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REVIEW PAPER

Research progresses on *GH3s*, one family of primary auxin-responsive genes

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Abstract Auxin plays a very important role in plant growth and development. Those genes that are specifically induced by auxin within minutes of exposure to the hormone are referred to as early/primary auxin-responsive genes, mainly including the *auxin/indole-3-acetic acid* (*Aux/IAA*), the *small auxin-up RNA* (*SAUR*), and the *GH3* gene families. So far, *GH3* genes have been identified in various plant species including soybean, *Arabidopsis*, rice, tobacco, pungent pepper, sweet orange, pine, and moss. Twenty members of *GH3* family were identified in *Arabidopsis* and these genes were classified into three groups (Group I–III) based on their sequence similarities and substrate specificities. GH3s belong to acyl adenylate-forming firefly luciferase superfamily and can catalyze adenylation of specific substrates. Group I adenylates jasmonic acid (JA), and Group II adenylates indole-3-acetic acid (IAA) and salicylic acid (SA), respectively. Because of the presence of Auxin-Responsive Elements (AuxRE) in the *GH3s'* promoter regions, Auxin Response Factors (ARFs) are able to bind to the AuxRE and regulate expression of some *GH3s*, which in turn modulate the auxin homeostasis. Identification of *GH3* mutants in *Arabidopsis* reveals the function of *GH3s* in hypocotyl elongation under different light conditions, root growth, stress adaptation, sensitivity to MeJA, or susceptibility to *P. syringae*. Taken together, GH3s may be linkers among auxin, JA, SA and light signal transduction pathways.

Keywords GH3 · Auxin · Auxin response element (AuxRE) · Auxin response factor (ARF) · Phytohormone

Abbreviations

2,4-D	2,4-Dichlorophenoxyacetic acid
ARF	Auxin response factor
Aux/IAA	Auxin/indole-3-acetic acid
AuxRE	Auxin-responsive element
BL	Brassinolide
BR	Brassinosteroid
IAA	Indole-3-acetic acid
Ile	Isoleucine
JA	Jasmonic acid
phyA	Phytochrome A

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SAUR Small auxin-up RNA
SA Salicylic acid

Introduction

Auxin is a critical plant hormone that modulates diverse growth and developmental processes such as tropic responses to light and gravity, root and shoot architecture, organ patterning, vascular development, as well as growth and differentiation in tissue culture (Hagen and Guilfoyle 2002; Woodward and Bartel 2005). Over the past 20 years, auxin has been discovered to exert rapid and specific regulation on the expression of auxin-inducible genes at transcriptional level. Those genes that specifically induced by auxin within minutes of exposure to the hormone have been extensively studied and regarded as primary/early auxin-responsive genes. These genes, including *Aux/IAAs*, *GH3s* and *SAURs*, share auxin response elements (AuxREs) in their promoter regions (Abel et al. 1994; Hagen and Guilfoyle 1985; Walker and Key 1982; Woodward and Bartel 2005). The first member of *GH3* gene family was isolated from soybean and accumulation of *GH3* transcript is induced by auxin (Hagen and Guilfoyle 1985). There are increasing evidences indicating that *GH3* family members function in modulating the level of free auxin, JA as well as SA via amino acid conjugation and light signaling may be involved in this process (Hsieh et al. 2000; Woodward and Bartel 2005). The present review mainly focuses on the distribution, the promoter characteristics, biochemical function and biological functions of the *GH3* family in plant.

