Auxin and Temperature Stress: Molecular and Cellular Perspectives

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Abstract Temperature stress is one of the major abiotic stresses that limit plant growth and development and crop productivity worldwide. Plant growth and development is also influenced by endogenous factors such as hormones, and under environmentally stressed conditions. Plants adapt themselves through multiple processes, including a change in hormonal response. Recent evidence indicates that under optimal condition, the plant hormone auxin plays a key role in determining plant development processes through modulating other hormonal responses. However, little is known about the role of auxin under temperature stress. The emerging picture from recent experiments indicates that like under optimal condition, auxin also plays a crucial role in regulating plant growth under temperature stress. In this chapter, we tried to integrate our current understanding on the role of auxin in regulating plant developmental processes under temperature-stressed condition and the future direction of research that may help us in engineering plants/crops for sustainable agriculture.

1 Introduction

As a sessile organism, plant encounters various environmental changes during its life cycle. One of the major environmental changes that affect plant development is temperature. Shift in temperature either to the high or low end drastically affects plant growth and crop productivity. For instance, in 2009, a chilling temperature alone resulted in crop damages equivalent to 158 billion yen in Japan. Similarly,

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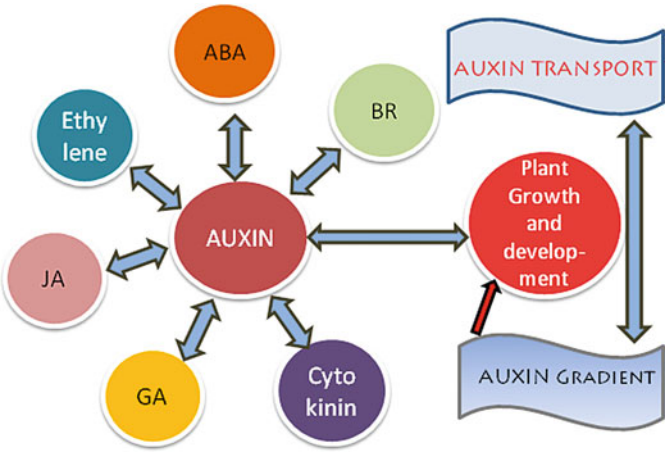

Fig. 1 Auxin at center of hormonal cross talk in regulating the plant growth and developmental processes under optimal condition. Reproduced from Rahman (2012) with permission from Physiologia Plantarum

early and late frost results in damaging vegetable and fruit production equivalent to 5–6 billion yen every year (Rahman 2012). High-temperature limitation of crop yield is also a well-recognized problem in India, Africa, and many other countries. High-temperature stress is a frequent occurring event during rice and wheat growing seasons. It has been shown that heading and flowering stages are most sensitive to the high-temperature stress (Matsui and Omasa 2002). In case of wheat, maize and barley, the combined annual loss rendered by high temperature is $5 billion (Lobell and Field 2007). These data strongly support the notion that temperature stress is one of the major abiotic stresses that limit the plant growth and crop productivity worldwide.

Plant growth is also influenced by endogenous factors such as hormones. In fact, every aspect of plant growth from germination to senescence is under hormonal regulation. In regulating plant developmental processes, hormones act in concert, resulting in a complex web of interactions. Interestingly, over the past decades with the aid of Arabidopsis genetics, it became evident that among all plant hormones, auxin plays a central role in determining plant developmental processes through modulating other hormonal responses (Fig. 1). Further, it has been established that an auxin gradient, which is regulated by auxin transport, plays a major role in regulating hormonal cross talks and plant development (Fig. 1; Rahman 2012). Under environmentally stressed conditions, plants adapt themselves to the adverse condition through multiple processes, including changes in hormonal responses. To date, a great deal of information is available about transcriptional regulators that play key roles in plant temperature stress responses (Hua 2009; Rahman 2012). However, the roles of hormones in regulating temperature stress responses are far from understood. Among the plant hormones, although auxin plays a central role in regulating plant developmental processes and responses to other hormones at an

optimal condition, our understanding of the role of auxin in temperature stress responses is limited. However, some recent work shed a light on this aspect and the emerging picture suggests that like under optimal condition, auxin also plays an important role in regulating plant development under temperature stress conditions. This chapter integrates auxin, temperature stress, and plant growth and development.

