УДК 575.22+577.218

A. Navalikhina, M. Antonyuk, T. Ternovska

GENETIC BASIS OF AWNEDNESS IN GRASSES

Some representatives of grasses, such as rice, barley, oats, and wheat, have long needle-like elongations of lemma – awns. Genetic mechanisms of awns development control remain poorly understood. This article give a literature review on the awnedness control in rice, barley, and wheat. Due to these species relationship and evolutionary conservatism of developmental mechanisms, it is possible to apply principle of synteny to transfer existing data on rice and barley to a wheat. Thus, it is possible to find out the mechanisms of awns development and discovery the corresponding genes in wheat, which is important and necessary task.

Keywords: cereal, wheat, awns, wheat, awnedness genetic control, homeotic genes.

Grasses have a specific type of flower with the structure that is typical for this taxon. Flower of grasses is composed of pistil, stamens, and lodicules (homologues of petals). Flower is covered with outer structures – lemma and palea. Several flowers constitute spikelet, covered with spikelet glumes. Spikelets form a spike (like in wheat) or panicle (like in rice).

Some representatives of grasses (such as rice, barley, oats and wheat) have long needle-like elongations of lemma – awns [1; 2]. Awns play important roles in transpiration, photosynthesis, protection against animals, grain distribution, and anchoring of seeds to the soil [3]. It is believed that the awn of grasses is homologous to the leaf [1; 4; 5].

Development of the awns is controlled by many genes. For example, rice have 20 known quantative trait loci (QTL), which are mapped to the 3, 4, 6, 7, 8, 9, 10, 11 and 12 chromosomes, that control the length of the awns [39]. Despite the large amount of data on QTLs localization, little is known about specific genes and molecular mechanisms that control the development of awns.

For today, most of the accumulated data on genetics of awns refers to rice. Most of the studied genes that control the development of awns in rice (*SHL2, SHO1, SHO2, WAF1*) encode proteins involved in TAS3 ta-siRNA (trans-acting small interfering RNA) regulatory pathway; another one (*OsETT2*) is regulated by this pathway (Fig. 1).

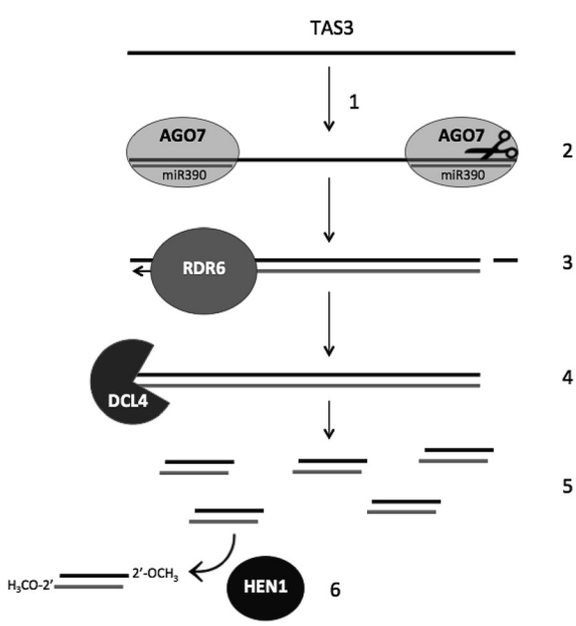

Fig. 1. Biogenesis of ta-siRNA from TAS3 predecessor. Corresponding protein names of rice are given in brackets [9–11]:

1 – transcription of the TAS3 gene;

2 – cutting of the TAS3 transcript by ARGONAUTE7 (SHO2) protein in the complex with the miR390 RNA that provides specificity of recognition;

3 – synthesis of double-stranded RNA by RDR6 RNA-dependent RNA polymerase 6 (SHL2);

4 – long double-stranded RNA is cut by DICER-LIKE4 (SHO1);

5 – mature 21-nt ta-siRNAs;

6 – methylation of ta-siRNA by HEN-1 methyltransferase (WAF1)