Distribution of *GH3* genes in plant

In the past 20 years, *GH3* genes were identified in angiosperms (including both dicotyledons and monocots), gymnospermae, and moss. The first identified *GH3* transcript was derived from auxin-treated soybean seedlings (Hagen and Guilfoyle 1985). The soybean *GH3* genes was shown to be specifically induced by exogenous auxin treatment within 5 min, and this induction was not affected by treatment with the protein synthesis inhibitor cycloheximide, suggesting that the induction does not need de novo synthesis of protein (Franco et al. 1990; Hagen and Guilfoyle 1985). The *Arabidopsis thaliana GH3* gene family consists of 19 members and an additional partial gene encoding only the amino-terminal residues of the protein. Members of *Arabidopsis thaliana GH3* gene family located on chromosomes 1, 2, 4 and 5, but not on chromosome 3 (Table 1). Based on the phylogenetic analysis along with the substrate specificities, the GH3 family in *Arabidopsis* has been classified into three groups and there are two, eight and ten members in Group I–III, respectively (Table 1; Staswick et al. 2002). Group I includes AtGH3-11/FIN219/JAR1 and AtGH3-10/DFL2 (Hsieh et al. 2000; Staswick et al. 2002; Takase et al. 2003). Group II consists of AtGH3-2/YDK1, AtGH3-5/AtGH3a/WES1, AtGH3-9, AtGH3-6/DFL1 and four other members (Nakazawa et al. 2001; Takase et al. 2004; Tanaka et al. 2002). Group III is composed of AtGH3-12/PBS3/GDG1 and nine other members (Jagadeeswaran et al. 2007; Nobuta et al. 2007).

In addition to soybean and *Arabidopsis*, *GH3*-like genes were also found in other dicotyledon species. Roux and Perrot-Rechenmann (1997) first isolated a *GH3*-like gene from *Nicotiana tabacum*, which was designated as *Nt-gh3* sharing 70% identity with the soybean *GH3*. Since then, Lahey et al. (2004) detected transcript of GH3-like protein in *Citrus madurensis*. Liu et al. (2005) also identified a *GH3*-like gene from pungent pepper (*Capsicum chinense* L.), whose predicted protein shares 95% identity with Nt-gh3.

GH3-like genes were also identified in monocots. Using genomic approaches and gene expression analysis, 13 *GH3-like* ORFs (including 12 active genes) were identified in rice (*Oryza sativa*). Unlike AtGH3s, however, OsGH3s are only able to be subdivided into two Groups (Group I, II) based on sequence similarity to *Arabidopsis* GH3 (Jain et al. 2006; Terol et al. 2006). Group III *GH3*-like genes have not been found in rice. Furthermore, an extensive survey of the EST database of other monocots, including wheat (*Triticum aestivum*), corn (*Zea mays*), *Sorghum bicolor*, sugarcane (*Saccharum officinarum*), and barley (*Hordeum vulgare*), indicates that Group III of GH3 is absence in monocots (Jain et al. 2006).

In the gymnospermae, *Pinus pinaster*, and the moss, *Physcomitrella patens*, *GH3*-like genes were

Table 1 *Arabidopsis* GH3 family members

GH3[a]	Gene code	Chromosome	Group (s)	Substrate of adenylation[b]	Synonymous
AtGH3-1	At2g14960	2	II	NT	
AtGH3-2	At4g37390	4	II	IAA	YDK1
AtGH3-3	At2g23170	2	II	IAA	
AtGH3-4	At1g59500	1	II	IAA	
AtGH3-5	At4g27260	4	II	IAA, SA	AtGH3a/WES1
AtGH3-6	At5g54510	5	II	IAA	DFL1
AtGH3-7	At1g23160	1	III	(−)	
AtGH3-8	At5g51470	5	III	(−)	
AtGH3-9	At2g47750	2	II	IAA	
AtGH3-10	At4g03400	4	I	(−)	DFL2
AtGH3-11	At2g46370	2	I	JA	FIN219/JAR1
AtGH3-12	At5g13320	5	III	(−)	PBS3/GDG1
AtGH3-13	At5g13350	5	III	NT	
AtGH3-14	At5g13360	5	III	NT	
AtGH3-15	At5g13370	5	III	(−)	
AtGH3-16	At5g13380	5	III	NT	
AtGH3-17	At1g28130	1	II	IAA	
AtGH3-18	At1g48670	1	III	(−)	
AtGH3-19	At1g48660	1	III	(−)	
AtGH3-20/ truncated	At1g48690	1	III	(−)	

[a] Full-length GH3 family members were designated as *GH3-1–GH3-19,* and the truncated one was designated as GH3-20 (Hagen and Guilfoyle 2002; Takase et al. 2003)

[b] Substrate specificity for each GH3 protein was tested in vitro (Staswick et al. 2002)

