Annals of Botany **112**: 391–408, 2013
doi:10.1093/aob/mcs285, available online at www.aob.oxfordjournals.org

ANNALS OF BOTANY
Founded 1887

REVIEW: PART OF A SPECIAL ISSUE ON MATCHING ROOTS TO THEIR ENVIRONMENT

Responses of root architecture development to low phosphorus availability: a review

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Received: 16 August 2012 Revision requested: 18 October 2012 Accepted: 14 November 2012 Published electronically: 23 December 2012

• *Background* Phosphorus (P) is an essential element for plant growth and development but it is often a limiting nutrient in soils. Hence, P acquisition from soil by plant roots is a subject of considerable interest in agriculture, ecology and plant root biology. Root architecture, with its shape and structured development, can be considered as an evolutionary response to scarcity of resources.
• *Scope* This review discusses the significance of root architecture development in response to low P availability and its beneficial effects on alleviation of P stress. It also focuses on recent progress in unravelling cellular, physiological and molecular mechanisms in root developmental adaptation to P starvation. The progress in a more detailed understanding of these mechanisms might be used for developing strategies that build upon the observed explorative behaviour of plant roots.
• *Conclusions* The role of root architecture in alleviation of P stress is well documented. However, this paper describes how plants adjust their root architecture to low-P conditions through inhibition of primary root growth, promotion of lateral root growth, enhancement of root hair development and cluster root formation, which all promote P acquisition by plants. The mechanisms for activating alterations in root architecture in response to P deprivation depend on changes in the localized P concentration, and transport of or sensitivity to growth regulators such as sugars, auxins, ethylene, cytokinins, nitric oxide (NO), reactive oxygen species (ROS) and abscisic acid (ABA). In the process, many genes are activated, which in turn trigger changes in molecular, physiological and cellular processes. As a result, root architecture is modified, allowing plants to adapt effectively to the low-P environment. This review provides a framework for understanding how P deficiency alters root architecture, with a focus on integrated physiological and molecular signalling.

Key words: Low phosphate, phosphorus acquisition, primary root, lateral root, root hair, cluster root, sugars, auxins, ethylene, cytokinins, nitric oxide, reactive oxygen species.

INTRODUCTION

Nutrient stresses have increasingly curtailed crop production due to scarcity of resources. Phosphorus (P) is a critical macronutrient required for numerous functions in plant, including energy generation, nucleic acid synthesis, photosynthesis, glycolysis, respiration, membrane synthesis and stability, enzyme activation/inactivation, redox reactions, signalling, carbohydrate metabolism and nitrogen fixation (Abel *et al.*, 2002; Vance *et al.*, 2003). Meanwhile, P deficiency is considered as one of the greatest limitations in agricultural production (Schachtman *et al.*, 1998; Lynch and Brown, 2008). It has been estimated that 5·7 billion hectares of land worldwide are deficient in P. Concentrations of phosphate in soil solutions are generally <10 μM, which are well below the critical level that is needed for the optimal performance of crops (Batjes, 1997). This problem of P deficiency might be mitigated by the application of concentrated fertilizers that provide soluble Pi for plants. The practice, however, is inherently inefficient due to chemical immobilization of P and agricultural run-off (Abel *et al.*, 2002). To overcome low P availability, plants have evolved a complex array of tightly controlled adaptive mechanisms for maintaining P homeostasis. One of the main mechanisms is to maximize the ability of the root to absorb P from the soil. The alteration of root architecture is a powerful vehicle for the development of crop plants with an efficient P acquisition ability.

Root architecture, the spatial configuration of a root system in the soil, has been shown to be important for plant P acquisition (Lynch, 1995). Root architecture is highly plastic in its developmental response to low-P conditions. A number of research papers and reviews have shown that genotypic adaptations to P deficiency allow the changes in root architecture that facilitate P acquisition (Gahoonia *et al.*, 2001; Lynch and Brown, 2001; Williamson *et al.*, 2001; López-Bucio *et al.*, 2002; Hermans *et al.*, 2006; Liu *et al.*, 2008; Lynch and

Brown, 2008; Vance, 2008; Richardson *et al.*, 2009; Wang *et al.*, 2009; Ramaekers *et al.*, 2010; Rouached *et al.*, 2010; Chiou and Lin, 2011; Péret *et al.*, 2011). The plant *Arabidopsis thaliana*, particularly the Columbia (Col-0) ecotype, has been shown to be a useful model to dissect the alteration of root morphology and architecture triggered by P deficiency (Williamson *et al.*, 2001; Sánchez-Calderón *et al.*, 2005). Low P availability in arabidopsis promotes the development of a highly branched root system to the detriment of the primary root, characterized by the stimulated formation and emergence of lateral roots and root hairs (Bates and Lynch, 1996; Williamson *et al.*, 2001; Linkohr *et al.*, 2002; López-Bucio *et al.*, 2002; Pérez-Torres *et al.*, 2008; Péret *et al.*, 2011). In other native and cultivated plants, including maize (*Zea mays*), rice (*Oryza sativa*), common bean (*Phaseolus vulgaris*), white lupin (*Lupinus albus*), tomato (*Solanum lycopersicum*) and *Brassica nigra*, low P availability also modifies root architecture traits such as primary root length, root branching, number and length of lateral roots and enhancement of root hair and cluster root formation (Dinkelaker *et al.*, 1995; Carswell *et al.*, 1996; Borch *et al.*, 1999; Kim *et al.*, 2008; Lambers *et al.*, 2011; Jin *et al.*, 2012).

Root architecture is a highly plastic trait, and varies among species, and its plasticity is strongly controlled by plant regulators and inherent genetic factors. It has been known that alteration in root architecture under low P is linked with changes in phytohormone composition and concentration, and involves expression of a number of genes. A suite of studies have implicated that the localized concentration, transport of and/or sensitivity to sugars and hormonal signals play a significant role in root development during low P supply (Nacry *et al.*, 2005; Jiang *et al.*, 2007; Karthikeyan *et al.*, 2007; Zhou *et al.*, 2008*a*; Rubio *et al.*, 2009; Santos-Beneit *et al.*, 2009; B.L. Wang *et al.*, 2010; Chiou and Lin, 2011; Péret *et al.*, 2011). In particular, sugars, auxin and ethylene have well-documented roles in modulating root architecture during P deficiency.

Genetic studies have identified a series of genes and a class of transcriptional regulators that mediate alteration of root architecture in response to low P in various plant species. There are increasing numbers of publications focusing on the need to improve plant P-uptake efficiency and crop yield, and for better management of P fertilizer use (e.g. Abel *et al.*, 2002; Lynch, 2007; Rubio *et al.*, 2009; Ramaekers *et al.*, 2010; Chiou and Lin, 2011; Hammond and White, 2011; Péret *et al.*, 2011; Jin *et al.*, 2012; Veneklaas *et al.*, 2012). However, these publications do not specifically consider the systemic strategies by which plants adjust their root structure and morphology in response to low-P conditions. This review provides a framework for understanding changes in root architecture in response to P limitation with a focus on integrated physiological and molecular signals.

THE INFLUENCE OF LOW P AVAILABILITY ON ROOT ARCHITECTURE

Root architecture includes the shape and structure of the root system (Hodge *et al.*, 2009). The shape of the root system refers to rooting depth, elongation and density of lateral roots and root hairs, the spatial location of roots and the way in which the root system occupies the soil. Root structure defines the variety of the components constituting the root system and their relationship (Hodge *et al.*, 2009). Two recent reviews on the root development and structure of monocots and dicots have been provided by Lynch (2007) and Ramaekers *et al.* (2010). The root architecture of monocots and dicots differs significantly, but the main adaptive root traits are common among all vascular plant species for the purpose of enhancing P acquisition. Therefore, in this review, a comprehensive 'root architecture' including root system shape and its structure has been used to summarize the multiplicity of root parameters. The majority of root architecture research in response to P has focused on the species *Arabidopsis thaliana* (arabidopsis) and white lupin; thus, these will henceforth be used as models for genetic purposes in this review.

Root distribution presented a strong positive relationship with P distribution that is most strongly influenced by soil tillage, rhizosphere pH, fertilizer management and cultivation time (Holanda *et al.*, 1998; Andraski and Bundy, 2003; Vu *et al.*, 2009). Although root architecture may play an important role in a no-till system, and P stratification was characterized in no-till systems as a result of a surface-applied fertilizer without incorporation, it is known that root architecture presented a strong positive relationship with P distribution. Meanwhile, the chemical nature of soil P is also affected by tillage practice, with P solubility being increased under conservation tillage (Zibilske and Bradford, 2003). Furthermore, tillage practices that mix the topsoil would also mix the previously applied P, which significantly affects building of the root architecture (Bolland and Brennan, 2006). Therefore, it appears that the effect of P distribution on the root architecture exists in both no-tillage and tillage systems. In addition, rhizosphere pH and fertilizer management also play an important role in root and P distribution in the soil profile. Jing *et al.* (2010) reported that localized supply of superphosphate plus ammonium sulfate stimulated root proliferation, acidified the rhizosphere, increased mobilization and uptake of P and enhanced maize growth in a calcareous soil in the field. Consequently, in the practice of spatial deployment of the root system response to P in soil, the following factors should be considered: the soil tillage, rhizosphere pH, water status, fertilizer management system and cultivation time, as all these strongly relate to root distribution.