Table. **The genes involved in the genetic control of awnedness in some grasses**

Gene/locus	Source	Chromosome	Functions	Reference
AN1	*Oryza sativa*	4	- activation of cells division - awn formation - elongation of grains - reduction of grain number in panicle	[6]
DL		3	- activation of meristem cells division - awn formation - leaf midrib formation - adaxial-abaxial leaf polarization - regulation of floral organs development	[1; 7]
OsETT2		1	- lemma growth activation - abaxialization of awn primordium - abaxialialization of leaf	[1]
TOB1		4	- activation of meristem cells division - formation and growth of lemma and palea - adaxial-abaxial leaf polarization - organization of floral meristem and shoot apical meristem	[8]
SHL2		1	- synthesis of ta-siRNAs in TAS3 pathway (OsRDR6 RNA polymerase) - initiation and maintenance of shoot apical meristem - adaxial-abaxial leaf specification - juvenile to adult leaf transition - lateral roots growth	[9]
SHO1		4	- synthesis of ta-siRNAs in TAS3 pathway (DICER-LIKE4)	[10]
SHO2		-	- synthesis of ta-siRNAs in TAS3 pathway (ARGONAUTE7)	[10]
WAF1		7	- synthesis of ta-siRNAs in TAS3 pathway (WAF1 RNA methyltransferase)	[11]
HvKNOX3	*Hordeum vulgare*	4H	- initiation and maintenance of meristems	[12]
SuKD *SuKB* *SuKE* *SuKF*		5H 7H 7H 7H	- supression of *hooded* phenotype	[13]
Lks2		7H	- awn length control - pistil morphology control	[14]
Hd	*Triticum aestivum*	4AS	- dominant supressor of awnedness	[15]
B1		5AL	- dominant supressor of awnedness	[15]
B2		6BL	- dominant supressor of awnedness	[15]
Wknox1a *Wknox1b* *Wknox1d*		4AS 4BS 4DS	- initiation and maintenance of meristems	[16; 17]

The list of genes involved in the genetic control of awn development in some grasses representatives is given in Table.

The main gene involved in the regulation of awns in rice is *An-1* (*Awn-1*). This gene is localized on 4 chromosome, and encodes bHLH (basic helix-loop-helix) transcription factor. *An-1* is expressed in the apex of the lemma primordia. The presence of its product, protein AN-1, promotes cell division and the formation of awn. Upregulation of *An-1* expression leads to elongation of awns and grains. It also causes reduction of the grains number in the panicle. In this regard, it is believed that *An-1* was one of the key target genes in rice artificial selection [6].

TOB1 (*TONGARI-BOUSHI 1*) and *DL* (*DROOPING LEAF*) from YABBY gene family are also involved in the regulation of awn formation in rice. YABBY genes encode transcription factors, which have YABBY domain with two conserved motives – helix-loop-helix and zinc fingers. These genes play an important role in the regulation of various processes in plants, such as adaxial-abaxial polarization of leaf, development of flower organs, and maintenance of meristems. YABBY proteins act indirectly on the meristem. These genes are expressed in lateral organs and their primordia, where certain secondary signal is produced as a result of YABBY proteins action. Thereafter, this signal enters the meristem cells and activates their division [1; 8; 18–20].

Rice *DROOPING LEAF* gene have a key role in the regulation of floral organs development. It is also necessary for the formation of awns and leaf midrib. Mutants for *DL1* gene have drooping leaves, and extra stamens that are formed instead of pistil.

The *dl1* mutants of *Oryza sativa* ssp. *indica* awned subspecies have no awns. The gene is expressed in carpel, cells of the central region of leaf primordium, and in the lemma under the awn primordium. It indirectly initiates division of awn primordium cells, causing the elongation of awn [1; 7]. Activity of *DL* is not sufficient for the formation of awns. It requires another gene – *OsETT2* from ARF gene family, which is orthologous to *ETTIN (ETT)/ AUXIN RESPONSE FACTOR3 (ARF3)* gene of *Arabidopsis thaliana*. In addition to leaf abaxialization and activation of lemma growth, *OsETT2* is nesessary for abaxialization of awn primordium. In this pair of genes, *DL* activates cell proliferation in awn primordioum and *OsETT2* specifies these cells so that the awn takes its native morphology.