2 Auxin

2.1 Auxin Synthesis

Auxin biosynthesis is a complex process involving multiple pathways acting in concert. The biosynthesis of the major form of auxin, indole-3-acetic acid (IAA) largely relies on tryptophan (Trp)-dependent pathway (Zhao 2010). Biochemical and physiological experiments indicated the existence of a Trp-independent pathway that may contribute to in vivo IAA biosynthesis. However, no genetic basis has been established for this pathway (Cohen et al. 2003; Strader and Bartel 2008; Zhao 2010). Four major pathways contribute in Trp-dependent IAA biosynthesis: (1) indole-3-acetaldoxime (IAOx) pathway, (2) indole-3-acetamide (IAM) pathway, (3) indole-3-pyruvic acid (IPA) pathway and (4) YUCCA (YUC) pathway (Zhao 2010; Mashiguchi et al. 2011). IAOx pathway was elucidated on the basis of three auxin overproduction mutants, *superroot 1* (*sur1*), *superroot 2* (*sur2*), and *CYP79B2* overexpression lines (Hull et al. 2000; Zhao et al. 2002). However, this pathway is probably not the major IAA biosynthesis pathway as CYP79B2 and its homologue CYP79B3 is absent in monocots (Sugawara et al. 2009). The IAM pathway possibly widely exists in plants. IAM is found in Arabidopsis and an *AMIDASE1* (AMI1) gene has been cloned in Arabidopsis, which can convert IAM to IAA (Pollmann et al. 2003). However, the physiological significance of this pathway is still elusive. The significance of IPA pathway has been recently demonstrated through genetic studies. Three independent studies identified an Arabidopsis aminotransferase, TAA1 (tryptophan aminotransferase of Arabidopsis 1) that can convert Trp to IAA in vivo (Stepanova et al. 2008; Tao et al. 2008; Yamada et al. 2009). The YUC pathway has been proposed as the major IAA biosynthesis pathway as YUC genes are identified ubiquitously across the plant kingdom (Zhao 2010). Further, the genetic evidence also indicates that *YUC* genes play a central role in IAA-regulated developmental processes (Zhao et al. 2001, Cheng et al. 2006; 2007; Yamamoto et al. 2007). *YUC* gene family encodes flavin monooxygenases and showed to catalyze the conversion of tryptamine (TAM) to N-hydroxy-tryptamine (HTAM) in vitro (Zhao et al. 2001; Kim et al. 2007). Based on this IAOx and indole-3-acetonitrile (IAN) were previously reported as possible intermediates of YUC pathway (Zhao et al. 2001). In a metabolite study, Sugawara et al. (2009) showed

that IAOx and IAN are not common intermediates of IAA biosynthesis in plants, raising a question about the validity of the in vitro study. The same group recently convincingly showed that TAA and YUC families function in the same pathway and YUC catalyzes the conversion of IPA to IAA, a rate-limiting step for IAA-regulated plant developmental processes (Mashiguchi et al. 2011).

Traditionally, it is believed that shoot is the sole source of auxin biosynthesis and the auxin supply to other parts of a plant relies on the polar transport of auxin. Recent evidence clearly suggests that root and shoot both can synthesize auxin (Cheng et al. 2006; Stepanova et al. 2008; Pagnussat et al. 2009). This indicates that each organ is possibly self-sufficient in terms of synthesizing auxin, but the question remains open how they maintain the optimal auxin gradient required for plant development and how polar transport of auxin contributes in maintaining the gradient.