Primary root development in response to low P

The adaptive response of primary root growth to low P shows species and genotypic variations. A significant P deficiency-induced root response is a reduction in primary root growth in plants such as arabidopsis (Williamson *et al.*, 2001; Linkohr *et al.*, 2002; López-Bucio *et al.*, 2002, 2003; Jain *et al.*, 2007; Pérez-Torres *et al.*, 2008; Tyburski *et al.*, 2010, 2012). The length from the root tip to the first lateral root or root hair was found to be significantly shorter in plants grown under low-P than under high-P conditions (Williamson *et al.*, 2001). Among 73 arabidopsis ecotypes, half showed reduced growth of primary roots at low P supply, and a quarter of the tested ecotypes were not responsive to P availability (Chevalier *et al.*, 2003), suggesting that

root growth inhibition is genetically determined rather than only metabolically controlled. For the last 25 % of accessions, low P was found to influence either the primary roots or lateral roots, indicating the occurrence of different adaptive strategies. Later, the identification of quantitative trait loci (QTLs) controlling the root growth response to low P further demonstrated the existence of such a control (Reymond *et al.*, 2006). Unlike arabidopsis, many other plant species displayed longer roots under low-P conditions. Alterations in primary root elongation are typical phenomena in response to P deprivation in rice (*O. sativa*) (Wissuwa, 2003; Shimizu *et al.*, 2004; Yi *et al.*, 2005). Similarly, there is no reduction or a slight enhancement in root elongation of maize (*Z. mays* L.) under low-P conditions (Mollier and Pellerin, 1999). Further, a survey of 14 dicots and monocots in solution culture showed that all the species tested had the same degree of primary root elongation under low and high P supply (Narayanan and Reddy, 1982).

A primary root is the first root to emerge in both dicots and monocots, and is derived from the embryonically formed meristematic tissue. When a primary root encounters a low-P medium, its growth is restrained. It is shown that reduced growth of the primary roots of P-deficient plants correlates with the reduction of cell differentiation within the primary root meristem and the inhibition of cell proliferation in the root elongation zone (Ticconi *et al.*, 2004; Sánchez-Calderón *et al.*, 2005, 2006; Svistoonoff *et al.*, 2007). It was observed that in low-P media, the number of cells in the elongation and meristematic regions of wild-type roots decreased >90 % when compared with the same regions in high-P plants (Sánchez-Calderón *et al.*, 2006). Ma *et al.* (2003) showed that in the arabidopsis primary root, the length of meristems was not affected by P deficiency, but the length of the rapid elongation zone was decreased. However, a reduction in cell length in the root tip of plants grown in low-P media has been reported previously in arabidopsis (Williamson *et al.*, 2001). This response is fast (within 2 d) and requires a physical contact between the root tip and the low-P medium (Linkohr *et al.*, 2002; Svistoonoff *et al.*, 2007); this is associated with exhaustion of the primary root meristem (Sánchez-Calderón *et al.*, 2005). However, root meristem exhaustion under low P availability is not a direct consequence of P deficiency, but rather is under a genetic programme that controls root growth (Sánchez-Calderón *et al.*, 2006). Kinetic studies of expression of the cell cycle marker *CycB1, 1::uidA* and the quiescent centre (QC) identity marker *QC46:GUS* showed that the reduction in cell proliferation in the primary root was preceded by alterations in the QC under low P supply (Sánchez-Calderón *et al.*, 2005). Overall, these results suggest that low P availability induced arabidopsis roots to enter a determinate developmental programme in which cell division is arrested and cell differentiation is promoted.

The reduced growth of the primary root results in a shallow root system, which exploits topsoil resources efficiently, and may be advantageous in low-P soils. However, this may inadvertently result in reduced water acquisition since the topsoil is often dried out and water availability typically increases with soil depth in a terminal drought environment. It was pointed out that plant genotypes selected for adaptation to low P soils might be more sensitive to drought (Ho *et al.*, 2004; Lynch and Ho, 2005). The optimization model from Ho *et al.* (2004) indicated that the marginal rate of substitution of water for P is the primary determining factor for predicting the distribution of roots in the soil. However, Beebe *et al.* (2008) showed that drought-resistant lines had superior yields in a low-P environment; one line even outperformed the low-P-tolerant control by a 41 % yield increase. This indicates that yield potential under drought stress and under low-P conditions may not be mutually exclusive.

Development- and hormone-related genes have been reported in arabidopsis as mediators of changes in root architecture in response to P deficiency (Table 1). Arabidopsis mutants defective in *LOW PHOSPHATE ROOT1* (*LPR1*) and its close paralogue *LOW PHOSPHATE ROOT2* (*LPR2*) strongly weaken growth inhibition of the primary root under P-limited conditions (<50 μM) (Svistoonoff *et al.*, 2007). The results of Svistoonoff *et al.* (2007) strongly indicated that the root cap is the site of the sensing and/or response to low concentrations of exogenous P. Furthermore, *LPR1* and *LPR2* encode multicopper oxidases (MCOs) that may play a role in plant root development in response to low-P conditions. A recent study revealed that *PHOSPHORUS STARVATION-INSENSITIVE* (*PSI*) was a new allele of *LPR1* and that an AC-repeat element in the promoter played an important role in controlling the expression of *LPR1* (X.M. Wang *et al.*, 2010). It is known that *LPR1* proteins in the root cap can influence the activity and distribution of a hormone-like compound (Svistoonoff *et al.*, 2007). Likewise, the *psi* mutation shows less sensitivity to auxin than the wild type, and enhances the ability to maintain the auxin response in the root tip under low P supply (X.M. Wang *et al.*, 2010). *SIZ1* negatively regulates P starvation-induced inhibition of primary root elongation through the control of auxin patterning in arabidopsis (Miura *et al.*, 2011). These results implied that auxin participates in the adaptation of primary root growth under low-P stress, but the mechanism underlying this process remains to be elucidated.

SCARECROW (*SCR*) and *SHORT-ROOT* (*SHR*) are members of the GRAS transcription factor (TF) family and key regulators of radial root patterning. *PHOSPHATE DEFICIENCY RESPONSE 2* (*PDR2*) is required for maintaining nuclear SCR protein under low-P conditions, which would reduce root meristematic activity (Ticconi *et al.*, 2004, 2009). The hypersensitivity to low P of the *pdr2* mutant (Ticconi *et al.*, 2004) appears as an exacerbation of such a response. In addition, the root meristem exhaustion probably perturbs auxin circulation in the root tip (Kramer and Bennett, 2006), and consequently impacts on branching in older parts of the root. Interestingly, *PDR2* and *LPR1* function in opposite ways in regulating the response of root growth to P deficiency in the endoplasmic reticulum (Ticconi *et al.*, 2004). The *siz1* mutant seedlings are also hypersensitive to low P, displaying a reduced growth of the primary root (Miura *et al.*, 2005). However, *SIZ1* may represent the element linking the low-P stress and reactive oxygen species (ROS) (Desnos, 2008). Whether *SIZ1* has a role in regulating P-deficiency-induced primary root growth via the ROS pathway is unknown.

Compared with the wild type and single knockout mutants, the elongation of primary roots of *PLDζ1* and *PLDζ2* (*PHOSPHOLIPASE D*) double knockout mutants was slower

TABLE 1. *Overview of genes and their functions, and related mutants with altered root architecture in response to low P*