TONGARI-BOUSHI 1 is the second gene from a YABBY gene family that is responsible for the formation of awns in rice. *TOB1* gene is localized on chromosome 4, and encodes OsYABBY5 transcription factor. *TOB1* is expressed in the primordia of lateral spike organs and in the primordia of leaves. Through indirect activation of cell division, OsYABBY5 initiates the formation and growth of the lemma and palea, and provides activity and organization of floral meristem and shoot apical meristem.

Mutations in the *TOB1* gene lead to the formation of several phenotypes, among which five major classes can be distinguished:

I. formation of the awn on the lemma;

II. reduction of the palea;

III. reduction of the lemma;

IV. formation of a cone-shaped organ from the lemma;

V. absence of flower due to the loss of the floral meristem activity.

In mutants of II, IV and V classes awns can be formed on the lemma, as well as in the plants from I class (Fig. 2).

TOB1 gene regulates expression of *OSH1* (*ORYZA SATIVA HOMEOBOX1*) gene from the KNOX1 family, which is homologous to the *Bkn3* gene of barley and *Wknox1* gene of wheat. Mutations in these genes are associated with the awnless *hooded* phenotype. *OSH1* gene serves as a marker of indeterminate cells in rice. Level of its expression is often measured in order to determine the condition of meristem. In *tob1* mutants *OSH1* is not expressed in floral meristem cells. This cause inactivation of the flower meristem and flower loss [8; 20].

The role of small RNAs in the regulation of awns formation has been shown in the studies of *OsETT2* gene function, transcription of which is regulated by TAS3 pathway. This was also confirmed by research data on *O. sativa* ssp. *japonica* mutants for genes involved in the biogenesis ta-siRNA, which displayed awned phenotype in contrast to awnless wild-type plants [1; 9–11; 21–24].

SHOOTLESS2 (*SHL2*) gene, which is localized on chromosome 1, is known to be involved in the processes of shoot apical meristem initiation and support, carpel and leaf development. The gene encodes the RNA-dependent RNA polymerase 6 (OsRDR6), which operates in the synthesis of small interfering RNAs precursors from TAS3 precursor in ta-siRNA gene expression regulation pathway (Fig. 1). The TAS3 pathway regulates expression of ARF protein family genes, in particular, *OsETT3* (ortholog of *Arabidopsis ETT/ARF4* gene) and the above-mentioned *OsETT2*, and also HD-ZIPIII protein family genes (including *OSHB1*) [9; 23]. TAS3 pathway regulates processes of adaxial-abaxial leaves specification, coordination of juvenile to adult leaf transition, and lateral roots growth [23].

In plants that have strong mutant allele *shl2*, shoot apical meristem does not develop at all. Weak mutant alleles cause defects in leaves and lemma abaxial-adaxial polarization, which result in theadlike appearance of these organs [23].