2.2 Auxin Transport

Among the plant hormones, auxin is unique as it moves from the site of synthesis through an active transport system (Goldsmith 1977). Auxin is transported both in rootward and shootward directions (Baskin et al. 2010). In the aerial part, auxin moves unidirectionally towards the root and in root, it moves in both directions (Muday and Rahman 2008). The components that regulate the transport pathway have been identified by using Arabidopsis genetics. There are two major protein families that regulate auxin transport, AUX/LAX family, which functions as auxin influx carriers (Bennett et al. 1996), and PINs, which function as auxin efflux carriers (Feraru and Friml 2008). These proteins show tissue-specific expression and regulate transport of auxin in specific tissues (Swarup et al. 2004; Feraru and Friml 2008). In general, influx and efflux transporters regulate intercellular auxin transport and maintain the local auxin gradient which is extremely important in regulating plant developmental processes (Muday and Rahman 2008). Besides these protein families, ABC transporter family has also been shown to mediate auxin transport (Peer et al. 2011). The functional specificity of the PINs has been substantiated by genetic evidence in Arabidopsis. For instance, PIN1 functions in rootward auxin transport and primarily expressed in the vascular tissue (Geldner et al. 2001; Blilou et al. 2005). PIN2 is expressed in outer cell layers with opposite polarity in lateral root cap cells, epidermis, and cortex and regulates the root gravity response (Muday and Rahman 2008; Rahman et al. 2010). The direction of PIN-mediated auxin transport depends polar targeting of the PIN proteins to the right plasma membrane domain of the cells (Friml et al. 2004; Michniewicz et al. 2007). PIN3 is localized symmetrically but relocates asymmetrically to the direction of gravity and partially involved in regulating the gravity response both in root and shoot (Friml et al. 2002b; Harrison and Masson 2008; Rakusova et al. 2011). PIN4 appears to work in the establishment of an auxin sink below the quiescent center of the root meristem (Friml et al. 2002a), and PIN7 is expressed at lateral and basal

membranes of provascular cells in the meristem and elongation zone and plays a role in forming and maintaining the rootward auxin transport (Blilou et al. 2005). Other members of the PIN family (PIN 5, 6, 8) are expressed in the endoplasmic reticulum (Mravec et al. 2009; Friml and Jones 2010).

One of the major factors that regulate the functionality of the auxin efflux carriers is intracellular trafficking of these proteins. PIN proteins continuously cycle between the plasma membrane and endosomes (Geldner et al. 2001). Efficient targeting to the plasma membrane and turnover of PIN proteins determine the proper functionality of these proteins to transport auxin and form an auxin gradient (Geldner et al. 2001; Vieten et al. 2007; Shibasaki et al. 2009; Rahman et al. 2010), which regulates many developmental fates of plants (Friml 2003).

2.3 Auxin Gradient

Multiple experimental approaches (physiological, molecular, and cellular) revealed that intracellular auxin distribution, regulated by auxin homeostasis, results in formation of local auxin gradient that functions as a regulatory factor for plant developmental processes (Sabatini et al. 1999; Bhalerao and Bennett 2003; Leyser 2006; Prusinkiewicz and Rolland-Lagan 2006; Tanaka et al. 2006; Benjamins and Scheres 2008; Ikeda et al. 2009). The patterning or formation of plant organs starts with the accumulation of auxin followed by its redistribution to form a cellular or tissue-specific auxin gradient, which directs major developmental decisions, such as specification of the apical and basal poles and establishment of root and cotyledon (Friml 2003). Auxin gradient also regulates organogenesis of leaves, flowers, and lateral roots, as well as tropisms (Benkova et al. 2003; Muday and Rahman 2008). Auxin gradient formation largely relies on the intracellular targeting of PIN proteins, which is a highly dynamic process with continuous cycling of the PINs between the cell membrane and intracellular compartments (Geldner et al. 2001). Recent evidence suggests that clathrin-dependent endocytosis (Dhonukshe et al. 2007) and ARF-GEF-dependent exocytosis (Geldner et al. 2001) regulate the constitutive cycling of PINs. Additionally, the phosphorylation status, which is regulated by the counterbalancing activities of PINOID kinase and protein phosphatase 2A, also regulates PIN polarity and hence the flow of auxin (Michniewicz et al. 2007; Sukumar et al. 2009; Rahman et al. 2010). Taken together, these results suggest that under an optimal condition, the intracellular trafficking of PIN proteins contributes in maintaining the cellular auxin homeostasis that functions as a prime regulator of plant developmental processes.