Species	Gene name	Gene function during P starvation	Related phenotype grown in low P	References
Primary root				
Arabidopsis	*LPI1*, *LPI2*	Pi starvation signalling	Longer primary root	Sánchez-Calderón *et al.* (2006)
Arabidopsis	*PDR2*	Pi starvation signalling	Shorter primary root	Ticconi *et al.* (2004)
Arabidopsis	*PLDζ1*	Pi starvation signalling	Retarded primary root growth	Li *et al.* (2006)
Arabidopsis	*PLDζ2*	Phospholipases	Retarded primary root growth	Li *et al.* (2006)
Arabidopsis	*over Pht1;5*	Pi starvation signalling	Shorter primary root	Nagarajan *et al.* (2011)
Arabidopsis	*PHR1*	Transcription factor, Pi starvation signalling	Slight reduction in the root-to-shoot ratio	Rubio *et al.* (2001); Bustos *et al.* (2010)
Arabidopsis	*LPR1*, *LPR2*	Hormone-like signalling	Primary root growth maintained	Svistoonoff *et al.* (2007)
Arabidopsis	*PLT1*/*PLT2*	Auxin signalling, meristem maintenance	Shorter primary root in double mutant	Aida *et al.* (2004)
Arabidopsis	*SIZ1*	SUMO E3 ligase; auxin signalling	Cessation of primary root growth	Miura *et al.* (2005, 2011)
Arabidopsis	*PSI*	Auxin signalling	Impaired inhibition of primary root	B.L. Wang *et al.* (2010)
Arabidopsis	*VTC1*	Ascorbate signalling	Shorter primary root	Tyburski *et al.* (2012)
Arabidopsis	*GIN2*	Sugar signalling	Shorter primary root	Karthikeyan *et al.* (2007)
Arabidopsis	*FRY1*	Phytochrome, light signal	Decrease in primary root elongation growth	Chen and Xiong (2010); Hirsch *et al.* (2011)
Rice	*over OsPHR2*	Pi starvation signalling	Enhanced induction rate of primary root	Zhou *et al.* (2008*b*)
Rice	*OsMYB2P-1*	Pi starvation signalling	Longer primary root	Dai *et al.* (2012)
Rice	*LTN1*	Pi starvation signalling	Enhanced elongation of primary root	Hu *et al.* (2011)
Lateral roots				
Arabidopsis	*LPR1*	Hormone-like signalling	Reduced lateral root development	López-Bucio *et al.* (2005)
Arabidopsis	*LPI*	Pi starvation signalling	Reduced lateral root number	Sánchez-Calderón *et al.* (2006)
Arabidopsis	*ARF7*	Auxin signalling	More lateral roots	Pérez-Torres *et al.* (2008)
Arabidopsis	*ARF19*	Auxin signalling	More lateral roots	Pérez-Torres *et al.* (2008)
Arabidopsis	*FRY1*	Phytochrome, light signal	Reduced lateral root initiation and elongation	Chen and Xiong (2010); Hirsch *et al.* (2011)
Arabidopsis	*SIZ1*	SUMO E3 ligase; auxin signalling	Promoted lateral root growth	Miura *et al.* (2005, 2011)
Arabidopsis	*PDR2*	Pi starvation signalling	Numerous, short, highly branched lateral roots	Ticconi *et al.* (2004)
Arabidopsis	*PLDζ1*/*PLDζ2*	Phospholipases	Longer lateral roots	Li *et al.* (2006)
Arabidopsis	*PLT1*; *1PLT1*; *4*	Phosphate transport	Faster lateral root growth	Shin *et al.* (2004)
Arabidopsis	*PNP*	Methylerythritol-dependent biosynthesis	Numerous, short, highly branched lateral roots	Marchive *et al.* (2009)
Arabidopsis	*GIN2*	Sugar signalling	Reduction in the number of lateral roots	Karthikeyan *et al.* (2007)
Arabidopsis	*WRKY 75RNAi*	Transcription factor	Promoted formation of lateral roots	Devaiah *et al.* (2007*a*)
Rice	*OsMYB2P-1*	Pi starvation signalling	Longer adventitious roots	Dai *et al.* (2012)
Rice	*over OsPHR2*	Pi starvation signalling	Enhanced induction rate of adventitious roots	Zhou *et al.* (2008*b*)
Rice	*LTN1*	Pi starvation signalling	Enhanced elongation of adventitious roots	Hu *et al.* (2011)
Root hairs				
Arabidopsis	*LPI1*, *LPI2*	Pi starvation signalling	Shorter root hair	Sánchez-Calderón *et al.* (2006)
Arabidopsis	*bHLH32*	Transcription factor	Increased root hair formation	Chen *et al.* (2007)
Arabidopsis	*over Pht1;5*	Pi starvation signalling	Increased root hair proliferation	Nagarajan *et al.* (2011)
Arabidopsis	*SIZ1*	SUMO E3 ligase; auxin signalling	Promoted root hair growth	Miura *et al.* (2005)
Arabidopsis	*FBX2*	Pi starvation signalling	Increased root hair formation	Chen *et al.* (2008)
Arabidopsis	*IPK*	Phytate synthesis	Constitutive production of root hairs	Stevenson-Paulik *et al.* (2005)
Arabidopsis	*UBP14*/*PER1*	Ubiquitin protease	Inhibition of root hair growth	Li *et al.* (2010)
Arabidopsis	*HSP2*	Ethylene signalling	Increased root hair formation	Lei *et al.* (2011*b*)
Arabidopsis	*GA1,3*	Gibberellic acid signalling	Shorter root hair	Jiang *et al.* (2007)
Arabidopsis	*RSI4*	bHLH transcription factor	Development of very short root hairs	Yi *et al.* (2010)
Arabidopsis	*WRKY 75RNAi*	Transcription factor	Constitutive production of root hairs	Devaiah *et al.* (2007*a*)
Arabidopsis	*PHR1*/*PHL1*	Pi starvation signalling	Inhibition in root hair growth	Bustos *et al.* (2010)
Arabidopsis	*PHR1*	Transcription factor, Pi starvation signalling	Increase in root hair length	Bustos *et al.* (2010)
Arabidopsis	*PHL1*	Pi starvation signalling	Sustains root hair length	Bustos *et al.* (2010)
White lupin	*GPX-PDE1RNAi*	Glycerophosphodiester phosphodiesterases	Reduction in root hair development	Cheng *et al.* (2011)
Cluster roots				
White lupin	*LaSAP1*	Secreted acid phosphatase	Upregulated in P-deficient cluster roots	Wasaki *et al.* (1999)
White lupin	*LaPT1*	High-affinity Pi transporter	Upregulated in P-deficient cluster roots	Liu *et al.* (2001)
White lupin	*LaMATE*	Multidrug and toxin efflux	Upregulated in P-deficient cluster roots	Uhde-Stone *et al.* (2005)
White lupin	*LaPEPC*	Phosphoenolpyruvate	Upregulated in P-deficient cluster roots	Uhde-Stone *et al.* (2003*a*)
White lupin	*LaGPX-PDE1*	Glycerophosphodiester phosphodiesterases	Highly expressed in P-deficient cluster roots	Cheng *et al.* (2011)
White lupin	*LaGPX-PDE2*	Glycerophosphodiester phosphodiesterases	Highly expressed in P-deficient cluster roots	Cheng *et al.* (2011)
White lupin	*CKX*	Cytokinin signalling	Upregulated in P-deficient cluster roots	Vance *et al.* (2003)

(Li *et al.*, 2006), indicating that either *PLDζ1* or *PLDζ2* can function to promote apical growth. However, only loss of *PLDζ2* results in lower concentrations of phosphatidic acid (PA) under low-P conditions. This raises the possibility that PA generated by *PLDζ* genes may promote root elongation under P deficiency. Another study indicates that PA interacts with *AtPDK1* (*PHOSPHOINOSITIDE-DEPENDENT PROTEIN KINASE 1*), stimulates a protein kinase cascade, and promotes root apical growth and initiation (Anthony *et al.*, 2004). It has been speculated that *AtPDK1* is the target for PA under low-P conditions.

The available data have indicated that both *LPI1* and *LPI2* regulate the growth of the primary root under P deprivation (Sánchez-Calderón *et al.*, 2006). Further *lpi* lines had neither a drastic reduction in cell length in the primary root nor a decreased cell number in the root elongation zone (Sánchez-Calderón *et al.*, 2006), suggesting that *LPI1* maintains an indeterminate root development programme under low P conditions. Notably, in addition to the insensitivity of growth inhibition of the primary root to P deprivation, the low-P-insensitive mutant *lpi* showed a reduction in the expression of genes involved in the P-starvation rescue system *PHOSPHATE TRANSPORTER 1* (*AtPT1*), *AtPT2*, *PURPLE ACID PHOSPHATASE 1* (*PAP1*), *ACID PHOSPHATASE 5* (*AtCP5*) and *INDUCED BY PHOSPHATE STARVATION 1* (*AtIPS1*) (Casamitjana-Martínez *et al.*, 2003). It has been speculated that the transcriptional or post-transcriptional regulation of *SHR*, *SCR*, *PLETHORA1* (*PLT1*), *PLETHORA2* (*PLT2*) and/or CLV3/ESR-like gene *CLE19* (*AtCLE19*) could be negatively regulated by *LPI1* and *LPI2*, depending on P availability (Sánchez-Calderón *et al.*, 2006). These results suggest that high sensitivity to P starvation plays a regulatory role in primary root growth.

Recent molecular studies in arabidopsis and rice (*O. sativa*) have identified several genes and TFs involved in P absorption, transport and remobilization, and regulation of primary root elongation under low P availability. Arabidopsis has a family of high-affinity Pi transporters which includes nine members, named *PHOSPHATE TRANSPORTER 1;1–9* (*PHT1;1–9*), accounting for 70 % of the total root P transport activity (Shin *et al.*, 2004). Phosphate transport double mutants, *plt1;1 plt1;4*, exhibit stunted plant growth, including slow growth of primary roots (Shin *et al.*, 2004). Recently, it was found that *PHT1;5* played a negative role in primary root growth in response to low P supply (Nagarajan *et al.*, 2011). *PHO2* was shown to be involved in phloem transport of P between shoots and roots or in regulating leaf P concentration (Dong *et al.*, 1998). In rice, *LTN1* (the homologue of arabidopsis *PHO2*, *LOC_Os05g48390*) can alter P-deficiency-dependent root morphology including the promoted elongation of the primary and adventitious roots in *ltn1* mutants (Hu *et al.*, 2011). This implies that *LTN1* has a negative role in primary root development in response to P deficiency. *ZAT6*, a Cys-2/His-2 zinc finger TF, also negatively regulates primary root growth, but the regulation is independent of the P status in arabidopsis (Devaiah *et al.*, 2007*b*). *PHOSPHATE STARVATION RESPONSE 1* (*AtPHR1*), a MYB TF, activates a sub-set of P starvation-induced genes, and *phr1* showed a reduced response of *AtIPS1*::*GUS* to P starvation (Rubio *et al.*, 2001). Zhou *et al.* (2008*b*) found that *OsPHR2*, the homologue of *AtPHR1*, was a key regulator for P starvation signalling in rice, and *OsMYB2P-1*-over-expressing arabidopsis displayed an enhanced induction rate of primary roots. *OsMYB2P-1*, an R2R3 MYB TF, was identified from microarray data by monitoring the expression profile of rice (*O. sativa* ssp. japonica) seedlings exposed to a P-deficient medium (Dai *et al.*, 2012). Compared with *OsMYB2P-1*-over-expressing rice grown in P-deficient media, the reduced expression of *OsMYB2P-1* in wild-type rice led to suppression of the growth rate of primary roots. Similarly, arabidopsis plants over-expressing *OsMYB2P-1* exhibited longer primary roots and more lateral roots than wild-type arabidopsis plants (Dai *et al.*, 2012). These results suggest that *OsMYB2* plays a positive regulatory role in the modulation of primary root elongation in response to P deficiency.

Lateral root development in response to low P

Lateral roots play an important role in P acquisition by increasing soil exploration (Zhu *et al.*, 2005*a*), the absorptive surface of the root system (Pérez-Torres *et al.*, 2008) and P solubilization (Lynch, 1995, 2007). Many dicotyledonous plants, such as arabidopsis, cotton (*Gossypium* spp.) and soybean (*Glycine max*), generate several orders of lateral roots resulting from repeated branching of a primary root, whereas the root systems of cereal crops, such as rice (*O. sativa*) and maize (*Z. mays*), are principally composed of adventitious roots (Hochholdinger *et al.*, 2004; Osmont *et al.*, 2007). Phosphorus supply affects the growth and proliferation of lateral roots. Studies have shown that low P favours lateral root growth by reducing primary root elongation and increasing lateral root elongation and density in arabidopsis (Williamson *et al.*, 2001; Linkohr *et al.*, 2002; Reymond *et al.*, 2006). In common bean, P availability regulates adventitious rooting (Borch *et al.*, 1999; Miller *et al.*, 2003), and two major QTLs accounted for 19–61 % of the total phenotypic variation for adventitious root traits under low-P conditions (Lynch *et al.*, 2006).