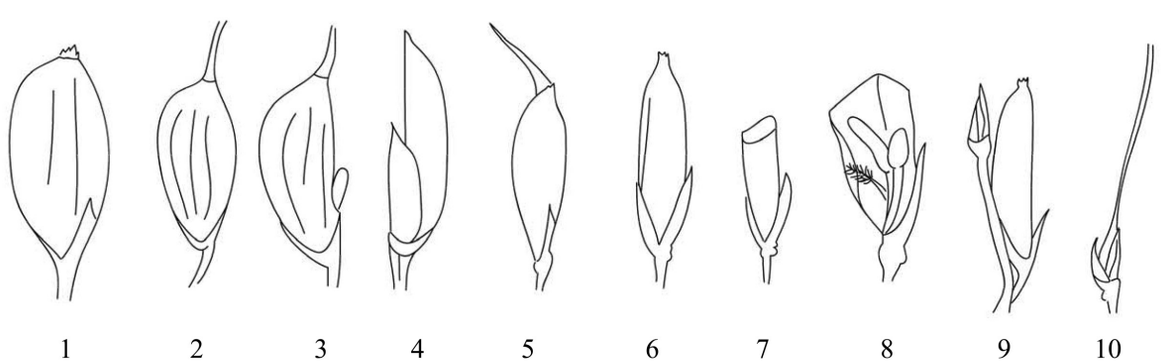

Fig. 2. Spikelets of rice mutants for TOB1 gene [7]: *1* – wild type; *2* – formation of awns on lemma; *3* – reduction of palea, awn is formed on lemma; *4* – reduction of lemma; *5, 6, 7, 8* – cone-shaped organ with awn (5), and without awn (6), in section (7, 8); *9, 10* – sterile lemma without awn (9), with awn (10)

Mutant alleles of *SHL2* may also demonstrate another phenotypes such as changes in the leaves morphology (wide leaves, splitting leaves, small leaves size), and abnormal nature of its location (formation of leaf primordum instead of shoot apical meristem). Region of expression of indeterminate cells marker *OSH1* in the shoot apical meristem of these mutants is smaller compared with the wild-type. This indicates decreased activity of meristem, which often cause arrest of plant growth before the reproductive phase [9].

SHOOT ORGANIZATION1 (*SHO1*) and *SHOOT ORGANIZATION2* (*SHO2*) genes encode DICER-LIKE4 and ARGONAUTE7 proteins, respectively. They are both involved in the same ta-siRNA gene expression regulation pathway along with SHL2: ARGONAUTE7 cuts long TAS3 transcript, which is then converted into double-stranded RNA by SHL2, and DICER-LIKE4 cuts double-stranded RNA into the 21-nt ta-siRNAs (Fig. 1).

Mutants for *sho1* and *sho2* genes are phenocopies of *shl2* mutants. They have altered leaves and lemma morphology (thread-like lemma or formation of awn-like sprouts) (Fig. 3). They also have smaller region of *OSH1* expression in the shoot apical meristem and reduced proportion of indeterminate cells [10; 22; 24]. In contrast to *shl2*, in *sho1* and *sho2* mutants shoot apical meristem develops, and most plants with mutant alleles continue their growth in the reproductive phase. Mutation in *SHO2* is hypostatic to *shl2*, which indicates that these two genes are connected in common regulatory pathway, that is likely to be TAS3 pathway [9]. Furthermore, the *SHO2* gene is associated with *DL*, as in most double mutant *sho2;dl* plants phenotype is almost normal. Double mutant plants have normal leaf form, non-misshapen lemma, and no awns. It is not known whether *SHO2* and *DL* interact in rice. But in *Arabidopsis* it was shown that *ETT/ARF4* genes (orthologues of *OsETT3*) positively regulate the expression of *FIL* gene that encodes a transcription factor from YABBY family (to which *DL* belongs). *OsETT3*, as was mentioned, is one of the genes, the expression of which is regulated by ta-siRNAs synthesized in the pathway in which SHO2 is involved [24].

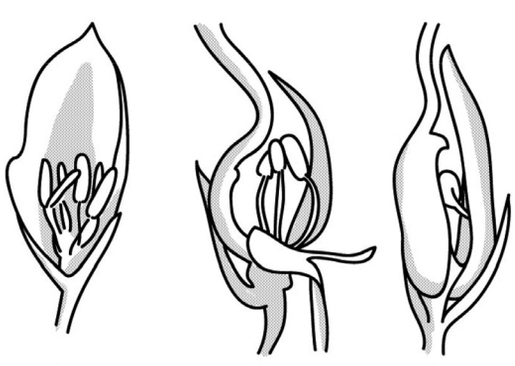

Fig. 3. Spikelets of rice mutant for SHO2 gene [24]:
1 – wild-type;
2 – thread-like lemma, reduced palea;
3 – phenotype is close to wild-type, formation of awn on the lemma

WAF1 (*WAVY LEAF1*) gene encodes RNA methyltransferase, which is homologous to *A. thaliana* HEN1. WAF1 RNA methyltransferase is necessary for stabilization of mature ta-siRNAs by methylation of the 3' terminal nucleotide in the TAS3 pathway (Fig. 1). Mutants for *WAF1* gene are phenocopies of *sho1* and *sho2* mutants [11].