NT, Not tested; (−), inactivity on substrate tested. Substrates tested: ABA, abscisic acid; IAA, indole-3-acetic acid; ACC, 1-aminocyclopropane-1-carboxylic acid; GA, gibberellic acid; JA, jasmonic acid; SA, salicylic acid. SA was only tested for AtGH3-5 (Staswick and Tiryaki 2004)

also identified and/or characterized (Bierfreund et al. 2004; Reddy et al. 2006). To our knowledge, Pp-GH3.16 showed the highest homology to Group II GH3 members of *Arabidopsis*, and was the only GH3 member studied in *Pinus pinaste* until now. In *Physcomitrella patens,* there were three *GH3*-like genes: *PpGH3-1*, *PpGH3-2* (belong to Group I) and a truncated gene. Phylogenetic analyses indicated that GH3 proteins are highly conserved all over the plant kingdom (Hagen and Guilfoyle 2002; Terol et al. 2006).

Promoter characteristics of *GH3* genes

The *GH3* gene of soybean is the first auxin primary/early response gene identified and its expression could be induced by exogenous auxin treatment within 5 min (Hagen and Guilfoyle 1985; Woodward and Bartel 2005). To address how *GH3* can be induced by auxin treatment, the promoter of *GH3* was analyzed in detail through gel mobility shift assays, methylation interference, deletion analysis, linker scanning, site-directed mutagenesis, and gain-of-function analysis. These analyses identified that the sequence TGTCTC is the core sequence of auxin response element (AuxRE) in *GH3* promoter (Liu et al. 1994; Ulmasov et al. 1995). This type of AuxRE and/or its variants were also found to be present in the promoters of other auxin-responsive genes. Furthermore, ARFs are able to specifically bind to the AuxREs to repress or activate expression of these genes (Ulmasov et al. 1995, 1997a, 1997b).

The auxin-responsive ability of the TGTCTC element can be enhanced by the combination with an adjacent or overlapping coupling element (such as CACGCAAT, CCTCGTGtctc). Through combination with different coupling elements, the simple AuxRE, TGTCTC, can make up three kinds of composite AuxREs. Although each shows auxin-inducible activity independently, they can contribute incrementally to the overall level of auxin induction (Liu et al. 1994; Ulmasov et al. 1995). In addition, the simple AuxRE without coupling elements may also function strongly in expression of auxin-inducible genes if the TGTCTC elements occur as tandem

direct or palindromic repeats, although this scenario has not yet been found occurring naturally. P3 (4×) consisting of four palindromic repeats spaced by 3 bp is more active than natural AuxREs in response to auxin treatment (Ulmasov et al. 1997a). DR5 (7×) element, which is composed of seven direct repeats of 11 bp fragment including the TGTCTC element, also shows greater auxin inducibility than a natural composite AuxRE and the *GH3* promoter (Ulmasov et al. 1997b). Because of their higher auxin inducibility than identified natural promoters, the DR5 (7×) and P3 (4×) constructs have been used as valuable tools to study spatial-temporal expression patterns of auxin-responsive genes in the life cycle of plants (Bierfreund et al. 2003; Hagen et al. 1991; Li et al. 1999; Schwalm et al. 2003). Nevertheless, Goda et al. (2004) found recently that TGTCTC element was not enriched in genes specifically regulated by IAA, but was enriched in genes up-regulated by both brassinolide (BL) and IAA. Therefore, these constructs may be not specific to auxin action, but can also be used as important markers for studying the brassinosteroid (BR)/auxin interaction.

Besides AuxREs, ethylene responsive element (ATTTCAAA) has also been found in promoters of *GH3* genes (Liu et al. 2005). Consistent with this, *CcGH3* (*GH3* in *Capsicum chinense* L.) was found to be regulated by both auxin and ethylene (Liu et al. 2005).

Biochemical function of the *GH3* genes

By sequence analysis and three-dimensional prediction of proteins, GH3s were found to belong to acyl adenylate-forming firefly luciferase superfamily. AtGH3-11 (JAR1, Group I) is the first demonstrated GH3 that is able to specifically catalyze adenylation of JA in vitro. It was suggested that adenylation of JA might initiate conjugation of several amino acids to JA (Staswick et al. 2002). The conjugation of JA to isoleucine (Ile) mediates JA response in *Arabidopsis*, because exogenous JA-Ile was able to rescue the defect of *jar1* in response to JA. Interestingly, the level of JA-ACC conjugates was shown to be up-regulated in *jar1-1*, suggesting that JAR1 may also play a role in crosstalk between JA and ethylene signaling (Staswick and Tiryaki 2004).