Under normal conditions, lateral roots originate from a small number of pericycle cells that are adjacent to the xylem pole and initiate a series of asymmetric and transverse divisions (Torrey, 1950). The organization of cells in lateral roots is analogous to that in primary roots, but the numbers of cell files in each layer are more variable (Dolan *et al.*, 1993). Early development of lateral roots, which may recapitulate embryonic and primary root formation, can be separated into five major stages: initiation, establishment and emergence of lateral root primordia, followed by meristem activation and maintenance (Malamy and Benfey, 1997). The pattern of lateral root formation close to the root tip under P deficiency is comparable with that of roots with damaged primary root meristemic activity, including destruction of meristemic activity by cell ablation or physical decapitation of the primary root meristem, eliciting an increase in lateral root number (Torrey, 1950; López-Bucio *et al.*, 2002). It has been suggested that low-P stress not only reduces the rate of cell division but also inhibits cell growth in the root elongation zone (Sánchez-Calderón *et al.*, 2006). Under low P concentrations, the mitotic activity is relocated to the sites of lateral root formation, which leads to increased lateral root density (Tyburski *et al.*, 2012). Similar to the primary root tip, the manner of cell differentiation in older lateral roots occurs

within the root apical meristem (RAM), which is followed by an inhibition of lateral root elongation (Nacry *et al.*, 2005; Sánchez-Calderón *et al.*, 2005). As mentioned above, the reduced growth of primary roots correlates with a decrease in the number of cells in the elongation zone and a corresponding decrease in the distance between the root tip and the first lateral root (Williamson *et al.*, 2001). Therefore, as for the whole root system, lateral growth is favoured over elongation of the primary axis at the cellular level. Interestingly, each lateral root can produce more lateral roots, and a complex root system is constructed by the reiteration of a single developmental process.

The response of lateral roots to P deficiency, however, shows species and genotypic variations. In maize, some genotypes show an increase in the number and length of lateral roots while others show the opposite effect. In this crop, the mature root system is mainly composed of nodal roots, so the maintenance of nodal root formation could result in an increased proportion of root length in the adventitious root system when the overall growth is inhibited by P deficiency (Bayuelo-Jiménez *et al.*, 2011). Maize plants have a fibrous root system that is generally more deeply distributed than the basal roots of the tap-root system, such as in common bean and soybean. In maize–soybean intercropping, soybean genotypes with shallower roots might be able to take up P from topsoil, and could avoid competition for sub-soil P with maize (Tang *et al.*, 2005). Similar results have been observed in adventitious roots of common bean genotypes. Consequently, P-induced adventitious rooting could vary widely among common bean genotypes (Miller *et al.*, 2003), and this trait is moderately heritable (Lynch *et al.*, 2006). Architectural traits associated with promoted topsoil foraging in common bean are shallower basal roots, increased adventitious rooting and greater dispersion of lateral branching from the basal roots (Lynch, 2007; Ramaekers *et al.*, 2010). These imply that plants grown with a low P supply change the angle of basal roots in favour of outward rather than downward growth, resulting in a shallower and broader root system. This point was supported by the observed correlation between the ability of bean cultivars to reduce root angle in low-P and yield in P-poor soils (Bonser *et al.*, 1996). As mentioned above, a shallow root system exploits topsoil resources efficiently, which is advantageous in low-P soils. However, this may inadvertently lead to reduced water uptake. Indeed, drought tolerance in common bean has been associated with rooting depth (Sponchiado *et al.*, 1989; Sanders and Markhart, 1992).

Genetic analyses have identified a suit of genes that control lateral root development under low P supply (Table 1). Three low-P-resistant root lines (*lpr1-1*, *lpr1-2* and *lpr1-3*) of arabidopsis were reported to have reduced formation of lateral roots in low-P conditions (López-Bucio *et al.*, 2005). Svistoonoff *et al.* (2007) verified that *LPR1* and *LPR2* in arabidopsis lines were strongly induced by P starvation, and that this induction was closely related to elongation of primary and lateral roots. The phosphate transport double mutant *plt1; 1plt1; 4* showed faster lateral root growth but slower primary root growth than wild-type plants under low P availability (Shin *et al.*, 2004). This supports the notion that the response to low P could be different in primary roots as compared with lateral roots. Also, *PLDζ* genes have been suggested to be involved in these different responses to low P between primary and lateral roots because these genes promote primary root elongation but inhibit lateral root elongation under low-P conditions (Li *et al.*, 2006).

The typical increase in the number of lateral roots under low-P conditions was observed in the low-P-insensitive mutant *lpi1* but not in *lpi2* (Sánchez-Calderón *et al.*, 2006), implying that *LPI2* regulates lateral root formation whereas *LPI1* is not involved in lateral root induction by P deprivation. Genetic and molecular analyses have revealed that all *lpr1* mutants are allelic to *BIG*, which is required for normal auxin transport in arabidopsis (López-Bucio *et al.*, 2005). This provides genetic evidence that lateral root formation is not a direct consequence of the observed meristem exhaustion in the low-P response but could be mediated by an independent alteration of auxin sensitivity of pericycle cells during P deprivation. The *PLEIOTROPIC DRUG RESISTANCE 2* (*pdr2*) mutant displays hypersensitive responses to P deficiency, showing higher density of lateral root meristems under the stress (Ticconi *et al.*, 2004). It indicates that *PDR2* is necessary for maintenance of root meristem function when external P supply is limited. The *pdr2* phenotype in low P is strikingly similar to the auxin-conditional *ABERRANT LATERAL ROOT FORMATION 3* (*alf3*) root phenotype (Celenza *et al.*, 1995). The *alf3* mutation causes primary root growth arrest and death, followed by increased formation of lateral root primordia that fail to mature and subsequently die. As auxin fails to rescue or mimic the *pdr2* root phenotype, and auxin sensitivity of primary root growth is not altered in *phr2* seedlings, it is unlikely that auxin is directly involved in the response of root meristems to low external P availability. Another study has shown that a class of transcriptional regulators mediating growth and developmental responses to auxins [*AUXIN RESPONSE FACTOR 7* (*ARF 7*) and *AUXIN RESPONSE FACTOR 19* (*ARF 19*)] are required for lateral root formation under P starvation and that this response involves an SCFTIR1 [a ubiquitin-protein ligase (E3)]-dependent signalling mechanism (Pérez-Torres *et al.*, 2008). These reveal that an auxin response signal plays a key role in the adaptation of lateral root formation under P deficiency.

Polynucleotide phosphorylase (PNPase) is involved in P sensing and signalling, as suggested by the constitutive expression of P-responsive genes in their corresponding mutants (Ticconi *et al.*, 2004; Marchive *et al.*, 2009). The *pnp* mutant produces numerous, short and highly branched lateral roots under low-P conditions. *SIZ1* (encodes an arabidopsis SUMO E3 ligase) has been found to inhibit lateral root formation at low P, as demonstrated by the increased lateral root response of the *siz1* mutant (Miura *et al.*, 2011). X.M. Wang *et al.* (2010) suggest that *LPR1* functions independently of the *PHR1/SIZ1* signalling pathway to regulate root growth under P starvation in arabidopsis.

Transcriptome analysis reveals several TFs involved in lateral root response to P deficiency. *WRKY DNA-BINDING PROTEIN 75* (*WRKY75*), a member of the WRKY TFs, is up-regulated during P deficiency and negatively regulates lateral root growth which is independent of P status in plants (Devaiah *et al.*, 2007*a*). In contrast, the *OsPHR2*-over-expressing plants showed a higher sensitivity to P deficiency with an enhanced induction rate of adventitious roots than wild-type plants (Zhou *et al.*, 2008*b*). *ZAT6*, a Cys-2/His-2 zinc finger TF, positively

regulates lateral root growth, and this regulation is independent of the P status in plants (Devaiah *et al.*, 2007*b*). Arabidopsis plants over-expressing *OsMYB2P-1* exhibited more lateral roots than wild-type arabidopsis plants (Dai *et al.*, 2012). These results suggest that *MYB2* plays a positive regulatory role in lateral root development during P deficiency.

Development of root hairs in response to low P

It has been known for some time that low P causes increased extension of root hairs in many plant species (Foehse and Jungk, 1983). Among the root traits, increased root hair proliferation is an early response of plants to low P (Ma *et al.*, 2001, 2003; Jain *et al.*, 2007). In addition, the root hair/P response is a localized cellular response and is specific for P (Bates and Lynch, 1996). At a P concentration of 1 μM, representative of many low-P soils, the root hair length of arabidopsis can exceed 1 mm. Yet at a P concentration of 1000 μM, root hair length is decreased to 0·3 mm, and it is suppressed completely at 3000 μM P (Bates and Lynch, 2000*a*). However, high P supply does not completely suppress root hair growth, suggesting that high-P plants maintain the potential for plasticity. Bates and Lynch (2000*a*) pointed out that low P increased root hair length by increasing both the rate and duration of hair elongation, and suggested that P deficiency accelerated and prolonged metabolic activity in elongating hairs. Overall, the increase in the root hair density and length in response to P deficiency is a general and well-researched phenomenon in plant biology (Péret *et al.*, 2011).

The plasticity response of root hairs is relatively faster than root growth and branching, thus root hair number has been used as a measure of root response to low P in some specific plants. For example, genetic variation in root hair length and plasticity was suggested to be an appropriate target for marker-aided selection to improve the P efficiency in maize (Zhu *et al.*, 2005*b*). The presence of root hairs in maize, particularly dense root hairs of the main axis and nodal first-order laterals, was associated with plant performance at low P (Bayuelo-Jiménez *et al.*, 2011). In addition, root hairs can grow to terminal length, change the effective root radius and absorb available P prior to the increase in root surface area by lateral root branching. Therefore, root hair growth may represent the earliest morphological response to changing environmental conditions.