Barley is a major model organism for studying the genetics of awnedness. Wild type plants are awned, and for today a lot of mutants are known with changes in the awns phenotype and/or defects in their development: *adp1 (Awned palea 1), Lks1 (Awnless 1), cal-a.3 (Calcaroides-a), Kap1.e (Elevated hood), Kap1 (Hooded lemma 1), mk (Hooded/branched), vrs1.c (Lateral lemma appendix reduced*), *lel1 (Leafy lemma* 1*), Lks2 (Long awn 2), lep-e.1 (elongated outer glume 1), rcc (Rococò* spike*), Raw/Srh1 (Rough awn/long rachilla hair 1), lks2 (*Short awn 2*), raw1 (Smooth awn 1), tri.20 (Triaristatum.20), trp1 (Triple awned lemma 1)* [25]. Most of these mutants are characterized only by phenotype, and only for some of them genes and molecular mechanisms that are responsible for the development of this particular phenotype are known.

One of the best described gene, the function of which is associated with awnedness, is *HvKNOX3* (*BKn3*, *K*) from a KNOX1 gene family, homologous of maize *KN-1*. Duplication of 305 bp fragment in the fourth intron of *HvKNOX3* cause appearance of the *Hooded* barley phenotype – the substitution of awn by the extra flower of inverse polarity, which is called the "hood" (Fig. 4). This mutation is dominant, it causes overexpression of *HvKNOX3* in lemma [12]. *HvKNOX3* promoter sequence and fourth intron contain $(GA)_8$ repeats in their sequences. These repeats function as the binding sites for nuclear protein BBR (Barley B Recombinant). BBR activates expression of genes that have $(GA)_8$ repeats in their regulatory sequences. Since fourth intron of *HvKNOX3* contains such repeat, this means that it has a regulatory function. Duplication of binding site for expression activator (BBR) in this intron may lead to excessive activation of *HvKNOX3* expression. It is also possible, that duplication breaks another regulatory sequence in the fourth intron – PHO site silencer, which may

lead to inactivation of this silencer function, and cause overexpression of *HvKNOX3* [26].

In order to find out which of the two mechanisms of *HvKNOX3* overexpression in barley mutants lemma is actually implemented, the study of genes that are epistatic to *Hooded* was conducted. These include mutant loci of *suK* series (supressor of K) and *lks2* gene [27]. It can be assumed, that the mutant product of *suK* or *lks2* gene regulates expression through the binding to *HvKNOX3* fourth intron sequence.

Fig. 4. Flower of barley mutant for HvKNOX3 gene [12]: *1* – wild-type; *2* – Hooded phenotype

SuK loci are mapped on the 5H (*suKD*) and 7H (*suKB*, *suKC*, *suKE*, and *suKF*) chromosomes. Plants with recessive (mutant) *suK* alleles and the dominant *K* mutation have shorter than wild-type awns, and no "hood" is formed. It is believed that *suK* loci do not have their own phenotypic display, they function only as suppressors of ectopic flower formation caused by the action of *K*, and shortening of the awns is a manifestation of the dominant *K* mutation [28].

Lks2 (*short awn 2*) gene, localized on 7H chromosome, encodes the transcription factor of SHI (SHORT INTERNODES) family [14]. In barley, *Lks2* controls the length of the awns and morphology of the pistil. In *A. thaliana*, transcription factors of this family regulate auxin biosynthesis and function in many important processes, such as leaf and flower development [29; 30]. The product of *Lks2* has RING-like motif "zinc fingers" and IGGH domain. The mutations in the sequence encoding IGGH domain are recessive; in homozygous state they cause shortening of awns in half. In plants with mutation in *K* (*Hooded*) and recessive mutation in *Lks2* (genotype *lks2;K*) "hood" does not develop and shortened awn is formed instead [14].