All of the Group II members (except for *GH3-1*, which needs to be studied) can catalyze adenylation of IAA in vitro (Table 1; Staswick et al. 2002, 2005). This modification converts the free IAA to a conjugated form. Via in vitro studies, IAA-Asp has been found to be the major conjugate when treated with IAA (Staswick et al. 2005). Besides IAA, AtGH3-5 (AtGH3a/WES1, Group II) can also catalyze adenylation SA in vitro (Staswick et al. 2002), indicating that AtGH3-5 is involved in crosstalk between IAA and SA signaling.

Biological functions of the *GH3* genes

Using in situ hybridization and analysis of P_{GH3}:*GUS* expression, soybean *GH3* was found to be expressed in the inner cortex and protoxylem ridges of roots. In addition, it is also transiently expressed during flower and pod development. When treated with 2,4-D (2,4-dichlorophenoxyacetic acid), *GH3* transcripts became more abundant in the vascular regions of all organs studied. Furthermore, a high level of *GH3* mRNA was also detected in developing palisade mesophyll cells of leaves, cotyledons, and flowers (Gee et al. 1991; Guilfoyle et al. 1993). All of these indicate that GH3 may be involved in auxin-regulated growth and development.

A number of *gh3* mutants have been isolated and characterized in *Arabidopsis*, and the biological function and significance of GH3s in plant growth and development have been clarified. So far, mutants of seven *Arabidopsis GH3* genes were identified via morphological screening and they displayed distinct but interrelated phenotypes (Table 2; Hsieh et al. 2000; Khan and Stone 2007; Nakazawa et al. 2001; Staswick et al. 2002; Takase et al. 2004; Takase et al. 2003; Tanaka et al. 2002; Zhang et al. 2007). *AtGH3-11*, one Group I gene, is induced by auxin *and* encodes a protein that specifically adenylates JA but not IAA or other hormones in vitro (Staswick and Tiryaki 2004; Staswick et al. 2002). There are two interesting mutant alleles identified for this locus: *jar1-1* and *fin219*. *fin219* exhibited long hypocotyl only under continuous far-red light, suggesting that FIN219 mediated signal transduction of phytochrome A (phyA). Therefore, the FIN219 may be a cross-talk junction between auxin and phyA signaling (Hsieh et al. 2000). *jar1*, a null mutant of *AtGH3-11*,

Table 2 Most notable phenotype (s) of mutants in *Arabidopsis GH3* genes

Group	Gene	Mutant (s)	Prominent phenotype (s)	References
I	*AtGH3-10*	*DFL2OX*	Shorter hypocotyl in red/blue light	Takase et al. (2003)
		DFL2AS	Longer hypocotyl in red light	Takase et al. (2003)
	AtGH3-11	*fin219*	Longer hypocotyl in far-red	Hsieh et al. (2000)
		jar1-1	Insensitivity to MeJA in root growth	Staswick et al. (2002)
II	*AtGH3-2*	*ydk1-D*	Shorter hypocotyl in light and darkness	Takase et al. (2004)
	AtGH3-5	*wes1-D*	Shorter hypocotyl in red light; reduced growth, small plant organs and enhanced stress adaptation	Park et al. (2007a, b)
		gh3.5-1D	Smaller curly rosette leaves, shortened primary roots and reduced lateral roots	Zhang et al. (2007)
	AtGH3-6	*dfl1-D*	Shorter hypocotyl in light	Nakazawa et al. (2001)
	AtGH3-9	*gh3.9-1*	Longer primary root	Khan and Stone (2007)
III	*AtGH3-12*	*pbs3-1*	Enhanced susceptibility to avirulent and virulent *P. syringae* strains	Nobuta et al. (2007)
		gdg1		Jagadeeswaran et al. (2007)