Interestingly, a maize mutant defective in root hair growth shows normal growth and development under field conditions (Wen and Schnable, 1994). This raises a question about the importance of root hairs in P acquisition under field conditions. Later experiments using root hair mutants showed that root hairs facilitate P uptake by increasing the absorptive surface area of the root (Bates and Lynch, 2000*b*). Genetic variation in root hair length and density in maize is controlled by several major QTLs (Zhu *et al.*, 2005*b*), suggesting that this trait could be selected in breeding programmes through marker-assisted selection. This offers the possibility of quick screening of a large number of maize accessions based on variation in root hairs (Lynch and Brown, 2008) and further dissection of genetic control of root hair formation. Importantly, the effect of P deficiency on root hair development should be investigated in soil experiments where P cannot easily diffuse to the root surface.

Root hairs are long tubular outgrowths of specialized root epidermal cells that play a critical role in water and nutrient uptake from soils (Peterson and Farquhar, 1996). Each root hair is derived from a single epidermal cell, and only certain epidermal cells termed 'trichoblasts' actually develop into root hair-bearing cells. Trichoblasts are smaller and more densely cytoplasmic cells than the surrounding hairless epidermal cells (Leavitt, 1904). Root hair distribution can be grouped into three categories in angiosperms (Datta *et al.*, 2011). In type I, all epidermal cells are morphologically identical before hair initiation, and any cell in the epidermis can possibly develop into a hair cell. Type I appears to be the most widespread pattern in plants, including tomato and lettuce (Pemberton *et al.*, 2001), and can be significantly affected by environmental conditions (Tsai *et al.*, 2004). In type II, a root hair develops from two smaller cells produced by asymmetric cell division of an epidermal cell in the meristematic zone, and the larger cell remains hairless. For type III, occurring in most members of the Brassicaceae family, including arabidopsis, the hair cells occur in files separated by 1–3 files of non-hair cells.

Root hair development in plants can be divided into three phases: cell specification, initiation and elongation (Schiefelbein, 2000). These processes are of crucial importance for building the structure of the plant body, and developing and maintaining its interface with the environment. It has been reported that the increase in root hair density in plants grown with low P can be explained by alterations in trichoblast file number, trichoblast length and/or percentage of trichoblast cells forming hairs (Ma *et al.*, 2001; Zhang *et al.*, 2003). Phosphorus-deficient plants of arabidopsis had smaller but more cortical cells, and hence a larger number of root-hair-bearing epidermal cell files than P-adequate plants (Zhang *et al.*, 2003). Schmidt and Schikora (2001) found only a small number of ectopic hairs in arabidopsis plants after transfer from a very high-P (2·5 mM) to a no-P medium. These ectopic hairs might result from the stress of transfer, and are unlikely to contribute to the enhanced root hair density under low P.

Numerous experimental observations indicate that root hair development involves different cellular and genetic processes (Schiefelbein, 2000; Foreman and Dolan, 2001). During the past years, there has been a surge in research activity in root hair development, particularly in arabidopsis. Grierson *et al.* (2001) reported that >40 genes in arabidopsis affect root hair initiation and development, and most of these genes might be responsive to P deficiency. Two extensive reviews on the root hair development genes and cell patterning information have been provided by Griersona and Schiefelbein (2002) and Péret *et al.* (2011). Our review here provides further insight into the signalling pathways based on isolation of genes affected in low-P-insensitive mutants that regulate changes in root hair growth in response to low P availability (Table 1).

Several TFs have been shown to control root hair growth during P deficiency. For instance, low P strongly affected root hair length in the *phr1* and *phr1ph1* double mutants of arabidopsis, whereas it did not change root hair length in the *phl1* mutant (Bustos *et al.*, 2010). These authors concluded that this effect on root hair length reflected a lack of correct protection against the stress inherent in P deficiency, and that increased *PHR1* activity increased reproductive success in

these stress conditions. In contrast, a mutation in the *PHR1* regulator *SIZ1* enhanced the sensitivity of arabidopsis plants to P deficiency, resulting in a greater number of root hairs (Miura *et al.*, 2011). These indicate that transcriptional repression responses are an integral part of adaptive responses to the stress. Except for the *PHR*-like gene, the two major arabidopsis transporters involved in P uptake are the *PHOSPHATE TRANSPORTER1* (*Pht1*) family members, *Pht1;1* and *Pht1;4* (Shin *et al.*, 2004). Both of these transporters are localized in the root epidermis (Karthikeyan *et al.*, 2002; Mudge *et al.*, 2002), an appropriate site for P perception. Irrespective of the P regime, the *Pht1;5* over-expressors showed significant increases in both the number and length of root hairs compared with the wild type (Nagarajan *et al.*, 2011). Moreover, low P controls root hair length by modulating steady-state levels of *ROOT HAIR DEFECTIVE 6-LIKE4* (*RSL4*) transcript and protein, which requires the phosphate signalling activity of *LPR1* and *LPR2* (Yi *et al.*, 2010), implying that *LPR1* interacts with downstream components of the P-sensing pathway. Recently, two glycerophosphodiester phosphodiesterase genes (*GPX-PDE1* and *GPX-PDE2*) were characterized from white lupin (Cheng *et al.*, 2011). These two genes were highly expressed in root hairs, epidermal cells and vascular bundles, particularly in P-deficient plants. All of these results suggest that genes involved in P metabolism such as secondary metabolism, P scavenging and remobilization, plant hormone metabolism, and signal transduction play a role in modulating low-P-induced root hair development. However, the double knockouts of *PLDζ1* and *PLDζ2* in arabidopsis affect root elongation but do not affect root hair patterning under P deficiency (Li *et al.*, 2006). Thereby, the relationship between genes involved in the pathway of P response and metabolism and the early phase of root epidermal cell specification should be considered in future research.

Cluster root formation in response to low P

Cluster roots, specialized tertiary lateral root structure, are densely clustered secondary roots with determinant growth and are a feature of the Proteaceae and several other plant species (Johnson *et al.*, 1996; Watt and Evans, 1999; Skene, 2000; Shane and Lambers, 2005; Shu *et al.*, 2005; Lambers *et al.*, 2006). Recent research has revealed clearly that the morphological and physiological features of cluster roots (such as release of protons and carboxylates) play a key role in the high P efficiency (Shane *et al.*, 2003; Shen *et al.*, 2003; Cheng *et al.*, 2011; Lambers *et al.*, 2011). Several reviews have covered this aspect (e.g. Hinsinger *et al.*, 2003; Lambers *et al.*, 2006). It appears that cluster root formation may be an alternative strategy to arbuscular mycorrhiza (AM) formation, as in most Proteaceae and also white lupin. Also, some plants, such as *Casuarina*, develop both cluster roots and AM (Lambers *et al.*, 2008). This review will not discuss the roles of AM in P acquisition because many excellent publications have covered various aspects of AM (e.g. Jasper *et al.*, 1979; Bolan, 1991; Brundrett, 2002, 2004, 2009; Lynch, 2007; Smith and Smith, 2011; Smith *et al.*, 2011).

White lupin is the tool of choice to improve our understanding on how P nutrition affects cluster root formation and development (Liu *et al.*, 2005). Increased formation of cluster roots is the earliest response to P deficiency in seedlings of white lupin grown in a hydroponic culture system without P supply (Neumann *et al.*, 2000). This rapid development of cluster roots within several days could be an advantage, particularly in extreme environments.

Formation of cluster roots appears to be mainly induced by P deficiency. However, it is argued whether external P (rooting media) or internal P concentration (in shoot or root) controls the formation of cluster roots. Some studies supported the idea that availability of external P in soil patches was important in enhancing cluster root formation (Shu *et al.*, 2007). However, it was also suggested that shoot P concentrations regulated the initiation, growth and functioning of cluster roots. Suppression of cluster root formation by foliar P application (Marschner *et al.*, 1987) and high internal P concentration (Keerthisinghe *et al.*, 1998; Shane *et al.*, 2003; Pearse *et al.*, 2006) suggests that induction is determined by the internal P concentration rather than by P levels of the substrata. Later, Li *et al.* (2008) and Zhou *et al.* (2008*a*) demonstrated that the increased cluster root formation of white lupin was particularly regulated by the internal P concentration of the shoots, rather than by the P supply in the root medium or total P concentration in the roots. However, it was proposed that enhanced P uptake by cluster roots in turn had a negative effect on cluster root formation and root exudation (Keerthisinghe *et al.*, 1998; Shen *et al.*, 2003). These results indicate that shoot-derived signals transmit the message of P deficiency to stimulate cluster root formation and that adequate P supply to plants inhibits cluster root formation to avoid excessive loss of carbon from the root system.

Interestingly, a previous study using a split-root system of white lupin showed that local P supply had an additional effect on the formation of cluster roots, and that this cluster root formation depended on an interaction between local P supply and shoot P concentration (Shane *et al.*, 2003). Importantly, if a high concentration of P in the shoot is a signal for prevention of cluster root formation, different species with inherent differences in maximum growth rate and P uptake may differ in the foliar P concentration that either enhances or suppresses cluster root formation (Abdolzadeh *et al.*, 2010). In conclusion, on the one hand, shoot P concentration is responsible for formation of the cluster root in P-deficient plants. On the other hand, increased P concentration in shoots resulted from cluster root formation, which is partly due to stimulation by external P availability in soil.

The supply of other nutrients such as nitrogen is involved in the development of cluster roots under low P supply. For example, low levels of N enhance P-deficiency-induced formation of cluster roots, whereas high N supply has inhibitory effects (Dinkelaker *et al.*, 1995). The omission of P increased cluster root formation only under low N supply (Sas *et al.*, 2002; Paungfoo-Lonhienne *et al.*, 2009). The large overlap of genes differentially expressed in cluster roots under N and P deprivation indicates that a large number of genes involved in cluster root formation are not specific to a particular nutrient stress (Rath *et al.*, 2010). In non-cluster-forming species, localized application of P combined with ammonium can significantly improve maize growth and nutrient use at the early stages by stimulating root proliferation and rhizosphere acidification (Jing *et al.*, 2010). Similarly, localized supply of nitrate

and P to acid sub-soil increases wheat root length and the number of root tips (Weligama *et al.*, 2008). It is therefore of interest to evaluate the interaction between rhizosphere pH and N availability during low P availability in influencing cluster root formation.