In other barley mutants with defects in awn development, only localization of genes or loci mutations in which are linked to the corresponding phenotype is determined. Loci *calcaroides* (*cal*) are mapped on 3H and 7H chromosomes, locus *subjacent hood* (*sbk*) is localized on chromosome 2H. Phenotype of *cal* and *sbk* mutants is similar: terminally-awned ectopic structures, called "sac", are formed on the lemma [31]. Severity of *cal* mutations display varies. Depending on the mutant alleles, other structures can be formed on the lemma – "wings" or "knee" [30]. In *leafy lemma (lel)* mutants leaf is formed on the lemma instead of the awn. Locus *lel* is localized on 5H chromosome. This homeotic mutation may point to the fact that awn and leaf are homologous organs in grasses [5]. Another gene, associated with a defect in the development of awns is *Lks1*, mapped to chromosome 2H. Plants with dominant allele of this gene in the homozygous state are awnless. Heterozygotes may be awned or awnless depending upon the source of alleles and the genetic background [31].

In bread wheat *Triticum aestivum* three loci that control the development of awns are known: *Hd* (4AS), *B1* (5AL), and *B2* (6BL). These loci contain genes that are dominant suppressors of awnedness. Homozygotes by three recessive alleles *hd*, *b1,* and *b2* are awned. The presence of one supressor dominant allele inhibits the development of awns to some extent. Plants with dominant alleles of two supressor genes are awnless [33; 34]. Mutation *Hd* in wheat is analogous to barley *Hooded* mutation. Wheat plants with this mutation have shortened and twisted awns [17]. Attempts to identify a gene, mutation in which is associated with *Hd*, have led to discovery of *HvKNOX3* homologues – *Wknox1a*, *Wknox1b*, and *Wknox1d*. Wknox1 genes belong to a family of KNOX class I genes. Like other members of this family, they have a large intron, containing regulatory sequences, which regulates the level and tissue specificity of expression. Mutations in this intron cause an increase in expression level, or the ectopic expression of the gene. *Wknox1* genes are localized on the fourth homoeologic group of chromosomes. *Wknox1a* gene, localized on chromosome 4A, has been identified as a candidate gene for *Hd*. In the *Hd* near-isogenic lines, *Wknox1a* gene was found to be ectopically expressed in the lemma. Regulatory fourth intron of the *Wknox1a* has numerous MITE transposon insertions, duplications, deletions and SNPs that could trigger *hooded* phenotype. However, none of these mutations are localized in the regulatory sequences of intron (PHO silencer and BBR-binding repeat),

and therefore, they can not cause changes in the expression of *Wknox1* [16; 17]. Search for candidate genes for *B1* and *B2* still continues.

Another three potential awnless suppressor loci in wheat were identified. Since nullisomics on 3D chromosome and ditelosomics for 3AS and 6DL arms are awned, an assumption was made about localization of awnedness supressors on the 3D, 3AL, and 6DS chromosomes [34].

Little is known about the awnedness promoters genes in wheat. There is evidence on the series of such genes localized on the second homoeologous chromosome group, because in disomic-added lines for 2A, 2B or 2D chromosome of awnless wheat Chinese Spring (2n = 44) awns are formed [34–36].

It should be noted, that from all these genes that control the development of awns in wheat, the official McIntosh catalog of gene symbols indicate only three: *hd*, *b1* and *b2*. As for the others, today there is still no sufficient evidence regarding their existence, and there are no symbols for these genes [37].