3 Temperature Stress

3.1 Cold Temperature

The cold stress can be divided into two major categories, freezing and chilling. Compared with the chilling stress, freezing stress is severe and inhibits the expression of plant's full genetic potentials (Thomashow 1999). However, some of the plant species counter this severe stress through a process called cold acclimation. Cold acclimation is defined as a process by which plants acquire freezing tolerance through prior exposure to low nonfreezing temperatures. Winter-habit plants, such as barley, oat, and rye, require a low-temperature period (vernalization) to acquire the reproductive (flowering) phase (Kim et al. 2009). In Arabidopsis, vernalization induces histone modifications via the plant-homeodomain-PRC2 complex to generate high H3K27me3 levels (Angel et al. 2011). Interestingly, after vernalization, cold acclimation ability gradually decreases (Fowlerl et al. 1996). This phenomenon indicates that the effect of cold stress is reversible (Fowlerl et al. 1996). Many chilling-sensitive crop plants are incapable of cold acclimation. The chilling stress inevitably inhibits plant growth and development (Fukaki et al. 1996; Shibasaki et al. 2009). However, the stress is reversible as plants have the ability to regrow when they are returned to an optimal temperature (Fukaki et al. 1996; Wyatt et al. 2002, Shibasaki et al. 2009).

The cellular and molecular mechanisms that are integrated to cold stress responses are relatively well defined. At cellular level, although debatable, the plasma membrane has been suggested to be the primary site of cold perception as membrane composition changes both qualitatively and quantitatively in response to cold. Cold stress decreases the membrane fluidity due to changes in the fatty acid unsaturation and lipid-protein composition in the cell membrane (Wang et al. 2006). Rigidification of membrane either by mutation or by exogenous application of membrane rigidifier results in expression of cold-inducible genes even at room temperature (Inaba et al. 2003; Orvar et al. 2000; Sangwan et al. 2002). In addition, several membrane-localized proteins such as calcium-permeable channels, histidine kinases, and receptor kinases have been suggested to be putative sensors for cold response (Solanke and Sharma 2008). Calcium has been shown to affect the membrane composition through modulating phospholipid signaling (Vergnolle et al. 2005). Hence, it is thought that membrane rigidification may play an important role in cold perception as well as the cellular response to cold. However, in a recent experiment, it has been shown that membrane rigidification did not alter proper protein trafficking within plant cells and cold stress did not change intracellular localization and trafficking properties of the cold-inducible protein, LTI6b (Shibasaki et al. 2009).

At the molecular level, several cold stress-induced transcription factors have been identified and defined as transcriptional regulators of cold stress-induced genes. Currently, a molecular model predicts that after cold perception, plant uses a phosphorylation cascade counterbalanced by protein phosphatases and MAP

kinases and regulated by cytosolic calcium to transduce the signal to downstream signaling components (Hannah et al. 2005; Rahman 2012). Transcription factors such as ICE1 (inducer of CBF expression 1), MYB15, and zinc finger proteins have been suggested to be the primary regulators of cold-responsive gene expression (Xiong et al. 2002; Zhu et al. 2007). Recent evidence suggests that phosphorylation and SIZ1-mediated SUMO conjugation and deconjugation of ICE1 are the key processes to regulate ICE1 binding to its target genes. Once ICE1 is activated, it binds to MYC *cis*-elements in the *CBF* (C-repeat binding factor) promoter to induce the expression of target genes (Chinnusamy et al. 2003). The induction of the CBF genes at low temperature and the enhanced freezing tolerance of the transgenics overexpressing the CBFs suggest that this pathway plays a central role in regulating cold-stress response (Vogel et al. 2005).

Although hormones play a major role in regulating plant development at the optimal condition, the role of hormones in regulating cold stress is still elusive. The only hormone that has a potential link to abiotic stresses, including cold stress, is abscisic acid (ABA). However, the direct role of ABA in cold stress remains a mystery as it has been shown that the low-temperature-regulated gene expressions occur relatively independently of ABA (Thomashow 1999; Xiong et al. 2002; Shinozaki and Shinozaki 2006).