In the past decade, a number of P-limitation-induced genes have been described in cluster roots of white lupin. These include a high-affinity phosphate transporter (*LaPT1*; Liu *et al.*, 2001), a secreted acid phosphatase (*LaSAP1*; Wasaki *et al.*, 1999; Miller *et al.*, 2001), and a multidrug and toxin efflux gene (*LaMATE*; Uhde-Stone *et al.*, 2005). The promoters of these genes contain one or more P1BS elements, which have been well characterized as upregulated in P-deficient cluster roots (Liu *et al.*, 2001; Miller *et al.*, 2001; Uhde-Stone *et al.*, 2005). Apart from sugar signal genes, Uhde-Stone *et al.* (2003*a*) identified 35 expressed sequence tags (ESTs) or EST contigs showing enhanced expression of the corresponding genes in cluster roots of P-deficient white lupine. The role of the glycerophosphodiester phosphodiesterase gene (*GPX-PDE1* and *GPX-PDE2*) products encoded by two ESTs in a P deficiency-induced phospholipid degradation pathway in cluster roots was proposed (Cheng *et al.*, 2011). All these indicate that P metabolism, secondary metabolism and signal transduction govern interactions between P deficiency and cluster roots. However, the genetics of the low-P-induced cluster root modification have not been well developed, and many mutants specifically altered in this response need to be isolated.

HOW DOES LOW P AVAILABILITY REGULATE ROOT ARCHITECTURE DEVELOPMENT?

Accumulating evidence suggests that a suite of classical signals involved in the process of root architecture development could be induced by low P. The role of these signals in the adaptive response of root growth to low P is complex. Some of these signals regulate only a specific change, whereas others modulate multiple effects. The mechanisms mediating the response of root architecture to P deficiency have not been fully understood, and here the current knowledge is summarized.

Sugars

Genetic studies show that low P availability induces sugar-dependent systematic expression of genes, which can modulate the root system architecture. Interactions between the sugar signal and systematic P starvation on plant responses have been explicitly demonstrated in several studies. For example, sugars modulate the expression of P deficiency-induced genes in white lupin (Liu *et al.*, 2005; Tesfaye *et al.*, 2007; Zhou *et al.*, 2008*a*) and arabidopsis (Franco-Zorrilla *et al.*, 2005; Jain *et al.*, 2007; Karthikeyan *et al.*, 2007; Müller *et al.*, 2007; Hammond and White, 2008; Lei *et al.*, 2011*a*). Li *et al.* (2008) reported that differences in citrate secretion, sugar metabolism and root cell proliferation were the main reasons for high P-deficiency tolerance of an *L. albus* 'Kiev' mutant. The plant displayed a specialized P-efficient root system with a high capacity for mobilizing external P and increased cell division in the root meristem under P starvation (Li *et al.*, 2008). Meanwhile, Zhou *et al.* (2008*a*) showed that P starvation responses in white lupin roots were controlled by at least two sugar-signal-mediated regulation systems. One system regulates *LaPT1* and *LaPEPC3* gene expression and acts when P supply is low, and the other system regulates cluster root formation and *LaSAP* expression and acts even in P-sufficient medium if roots receive a sugar signal. This study suggests that the sugar signal interacts with other signals of signalling networks such as a local P-starvation signal or another systematic signal, or both. Therefore, it is probable that endogenously supplied sugar stimulated cluster root formation under low P as a signal, not as an energy source or a secondary effect.

In arabidopsis, exogenously supplied sugars increased lateral root density dependent on the P status in the rooting media, but did not affect tap-root length (Jain *et al.*, 2007; Karthikeyan *et al.*, 2007), implying that the sugar sensor participated in some aspects of the P-deficiency-response mechanism in lateral roots. The activity of the RAM has been proposed to determine the magnitude of P starvation responses. Increasing RAM activity by external sucrose application enhances the starvation responses, as reported by the expression of P starvation markers (Péret *et al.*, 2011). This means that not only meristematic activity but also sugar signalling determine root developmental responses to P starvation in arabidopsis. It is worth noting that the expression of the microbial *GPX-PDE* genes induced by P stress is regulated in part by sugar availability (Santos-Beneit *et al.*, 2009). Furthermore, a recent study has characterized these two genes (*GPX-PDE1* and *GPX-PDE2*) from white lupin, which are highly expressed in root hairs, epidermal cells and vascular bundles, particularly in P-deficient plants (Cheng *et al.*, 2011). However, whether sugars play a role in root hair development under low-P conditions is currently unclear.

It has been reported that sugars act as signalling molecules in plants to integrate environmental conditions and intrinsic developmental programmes modulated by multiple plant hormones. For example, sucrose is the main photosynthate translocated between shoot and root via the phloem, and a high root-to-shoot ratio of sucrose concentration and changes in concentrations of phytohormones such as cytokinins are required for P-starvation responses (Hammond and White, 2008). Furthermore, sucrose is proposed as a global regulator of a whole suite of P-starvation responses in arabidopsis (Lei *et al.*, 2011*a*), implying that the sugar signal may act upstream in the low-P-induced root development pathway.

Auxins

Growing evidence suggests that auxins act as signalling intermediates in multiple pathways, including those involved in the responses of root architecture to low P supply. This was first supported by a study showing that exogenous application of auxins to P-adequate roots resulted in localized alterations in root architecture that mimicked those observed under low P (Gilbert *et al.*, 2000; López-Bucio *et al.*, 2002). It has been reported that changes in growth of lateral roots and cluster roots during low P are inter-related to auxin signalling (López-Bucio *et al.*, 2002, 2005; Nacry *et al.*, 2005; Jain *et al.*, 2007). Lateral root development and the arrest of cell divisions in the apices in roots of

P-starved plants result from changes in auxin transport and/or sensitivity (López-Bucio *et al.*, 2005; Nacry *et al.*, 2005). Moreover, both *TRANSPORT INHIBITOR RESPONSE 1* (*TIR1*)- and *AUXIN RESPONSE FACTOR 19* (*ARF19*)-dependent auxin signals are suggested to have roles in lateral root development under low P availability (Pérez-Torres *et al.*, 2008). Furthermore, P-deprived *pnp* mutants develop aborted clusters of lateral roots, which are characterized by decreased auxin responsiveness and cell division, and cell death at the root tips (Marchive *et al.*, 2009). Consequently, good evidence indicates that auxins are an important signal controlling the low-P-induced development of lateral roots. Many hormonally regulated developmental responses occurring in P-deficient arabidopsis also appear to be involved in cluster root formation (Cheng *et al.*, 2011). In the process of P-induced cluster root formation, many genes involved in auxin synthesis and signalling are abundantly expressed (Vance *et al.*, 2003; Yamagishi *et al.*, 2011). Impaired auxin transport in white lupin roots decreased formation of cluster roots under low P supply (Gilbert *et al.*, 2000). Therefore, the development of cluster roots under low P is also auxin related.

Similarly, the growth of root hairs is dependent on auxin signalling under P deficiency. This is supported by a study showing that the indole acetic acid (IAA) transport inhibitor CMPA produces a dramatic decrease in root hair elongation and root hair density of arabidopsis grown at low P availability (Bates and Lynch, 1996). This implies that an appropriate concentration of endogenous auxin and normal auxin transport in roots are critical for the regulation of low-P-induced root hair development. However, exogenous IAA increased both the density and elongation of root hairs by up to 3-fold under high P, but only slightly increased these parameters under low P, when root hair density and length were already high (Bates and Lynch, 1996). Future research should explore the role of auxin response and transport in regulating specific phases of root hair growth, such as root hair initiation and tip growth in response to low P.

Meanwhile, many other studies suggest that primary root and root hair growth in response to low-P stress are auxin independent (Williamson *et al.*, 2001; Linkohr *et al.*, 2002; Ticconi *et al.*, 2004; Jain *et al.*, 2007). Jain *et al.* (2007) found that auxin did not play a role in the determinate growth of the primary root exposed to localized P deficiency. Hence, it becomes clear that an auxin-dependent pathway involved in auxin transport or sensitivity and an auxin-independent pathway may coexist to modulate P-starvation-induced root architectural changes. These studies highlight the differential effects of auxin on P-deficiency-induced modulations of ontogenetically distinct root traits.

Other signalling pathways, including other phytohormones, may co-function with auxin in this process. For example, ethylene has been shown to act, at least in part, through changes in auxin biosynthesis and transport (Swarup *et al.*, 2007; Grierson and Schiefelbein, 2009). Auxin and ethylene can interact in their biosynthesis and the response pathways, or, sometimes independently, regulate the same target genes (Stepanova *et al.*, 2007). Additionally, auxin imposes a very rapid regulation of cytokinins and nitric oxide (NO) (see later). Strigolactone has recently been shown to act in concert with auxin to regulate lateral root development and shoot branching in arabidopsis differentially, depending on the P level in the growth medium (Ruyter-Spira *et al.*, 2011). In other words, the interactions between phytohormones in regulating root architecture under nutrient stress are still equivocal, and deserve further investigation.

Ethylene

Ethylene has been shown to play a role in modifying root development in response to low P availability. Transcriptome analyses show that transcript levels for ethylene biosynthetic genes are increased in arabidopsis under low P (Thibaud *et al.*, 2010). By altering ethylene biosynthesis or perception, several studies have suggested that ethylene plays a role in modulating root architecture under P deficiency (Borch *et al.*, 1999; Ma *et al.*, 2003; Zhang *et al.*, 2003; Kim *et al.*, 2008; Chacón-López *et al.*, 2011). However, the regulation of root architecture by low P depends on both the root type and the specific phase of root growth (e.g. lateral root and root hair formation or elongation). For example, addition of the ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC) to arabidopsis seedlings grown in low-P conditions inhibited primary root growth and lateral root formation but had no influence on lateral root density (López-Bucio *et al.*, 2002). Furthermore, the negative effect of ACC on lateral root formation and the reduced formation of lateral roots in response to low-P conditions in the *ETHYLENE OVERPRODUCER 1* (*eto1*) and *CONSTITUTIVE TRIPLE RESPONSE 1* (*ctr1*) mutants suggest that ethylene plays a negative rather than a positive role in lateral root induction.