exhibited insensitivity to JA. However, *fin219* did not show insensitivity to JA and *jar1* did not display the specific far-red light long-hypocotyl phenotype (Staswick et al. 2002). The discrepancy of these mutant phenotypes is difficult to explain. Staswick et al. (2002) ascribed this to that *fin219* was an epigenetic mutant whose nature needs to be further characterized. Recently, *jar1-1* and another *jar1* allele were found to exhibit much weaker specific long-hypocotyl phenotype than *fin219* under weak continuous far-red light condition (Chen et al. 2007). This result confirms that *FIN219/JAR1/AtGH3-11* mediates far-red light response. Unfortunately, the reasons for much weaker far-red light hyposensitivity of *jar1* and the JA sensitivity of *fin219* are still obscure. To address these questions, it is important to reveal the nature of *fin219* mutant.

DFL2/AtGH3-10, another Group I gene, is a red light-induced gene and is involved in seedling photomorphogenesis. When dark-grown seedlings were exposed to red light, *DFL2* expression was up-regulated and maintained for about two hours. Meanwhile, the hypocotyl length was dependent upon the expression level of *DFL2* under red-light condition. All of these results suggest that *DFL2* is involved in red light signal transduction. Unlike other characterized *GH3*, *DFL2* expression is not induced by exogenous auxin although there are putative AuxREs in the promoter of *DFL2* (Takase et al. 2003).

In contrast to *DFL2*, all other characterized Group II members are auxin up- or down-regulated genes and some of them are also regulated by light. *AtGH3a* (*AtGH3-5*, Group II) expression is induced by auxin and by end-of-day far-red light treatment. Furthermore, *AtGH3a* (*WES1*, Group II) is involved in the shade-avoidance responses and acts downstream of phytochrome B (Park et al. 2007b; Tanaka et al. 2002). GH3-5 displays adenylation activity not only on IAA but also on SA in vitro, and *gh3* activation-tagged mutants (*wes1-D* and *gh3.5-1D*) show enhanced auxin resistance and stress adaptation (Park et al. 2007a; Staswick et al. 2002; Zhang et al. 2007). Because some *GH3s* of Group II act may redundantly, their functions were generally identified through phenotypic characterization of over-expression mutants, such as *ydk1-D* and *dfl1-D*. Both *YDK1* (*AtGH3-2*, Group II) and *DFL1* (*AtGH3-6*, Group II) are induced by auxin. *ydk1-D* displayed a short-hypocotyl phenotype in dark- and light-grown seedlings, but *dfl1-D* displayed short-hypocotyl only under light conditions. Further analyses on *YDK1* and *DFL1* expression have shown that *YDK1* is inhibited by blue and far-red light, but *DFL1* is not influenced by light. Therefore DFL1 protein should function with one or more light-induced partner (s) in regulating hypocotyls elongation (Nakazawa et al. 2001; Takase et al. 2004).

Recently, the function of *AtGH3-9* (Group II) is studied through characterization of *gh3.9-1* and its RNAi lines. Unlike most other Group II genes, *AtGH3-9* expression is down-regulated by low concentrations of exogenous IAA in seedlings. Similar to *jar1-1*,

gh3.9-1 shows moderately JA resistance, indicating that AtGH3.9 is likely to be a juncture between auxin and JA response pathway (Khan and Stone 2007). Since most members of group II proteins including AtGH3-9 have an enzymatic activity for adenylation of IAA in vitro, they may function in auxin homeostasis by reducing the availability of free auxin and they may function redundantly (Khan and Stone 2007; Staswick et al. 2002, 2005).