3.2 High Temperature

In contrast to low temperature, which severely inhibits plant growth and development, high temperature affects plant developmental processes differently. It stimulates the hypocotyl elongation, promotes flowering and inhibits pollen production (Gray et al. 1998; Balasubramanian et al. 2006; Sakata et al. 2010; Kumar et al. 2012). Like cold stress, at cellular level, high-temperature stress also affects cellular functions and membrane-linked processes due to alterations in membrane fluidity and permeability (Sangwan et al. 2002). Enzyme function is also sensitive to high temperature. High-temperature-induced alterations in enzyme activities can lead to shifts in metabolic pathways and can cause enzyme inactivation due to protein denaturation (Vierling 1991; Kampinga et al. 1995). The damaged membrane and nonfunctional proteins facilitate the production of reactive oxygen species (ROS) (Dat et al. 1998a, b; Gong et al. 1998; Larkindale and Knight 2002), which ultimately lead to programmed cell death (PCD) (Vacca et al. 2004). As a defense to high-temperature stress, plants produce heat shock proteins (HSPs) to obtain thermotolerance (Vierling 1991). These proteins are induced in plants during high-temperature acclimation and proposed to act as molecular chaperons to protect cellular proteins against irreversible high-temperature-induced denaturation and to accelerate refolding of damaged proteins (Boston et al. 1996; Hong and Vierling 2000, 2001). Heat-stress transcription factors, which bind to the promoter of HSPs, regulate the expression of these chaperons (Larkindale and

Vierling 2008). Recent evidence suggests that HSP 1011, Hsa32, HSFA2, HSP110, HSFA7a, and HSFA3 play important roles in thermotolerance (Hua 2009).

Unlike cold stress, the hormonal involvement in regulating high-temperature-mediated developmental processes is better understood. Several hormones including auxins, gibberellins, and brassinosteroids have been implicated in regulating plant growth under high-temperature stress (Gray et al. 1998; Stavang et al. 2009).

4 Auxin and Temperature Stress

4.1 Low Temperature and Auxin

Despite the fact that auxin regulates almost all aspects of plant development, little is known about the response of auxin under cold stress conditions. The first demonstration of involvement of auxin in cold stress responses came from the study of Morris (1979), who showed that cold stress inhibits auxin transport in a variety of species. Fukaki et al. (1996) showed that cold treatment inhibits the inflorescence gravity response in Arabidopsis. They further revealed that the cold stress effect is reversible as the gravistimulated inflorescence in cold bends to the gravity vector when it is returned to room temperature. This demonstrates the existence of a gravity-persistent signal and suggests that cold stress affects steps after the gravity perception (Fukaki et al. 1996). Consistently, rootward auxin transport was found to be abolished at 4 °C but was restored to the wild-type level when the plants were returned to the room temperature (Wyatt et al. 2002; Nadella et al. 2006). Wyatt et al. (2002) took a genetic approach to identify components that separate the perception events from the response and screened for mutants in which the gravity-persistent signal was aberrant. Although these *gps* mutants respond abnormally to the gravity stimulus, amyloplast sedimentation is apparently normal, suggesting that the aberrant response is caused by an event (or events) that links gravity perception to auxin transport. Further studies of *gps* mutants revealed that these mutants fail to establish the proper auxin gradient in the inflorescence after gravistimulation and also show altered polar and lateral auxin transport (Nadella et al. 2006). Taken together, these results clearly demonstrate that cold stress affects the auxin response *in planta*. However, what remains obscure is the molecular mechanism that regulates the response of auxin under cold stress. In a recent study, using Arabidopsis root as a model, we tried to answer the question (Shibasaki et al. 2009). The physiological studies revealed that cold stress inhibits both root growth and gravity response in a reversible manner as the plants start to regrow and respond to gravity when they are returned to room temperature, albeit with a lag period. Genetic studies confirmed that the primary target of cold stress is auxin transport as the auxin signaling mutants (*axr1* and *tir1*) respond to cold stress like wild type. Direct transport assay with radiolabeled IAA further confirmed the notion. Under cold, the shootward auxin transport is drastically reduced, which also alters the

