide a framework by which one can understand auxin-CK signaling cross-talk in order to developmental programme of the plant. They highlight key challenges remaining. A characterization of processes that like auxin-CK cross-talk to changes in auxin-CK complex stability to allow for diverse responses to auxin, the exact manner in which this complexity is encoded to produce each specific auxin-response is still unknown. The relationship between auxin and CK in bud regulation has been debated and in recent years, there are still many fundamental unanswered questions. Alternative hypothesis have emerged, and efforts to design experiments to distinguish between these approaches showed continued progress in this field. Of particular importance are the arena of action of particular hormones, and the roles of transportation routes for both hormones and transcription factors involved in the processes.

The past decade has been impressive strides in dissecting the molecular basis of auxin-crosstalk to other plant hormones in plant body. Advances in our understanding of the integration of plant signaling processes at the transduction level have relied, relay and will continue to rely heavily on the application of genetic approaches in the model plant *Arabidopsis thaliana*. Such these studies have helped in the identification of important compounds of auxin and CK signaling pathways. At the protein level, novelistic interactions between newly discovered components from nominally discrete signaling pathways will be detached through the application of two-hybrid/proteomic-based approaches or the use of high-throughput protein-chip-based technologies. Nevertheless, micro-array-based expression analysis represents the genomic technology most likely to have an immediate impact in this area of research. By ability to transcript profiling, the entire Arabidopsis genome opens up unprecedented opportunities to investigate cross-talk at the level of genome expression. Transcript profiling primary response mutants from distinct transduction pathways is likely to uncover common target genes, whose potential dual signaling function can be treated by a reverse genetic approach. Large scale reverse genetic approaches will be needed to address the signaling functions of members within families of related transduction components. Moreover, the rich molecular researches available, Arabidopsis will continue to represent the model environmental systems to study hormone cross-talk but however, one must not overlook the rich diversity of signaling mechanisms that have evolved in other plant species and endeavour to adopt a comparative research approach.

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