For a long time it was believed that genetic control of awnedness in grasses is simple and involve only a few genes [15]. However, now it is evident, that development of awns requires many genes most of which are homeotic. Wheat *hd*, *b1* and *b2* genes were discovered in the 40s of XX century [38]. These were the first and currently the only more or less studied genes that are involved in the development of awns in wheat. The molecular nature of any of them is still not known. For today, it is possible and necessary to find genes that provide the development of awns in wheat, using available data for rice and barley, and applying the synteny principle.

Acknowledgements

We thank Bakan Olha for assistance with illustrations that greatly improved the manuscript.

References

1. Toriba T. The DROOPING LEAF and OsETTIN2 genes promote awn development in rice / T. Toriba, H.-Y. Hirano // Plant J. – 2014. – Vol. 17, № 4. – P. 616–626.
2. Prokopyk D. O. Homeotic genes and their role in development of morphological traits in wheat / D. O. Prokopyk, T. K. Ternovska // Cytol. Genet. – 2011. – Vol. 45, № 1. – P. 41–54.
3. Li X.-F. Awn Anatomy of Common Wheat (*Triticum aestivum* L.) and its Relatives / X.-F. Li, D. Bin, W. Hong-gang // Caryologia. – 2010. – Vol. 63, № 4. – P. 391–397.
4. The role of wheat awns in the seed dispersal unit / R. Elbaum, L. Zaltzman, I. Burgert, P. Fratzl // Science. – 2007. – Vol. 316, № 5825. – P. 884–886.
5. Thomas W. Grass Systematics and Evolution / W. Thomas ; ed. by T. R. Soderstrom, K. W. Hilu, C. S. Campbell, M. E. Barkworth // Brittonia. – 1988. – Vol. 40, № 3. – P. 293.
6. *An-1* encodes a basic helix-loop-helix protein that regulates awn development, grain size, and grain number in rice / J. Luo, H. Liu, T. Zhou [et al.] // Plant Cell. – 2013. – Vol. 25, № 9. – P. 3360–3376.
7. The YABBY gene DROOPING LEAF regulates carpel specification and midrib development in *Oryza sativa* / T. Yamaguchi, N. Nagasawa, S. Kawasaki [et al.] // Plant Cell. – 2004. – Vol. 16, № 2. – P. 500–509.
8. The YABBY Gene TONGARI-BOUSHI1 Is Involved in Lateral Organ Development and Maintenance of Meristem Organization in the Rice Spikelet / W. Tanaka, T. Toriba, Y. Ohmori [et al.] // Plant Cell. – 2012. – Vol. 24, № 1. – P. 80–95.
9. Satoh N. The SHOOTLESS2 and SHOOTLESS1 genes are involved in both initiation and maintenance of the shoot apical meristem through regulating the number of indeterminate cells / N. Satoh, J. Itoh, Y. Nagato // Genetics. – 2003. – Vol. 164, № 1. – P. 335–346.
10. Shoot organization genes regulate shoot apical meristem organization and the pattern of leaf primordium initiation in rice / J. I. Itoh, H. Kitano, M. Matsuoka, Y. Nagato // Plant Cell. – 2000. – Vol. 12, № 11. – P. 2161–2174.
11. WAVY LEAF1, an ortholog of Arabidopsis HEN1, regulates shoot development by maintaining MicroRNA and trans-acting small interfering RNA accumulation in rice / M. Abe, T. Yoshikawa, M. Nosaka [et al.] // Plant Physiol. – 2010. – Vol. 154, № 3. – P. 1335–1346.
12. The barley Hooded mutation caused by a duplication in a homeobox gene intron / K. J. Müller, N. Romano, O. Gerstner [et al.] // Nature. – 1995. – Vol. 374, № 6524. – P. 727–730.
13. Genetics of barley Hooded suppression / C. Roig, C. Pozzi, L. Santi [et al.] // Genetics. – 2004. – Vol. 167, № 1. – P. 439–448.
14. A SHORT INTERNODES (SHI) family transcription factor gene regulates awn elongation and pistil morphology in barley / T. Yuo, Y. Yamashita, H. Kanamori [et al.] // J. Exp. Bot. – 2012. – Vol. 63, № 14. – P. 5223–5232.
15. Molecular and physical mapping of genes affecting awning in wheat / P. Sourdille, T. Cadalen, G. Gay [et al.] // Plant Breed. – 2002. – Vol. 121, № 4. – P. 320–324.
16. Intragenic diversity and functional conservation of the three homoeologous loci of the KN1-type homeobox gene *Wknox1* in common wheat / R. Morimoto, T. Kosugi, C. Nakamura, S. Takumi // Plant Mol. Biol. – 2005. – Vol. 57, № 6. – P. 907–924.
17. Molecular cloning of three homoeologous cDNAs encoding orthologs of the maize KNOTTED1 homeobox protein from young spikes of hexaploid wheat / S. Takumi, T. Kosugi, K. Murai [et al.] // Gene. – 2000. – Vol. 249, № 1–2. – P. 171–181.
18. Molecular characterization the YABBY gene family in *Oryza sativa* and expression analysis of OsYABBY1 / T. Toriba, K. Harada, A. Takamura [et al.] // Mol. Genet. Genomics. – 2007. – Vol. 277, № 5. – P. 457–468.
19. Members of the YABBY gene family specify abaxial cell fate in *Arabidopsis* / K. R. Siegfried, Y. Eshed, S. F. Baum [et al.] // Development. – 1999. – Vol. 126, № 18. – P. 4117–4128.
20. Grass meristems II: Inflorescence architecture, flower development and meristem fate / W. Tanaka, M. Pautler, D. Jackson, H.-Y. Hirano // Plant Cell Physiol. – 2013. – Vol. 54, № 3. – P. 313–324.
21. Cytoplasmic Arabidopsis AGO7 accumulates in membrane-associated siRNA bodies and is required for ta-siRNA biogenesis / V. Jouannet, A. B. Moreno, T. Elmayan [et al.] // EMBO J. – 2012. – Vol. 31, № 7. – P. 1704–1713.
22. *Oryza sativa* dicer-like4 reveals a key role for small interfering RNA silencing in plant development / B. Liu, Z. Chen, X. Song [et al.] // Plant Cell. – 2007. – Vol. 19, № 9. – P. 2705–2718.
23. Rice RNA-dependent RNA polymerase 6 acts in small RNA biogenesis and spikelet development / X. Song, D. Wang, L. Ma [et al.] // Plant J. – 2012. – Vol. 71, № 3. – P. 378–389.