intracellular auxin gradient, as visualized by the auxin-responsive marker (Shibasaki et al. 2009). These results suggest that cold stress alters the intracellular auxin homeostasis, which possibly leads to altered growth and development. To further understand the cellular mechanism behind cold stress-induced changes in auxin homeostasis, polar targeting and trafficking of the auxin efflux carriers PIN2 and PIN3 were analyzed. As described earlier, both polar deployment and efficient targeting of the PIN proteins are essential for their functionality. Cold stress does not alter polar localization of PIN2 proteins but inhibits the intracellular cycling of PIN2, indicating that the reduced intracellular cycling affects PIN's functionality, resulting in reduced shootward auxin transport and altered intracellular auxin homeostasis. The selectivity of cold stress on PIN trafficking was substantiated by several markers, representing different pathways such as GFP-ARA7 (Ueda et al. 2004) for endosomal trafficking; NAG-GFP (Essl et al. 1999) for Golgi trafficking; and a GFP-LTI6b (Kurup et al. 2005), for trafficking induced by low temperature and FM4–64, a general endocytic tracker (Bolte et al. 2004). All the results indicated that cold stress inhibits a selective trafficking process and targets the early endosomal cycling pathway, including PIN trafficking (Shibasaki et al. 2009). Collectively, these results provide a mechanistic explanation of cold stress-induced changes in auxin response and shed light on the importance of intracellular trafficking in regulating cold-stress response. This is not surprising as intracellular trafficking pathways have been emerging as central regulators of plasma membrane protein homeostasis, controlling multiple signaling pathways, mediating interactions between multiple hormones, and controlling growth and development in both animals and plants (Grant and Donaldson 2009; Reyes et al. 2011).

4.2 Auxin and High Temperature

In contrast to cold temperature, the role of auxin is better understood in high-temperature stress responses. High temperature affects the growth of the plant in a tissue-specific manner. For example, it promotes the hypocotyl growth (Gray et al. 1998) and flowering (Kumar et al. 2012) but inhibits pollen formation (Sakata et al. 2010). Auxin has been found to be a common factor in regulating these distinct developmental processes under high temperature. Interestingly, current literature indicates that high-temperature-mediated alteration in developmental processes is tightly linked to auxin biosynthesis (Franklin et al. 2011; Sun et al. 2012). Several molecular studies indicated that phytochrome-interacting factor 4 (PIF4) is the primary regulator of the signaling mechanism that integrates auxin and plant development under high temperature (Koini et al. 2009; Franklin et al. 2011; Kumar et al. 2012). High-temperature-induced elongation of Arabidopsis hypocotyl is under regulation of PIF4-mediated auxin biosynthesis (Franklin et al. 2011; Sun et al. 2012). PIF4 directly activates the auxin biosynthetic gene YUCCA 8 (*YUC8*) by binding to the G-box-containing promoter region of *YUC8* and stimulates the auxin biosynthesis under high temperature (Sun et al. 2012). *yuc8* mutation can largely

suppress the long-hypocotyl phenotype of PIF4-overexpression plants and also can reduce the high-temperature-induced hypocotyl elongation, confirming that under high temperature PIF4 regulates auxin biosynthesis through activating the IPA auxin biosynthesis pathway (Franklin et al. 2011; Sun et al. 2012). PIF4 has also been shown to be instrumental in activating the *Flowering Locus T* (*FT*), which promotes the flowering at high temperature in short photoperiods (Kumar et al. 2012). Interestingly, the opposite effect of high temperature on auxin biosynthesis has been observed in developing anthers of barley and Arabidopsis (Sakata et al. 2010). In contrast to hypocotyls, high temperature inhibits the expression of auxin biosynthesis genes in developing anthers, which possibly results in a decrease in endogenous auxin level and results in male sterility. The notion that high-temperature-induced male sterility is due to reduction in tissue-specific auxin was supported by the fact that exogenous application of auxin completely reversed the male sterility (Sakata et al. 2010). These results are consistent with the previous finding that in high-temperature-tolerant rice variety Shanyou63, much slower rate of decrease of pollen activity, pollen germination, and floret fertility was observed compared with the high-temperature-susceptible variety Teyou559 (Tang et al. 2008). Not surprisingly, Shanyou63 showed an elevated level of endogenous auxin compared with Teyou559 (Tang et al. 2008). Taken together, these results support an elegant model where auxin plays a central role in regulating the high-temperature-induced alteration in plant developmental processes and also confirms that high-temperature-induced injury occurs in a tissue-specific manner.