Our knowledge for biological roles of Group III genes still remains rudimentary (Woodward and Bartel 2005). Two latest studies suggest that AtGH3-12/PBS3/GDG1 (Group III) plays an important role in the metabolism and signal transduction of SA, which may increase stress adaptation of plants. Synthesis of SA in plant can be induced by both abiotic stress and biologic stress. The damages of the stress on plants can be alleviated by SA (Fujita et al. 2006). Salicylic acid-2-*O*-*β*-glucoside (SAG) is the conjugated and primary storage form of SA (Dean et al. 2005). Comparing with the wild type, *pbs3-1* and *gdg1-1*, two loss-of-function mutants of *AtGH3-12*, exhibited lower-level SAG in the process of pathogen infection. Meanwhile, expression of the SA-dependent pathogenesis related marker, *PR1* (pathogenesis-related protein 1, a key component in SA signaling), was down-regulated in this process, and exogenous SA application was able to restore *PR1* expression and resistance to pathogens in these mutants (Jagadeeswaran et al. 2007; Nobuta et al. 2007). Surprisingly, Jagadeeswaran et al. (2007) reported that free SA level was decreased in *gdg-1* while Nobuta et al. (2007) found that free SA level was elevated in *pbs3-1* and *pbs3-2*. The reason for this contrary result may be the different nature of mutant alleles and/or the different conditions for SA analysis. Further work, such as measuring the SA contents of *pbs3* and *gdg1* under the same condition, will be useful to resolve this discrepancy.

So far, only the function of OsGH3.8 has been revealed (Ding et al. 2008), although there are at least 12 members of GH3 family in rice (Jain et al. 2006). Similar to group II GH3 proteins in Arabidopsis, OsGH3.8 (group II) is an IAA-amino synthetase which prevents free IAA accumulation. The overexpression line of GH3-8 displayed enhanced resistance to the rice pathogen *Xanthomonas oryzae* pv oryzae and abnormal plant morphology and retarded growth and development. The mechanism underlining both abnormal development and enhanced resistance may be the inhibition of the expression of expansins, proteins that control cell wall loosening and expansion, by preventing the accumulation of free IAA (Ding et al. 2008). This discovery is helpful to understand the interaction of plant defense systems and auxin signaling.

Relationship between GH3s and ARFs

Auxin response factors (ARFs) can bind specifically to the AuxREs within promoters of early/primary auxin response genes and regulate their expression (Ulmasov et al. 1997a; Woodward and Bartel 2005). There are 23 *ARF* genes in *Arabidopsis*, and some of which may bind to GH3's promoters to regulate gene expression. ARF8 was the first ARF that was demonstrated to regulate expression of three *AtGH3* genes, *AtGH3a*, *DFL1*, and *YDK1* (Tian et al. 2004). These genes were down-regulated in *arf8-1* mutant and up-regulated in *ARF8* overexpression lines. Although free auxin level was not remarkably elevated in *arf8-1*, it was indeed decreased in *ARF8* overexpression lines. These results suggest that ARF8 might positively regulate expression of *GH3*, which resulted in adenylating IAA to form IAA-AA. This might be one of the pathways in maintaining auxins homeostasis in vivo (Tian et al. 2004). Recently, Yang et al. (2006) found the microRNA167-ARF8-GH3-IAA pathway in rice. Through analysis of *ARF17* overexpression lines by expressing a microRNA160-resistent *ARF17* mRNA, it was found that *ARF17* could negatively regulate expression of *GH3-5* and *DFL1*, but positively regulate gene expression of *GH3-2* and *YDK1* (Mallory et al. 2005). In contrast to *ARF17*, a mutation in *ARF7* causes reduced expression of some *GH3* genes including *YDK1*, suggesting that *ARF7* also positively regulates *YDK1* expression (Stowe-Evans et al. 1998; Takase et al. 2004).

Conclusions

Our understanding of biological functions of *GH3* genes has advanced rapidly in recent years. Together with the progresses in functional studies on *ARF*, *Aux/IAA* and *SAUR* genes, an outline of primary/early auxin response pathways was revealed. Phenotype

characterizations of the mutant lines indicated that GH3s are involved in different growth and developmental processes. These analyses also provided evidences to identify the biochemical function of GH3. Although *GH3* transcripts were first identified from auxin-treated seedlings of soybean, the finding that GH3s belong to acyl adenylate-forming firefly luciferase superfamily and can catalyze adenylation of IAA, JA and SA suggests that GH3s play a role not only in auxin signaling but also in other signal transduction pathways. Some *GH3* genes are also regulated by light. Hence GH3 proteins may be key linkers among different signal transduction pathways, although their physiological functions need to be further studied. However our knowledge about GH3s mainly focus on group I and II, the little is known about the group III, especially in terms of their biochemical activities. Further understanding on the function of the GH3s will help to elucidate the complex signal transduction network in plants.

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