Although the precise role of ethylene in regulating root adaptations associated with P availability has not been fully understood, it appears that ethylene regulates root elongation via changes in both biosynthesis and responsiveness. Phosphorus-deficient roots produced twice as much ethylene per gram of dry matter as P-sufficient roots in common bean (*Phaseolus vulgaris*) (Borch *et al.*, 1999). Enhanced ethylene production and altered ethylene sensitivity in P-deficient common bean were responsible for root responses to P deficiency (Borch *et al.*, 1999). The angle of the basal roots in common bean can be modulated by sensitivity of the basal roots to ethylene stimulated by low P status (Basu *et al.*, 2007). The *ETHYLENE INSENSITIVE 1* (*etr1*) mutant of arabidopsis has normal responses in terms of lateral root formation and primary root inhibition when exposed to low-P conditions, whereas *eto1* and *ctr1* mutants are less responsive (López-Bucio *et al.*, 2002). Likewise, ethylene-insensitive 'Never-ripe' tomato plants are unable to respond to low P with increased adventitious root development (Kim *et al.*, 2008). These findings demonstrate that both the biosynthesis and responsiveness of ethylene regulate root elongation and some aspects of lateral root growth.

Direct sensing of low P and increased ethylene production are thought to induce the initiation and elongation of root hairs (Bates and Lynch, 1996; Zhang *et al.*, 2003). Ethylene enhances root hair density by increasing the number of hair cells and shortening trichoblast cells to increase the number of hair cells per unit length (Zhang *et al.*, 2003). The proportion of hair cells and root hair length were reduced in ethylene-insensitive mutants of arabidopsis, especially in the

presence of low P (Zhang *et al.*, 2003). Furthermore, a low-P signal may directly activate primary ethylene response genes involved in epidermal cell differentiation (Schmidt and Schikora, 2001). Hence, ethylene signalling may be utilized during the manifestation of P starvation responses as a way to fine-tune the adaptations.

Several lines of evidence indicate that ethylene stimulates auxin biosynthesis, and the synergistic interaction between them modulates root growth and root hair development (Osmont *et al.*, 2007; Stepanova and Alonso, 2009). Evidence has been presented that root hair formation is modulated by the developmental pathways, and, for environmental stress, ethylene/auxin signalling is a predominant feature. Some of these pathways regulate only a specific change, whereas others may modulate interactive effects. However, the precise role of ethylene in regulating cluster root adaptations associated with P availability is still unclear.

Cytokinins

Under normal conditions, cytokinins are traditionally associated with stimulation of shoot growth and inhibition of root growth (Aloni *et al.*, 2006). Cytokinins can reduce RAM activity, which results in a reduction of low-P responses (Lai *et al.*, 2007). *AtIPS1* and other P-starvation-inducible genes are repressed by cytokinins in roots of arabidopsis (Martín *et al.*, 2000). In turn, low P represses the action of cytokinins by reducing the cytokinin concentration (Horgan and Wareing, 1980; Kuiper *et al.*, 1988) and decreasing the expression of *CYTOKININ RESPONSE 1* (*CRE1*), a cytokinin receptor (Franco-Zorrilla *et al.*, 2002). Theoretically, a decrease in cytokinin concentration in plant roots during P deficiency could alleviate the inhibition of root growth. As expected, some studies have shown that cytokinins suppress lateral root initiation of arabidopsis plants under low-P conditions (López-Bucio *et al.*, 2002) and acts negatively for increased root growth and other P-starvation responses (Martín *et al.*, 2000; Franco-Zorrilla *et al.*, 2002). Similarly, the expression of the gene for cytokinin oxidase/dehydrogenase (CKX) increases significantly in cluster roots of P-deficient white lupin (Vance *et al.*, 2003). Also, many ESTs that annotate to cytokinin oxidase are found in mature segments of P-deficient cluster roots (Uhde-Stone *et al.*, 2003*b*), suggesting that cytokinins are involved in cluster root development and maturation. Moreover, addition of cytokinin to white lupin significantly reduces the number of emerged cluster roots and inhibits cluster rootlet elongation (Neumann *et al.*, 2000). Therefore, future research is warranted to clarify whether the stimulation of cluster root formation and development in P-deficient plants is partly due to the increased production of cytokinins induced by low P.

It is important to note that the changes in cytokinin signalling during P starvation are the secondary response, as a consequence of cross-talk between auxins and P signalling cascades. Auxins can impose a very rapid regulation of cytokinin biosynthesis (Nordström *et al.*, 2004; Aloni *et al.*, 2006). Conversely, by influencing auxin transport and homeostasis, cytokinins inhibit lateral root formation (Laplaze *et al.*, 2007). These findings imply that auxins and cytokinins could interact at the metabolic level in controlling plant root development. Consequently, the repressor effect of cytokinins does not appear to be a consequence of changes in root development caused by this hormone, as it occurs in all root cell types and in root tissue formed both before and after cytokinin treatment (Franco-Zorrilla *et al.*, 2005). This indicates a factor of potential importance for auxin–cytokinin-regulated root development. Apart from interacting with auxins, a complex interaction among cytokinins, sugars, auxins and P starvation signalling has been proposed (Franco-Zorrilla *et al.*, 2005). There is no apparent difference in growth and development when seeds of arabidopsis were germinated under P deficiency, or in response to the growth regulators ACC, IAA or cytokinin (Hong *et al.*, 2008). Therefore, future investigations should be focused on the multilevel interactions among sugars, auxins, ethylene and cytokinins in P-starvation-induced root architecture development, especially in the primary root and root hair growth.

Reactive oxygen species

Reactive oxygen species have been shown to be related to signal transduction responses, and play an important role in signalling, and hence affect root growth and development. Recently, it was reported that the redox status, probably mediated by jasmonic acid and ethylene, played a crucial role in the primary root meristem exhaustion process triggered by P starvation (Chacón-López *et al.*, 2011). In response to P deficiency, changes in ROS concentration and distribution in specific root cells occurred, although different patterns were reported (Potters *et al.*, 2002; Shin *et al.*, 2005; Tyburski *et al.*, 2009). These results suggest that ROS act positively in the root growth response to P deprivation.

The apex of the primary root has two areas of ROS production: the QC and the elongation zone (Jiang and Feldman, 2003; Liszkay *et al.*, 2004). The oxidative environment in the QC and the root elongation zone are important for maintaining a low rate of cell division and increasing cell wall extensibility, respectively (Jiang and Feldman, 2003; Liszkay *et al.*, 2004). Rapidly growing roots of plants in P-sufficient medium synthesize ROS in the root elongation zone and QC. Coincidentally, P deficiency is accompanied by a diminution of ROS accumulation in the elongation zone (Tyburski *et al.*, 2009) and the root tip (Chacón-López *et al.*, 2011). However, the research results of Tyburski *et al.* (2009) suggest that the mechanism responsible for growth inhibition of the primary root of arabidopsis under low P does not involve ROS accumulation, as would be expected from Fe toxicity, but rather a retranslocation of produced ROS. This is probably because of a typical pattern of ROS distribution in the root apex with the local maxima in the QC and root elongation zone.

It has been hypothesized that auxin, by regulating the ROS status of the discrete zones within the RAM, may act as a positional signal and mediate meristem patterning (Jiang and Feldman, 2003). ROS are also involved in the auxin-dependent regulation of cell elongation. The growth inhibition of maize roots by auxins is associated with a decrease in ROS production (Liszkay *et al.*, 2004). Tyburski *et al.* (2012) demonstrated that the differences in the length of primary roots observed at low and high P are accompanied by changes in ascorbate content and

redox status. Cells of the QC that accumulate high auxin levels are characterized by the oxidized status of ascorbate and glutathione and the overproduction of ROS (Tyburski *et al.*, 2012). Interestingly, the localization of ROS after P deprivation differs from that observed under nitrogen and potassium deprivation. Phosphorus deficiency increases ROS in the root cortex, whereas N and K deficiencies enhanced ROS in the epidermis (Shin *et al.*, 2005). Therefore, ROS production could be used as a measure of root response to nutrient deficiency.

Nitric oxide

Nitric oxide has been reported to be involved in diverse physiological and developmental processes in plants. It is also a second messenger in auxin signal transduction leading to root developmental processes. It has been reported that NO plays a key role in auxin-induced adventitious root development in cucumber (*Cucumis sativus*) (Pagnussat *et al.*, 2003, 2004) and that NO increases root hair length and density in lettuce (*Lactuca sativa*) (Lombardo *et al.*, 2006). Therefore, it is reasonable to hypothesize that NO is concerned with part of P-deficiency-induced root development. Evidence shows that NO plays a key role in the initiation of lateral roots in tomato and maize (Correa-Aragunde *et al.*, 2004; Creus *et al.*, 2005; Zandonadi *et al.*, 2010), formation of crown roots in rice (Xiong *et al.*, 2009*a*, *b*) and cluster root development in white lupin under low-P conditions (B.L. Wang *et al.*, 2010; Meng *et al.*, 2012).