5 Concluding Remarks

The emerging trend from the recent research clearly indicates that like optimal condition, auxin plays an important role in regulating plant developmental processes under temperature stress conditions. Both auxin synthesis and transport are found to be potential targets of temperature stresses. Our current understanding on the effect of temperature on cellular auxin response and plant developmental processes is summarized in Fig. 2. Although these findings bring new insights on how the plant hormone auxin is integrated in regulating plant developmental processes under temperature stresses, there are still many unanswered questions. Current data set represents involvement of two distinct processes that alter auxin response under temperature stress. Low temperature primarily targets auxin transport through modulating a subset of intracellular trafficking pathway that includes the trafficking of auxin efflux carriers, resulting in alteration of cellular auxin homeostasis as well as auxin gradient. On the other hand, high temperature primarily targets auxin biosynthesis through PIF4 transcriptional factor and affects the plant developmental processes. What still remains to be elucidated are whether auxin biosynthesis is affected under low temperature, what is the effect of cold

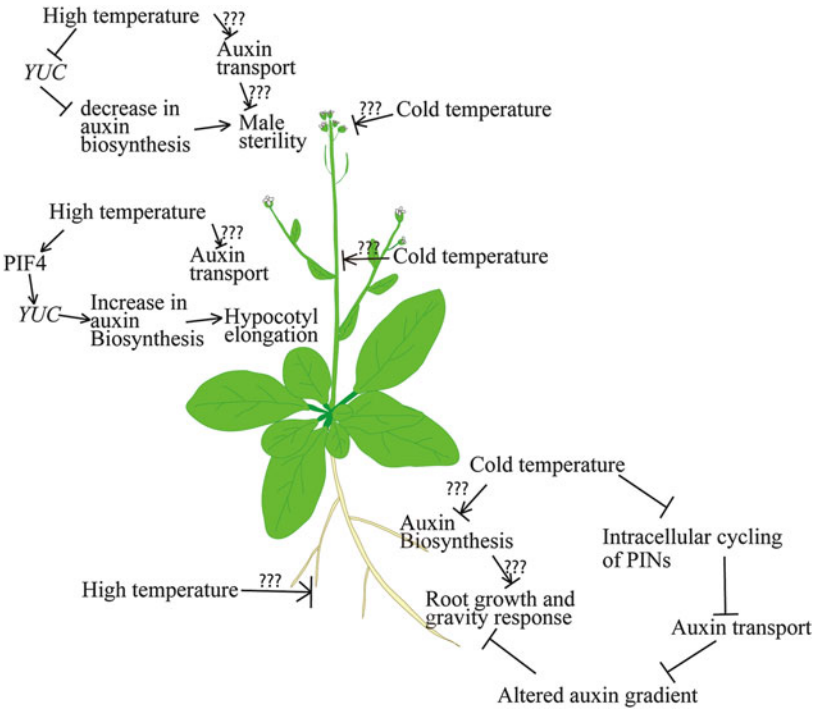

Fig. 2 Schematic summary of our current understanding on the effect of temperature on cellular auxin response and plant developmental processes. *Question marks* represent yet unidentified pathways that may influence plant auxin response under temperature stress

stress on transcriptional regulators of auxin response, and which transcriptional regulator plays a primary role in regulating the auxin responses under cold? Similarly, under high-temperature stress auxin biosynthesis is stimulated or reduced resulting in a change in cellular auxin homeostasis. However, the mechanism by which plants respond to this change is still unclear. Does high temperature also affect auxin transport process? Like cold, does protein trafficking play any roles in high-temperature-induced alteration of auxin homeostasis? Which protein trafficking pathway plays a central role in regulating the temperature stress? How do the changes of auxin response affect the other hormonal responses under temperature stress? Which components link the hormonal response to downstream signaling factors? A concerted effort is required to answer all these questions. Addressing these issues in future research will facilitate our understanding of temperature stress pathways and help engineer crops tolerant to temperature stresses.

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