It is worth mentioning that the patterns of NO production in the cluster roots of white lupin differ, depending on the root zone, developmental stage and P nutritional status. In addition, NO is involved in the root physiological response to Fe deficiency in tomato (Graziano and Lamattina, 2007) and enhances the citrate exudation in cluster roots of P-deficient white lupin (B.L. Wang *et al.*, 2010). These findings imply that NO is a node in the possible shared signalling pathway in the formation of cluster roots under P or Fe deficiency. Yet the physiological basis of these processes and the regulatory mechanisms involved in them are unknown and warrant further investigation. Moreover, since both cytokinins and NO may act downstream of auxins to influence root growth, the interaction between cytokinins and NO is important in controlling root development in response to low P, and further work is also needed to understand its mechanisms.

Abscisic acid

Abscisic acid (ABA) plays a role in root development (Beaudoin *et al.*, 2000; De Smet *et al.*, 2003). Liang *et al.* (2007) showed that ABA could function as a growth inhibitor in certain cells, tissues or organs (e.g. shoot) and as a growth promoter in other organs (e.g. root). Later, it was suggested that ABA might possess dual functions: a growth inhibitor in the presence of severe drought stress and a promoter of root growth in the absence of stress or under moderate stress conditions (associated with relatively low endogenous ABA levels) (Cheng *et al.*, 2002). The role of ABA in the P-stress response was addressed by Radin (1984) and Trull *et al.* (1997) who observed no differences in ABA concentration in P-deficient and P-sufficient cotton plants except when water stress was also applied. Trull *et al.* (1997) speculated on roles of ABA in influencing root development of the plant in response to P stress. There is indirect evidence showing that the ABA signal is involved in root growth induced by P deficiency, but no direct relationship between ABA signalling and P-response genes has been established. Interestingly, in castor bean (*Ricinus communis*), low P availability stimulated the xylem transport of ABA although it did not affect the concentration of ABA (Jeschke *et al.*, 1997). Moreover, recent genetic and phenotypic analyses have revealed complex interactions among ABA, sucrose and ethylene signalling (Beaudoin *et al.*, 2000; Ghassemian *et al.*, 2000; Sharp, 2002; Sharp and LeNoble, 2002). For example, Spollen *et al.* (2000) showed that the stunted growth of ABA-deficient maize plants was caused by the overproduction of ethylene and that ABA may function to prevent the overproduction of ethylene. Earlier studies showed that N or P deficiency increases stomatal responsiveness to ABA, perhaps through changes in the cytokinin concentration (Radin, 1984; Radin and Hendrix, 1988). However, the action of ABA in lateral root development is thought to be independent of auxins (De Smet *et al.*, 2003), but is modulated by nutrient availability (Signora *et al.*, 2001). Considering the above discussion about cytokinins, it is speculated that ABA and cytokinins act oppositely in regulating root architecture development under low-P conditions.

Other nutrients

Recent studies have revealed that morphological changes of roots under P starvation are actually an outcome of complex interactions between P and other nutrients, such as N, Fe and Ca. Dinkelaker *et al.* (1995) reported that low levels of N enhanced formation of proteoid root under P deficiency, while high N levels exhibited an inhibitory effect. However, Sas *et al.* (2002) found that the addition of ammonium stimulated cluster root formation and proton excretion in white lupin under low-P conditions. Similarly, Jing *et al.* (2010) reported that localized application of P combined with ammonium can significantly improve maize root growth and nutrient use at early stages by stimulating root proliferation and rhizosphere acidification. Our research group recently observed that there was a distinct difference between root architecture of nitrate-fed and ammonium-fed arabidopsis under low P availability (Niu *et al.*, 2012).

It has been shown that P deficiency enhances Fe accumulation in arabidopsis, and Fe supply influences the response of root architecture and primary root elongation to P deficiency. When the Fe concentration in the P-deficient medium was reduced, recovery of primary root elongation was observed in arabidopsis without an increase in P availability (Ward *et al.*, 2008). On the other hand, primary root inhibition is a well-documented response to the toxicity due to excess supply of some nutrients, including Fe (Marschner, 1995). This implies that manipulating Fe availability to a plant could be a valuable strategy for improving the tolerance of the plant to P deficiency.

Recently, it has been shown that increased Ca uptake enhances P availability by solubilization of Ca phosphates in the rhizosphere (Devau *et al.*, 2010). Two mutants of arabidopsis, *CATION EXCHANGER 1*, *3* (*cax1cax3*) and *INOSITOL-*

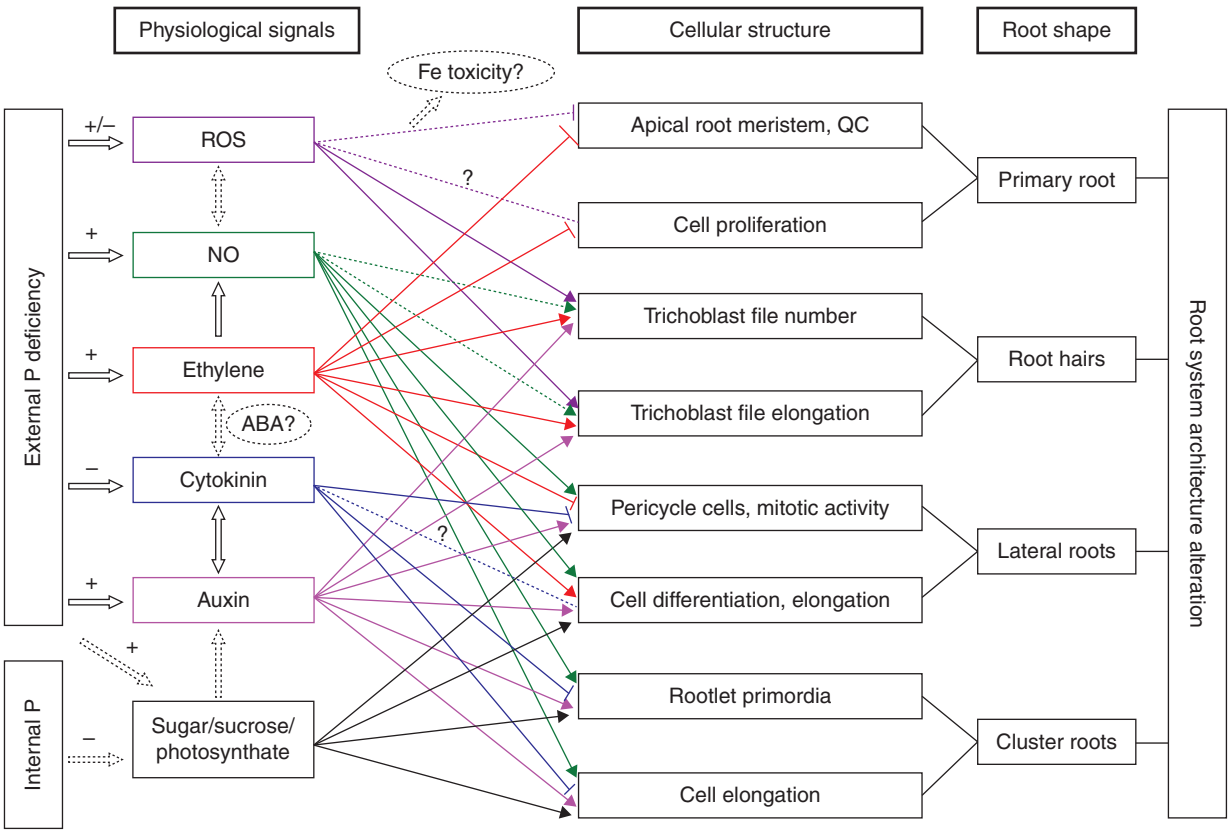

FIG. 1. Conceptual model showing the potential low-P target points in the signalling events leading to the alteration of root architecture. Solid arrows indicate links established in the induction of root development, and dashed lines denote indirect or as yet undescribed pathways. A bar at the end of a line indicates inhibition. Different colours indicate different regulatory signals. This model is developed based principally on information from arabidopsis and white lupin.

PENTAKISPHOSPHATE 2-KINASE 1 (*ipk1*), which are defective in two vacuolar Ca^{2+}/H^{+} exchangers and impaired in inositol polyphosphate kinase, respectively, have a greater P concentration in shoots (Stevenson-Paulik *et al.*, 2005). Our unpublished data showed that Mg availability is likely to play a role in affecting some aspects of root growth of arabidopsis through changes in cytosolic Ca^{2+} concentration. Thus, the combined application of other nutrients such as ammonium and Fe with P has the potential for modification of the root architecture. Consequently, nutrient interactions seem to be important for the manifestation of deficiency and toxicity symptoms in many plants, and root architecture under low-P conditions should be considered in the context of how it is influenced by the specific and non-specific interactions that P has with other nutrients.

CONCLUSIONS

Based on previous studies and the above discussion, a model can be generalized for how P deficiency regulates the development of root architecture (Fig. 1). This model is mainly based on models proposed by Cheng *et al.* (2002), Niu *et al.* (2011*a*, *b*) and Péret *et al.* (2011). Low P availability alters the local concentration, transport or sensitivity of intracellular compounds such as carbohydrates, especially sugars, auxins, ethylene, cytokinins, NO and ROS, and subsequently modulates the downstream genetic elements that control the initiation and maintenance of root development. Notably, this distinct pathway controlling the initiation and maintenance of root meristems in different root types may be involved in context-specific use of homologous factors or interactions that occur between these hormonal signalling pathways. Although there is much evidence to support this model, the current model of how the changes in root phenotype in response to low P availability are modified by the integrated output of these hormonal cross-talks is far from being complete due to the complexity of the synergistic, additive or antagonistic effects of phytohormones on signalling pathways. Nevertheless, the changes in root architecture to adapt to low-P conditions could greatly improve P-use efficiency. Understanding the relevant regulatory mechanisms would allow plant breeders to define the selection criteria for the development of P-efficient crops, thus reducing the use of fertilizers.

ACKNOWLEDGEMENTS

We thank the anonymous reviewers for their constructive comments on the manuscript. This work was financially supported by the Chinese Ministry of Agriculture (201103004), the State Key Development Program for Basic Research of China (973 Program, no. 2009CB119003) and the National Key Project on Science and Technology of China (2012BAC17B02).

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