24. Itoh J. I. The shoot organization2 gene coordinates leaf domain development along the central-marginal axis in rice / J. I. Itoh, Y. Sato, Y. Nagato // Plant Cell Physiol. – 2008. – Vol. 49, № 8. – P. 1226–1236.
25. Genome-Wide Association Mapping Identifies Disease-Resistance QTLs in Barley Germplasm from Latin America / L. Gutiérrez, N. M. Berberian, F. Capettini [et al.] // Zhang G. Advance in Barley Sciences : Proc. 11th Int. Barley Genet. Symp. / G. Zhang, C. Li, X. Liu. – Netherlands : Springer, 2013. – P. 209–215.
26. The GA octodinucleotide repeat binding factor BBR participates in the transcriptional regulation of the homeobox gene *Bkn3* / L. Santi, Y. Wang, M. R. Stile [et al.] // Plant J. – 2003. – Vol. 34, № 6. – P. 813–826.
27. Molecular analysis of homeotic genes involved in barley development / K. Müller, C. Pozzi, J. Müller [et al.] // Pflugers Arch. – 2000. – Vol. 439, № 3 Suppl. – P. R14–R15.
28. Genetics of Barley Hooded Suppression / C. Roig, C. Pozzi, L. Santi [et al.] // Genetics. – 2004. – Vol. 167, № 1. – P. 439–448.
29. SHORT INTERNODES/STYLISH genes, regulators of auxin biosynthesis, are involved in leaf vein development in *Arabidopsis thaliana* / T. Baylis, I. Cierlik, E. Sundberg, J. Mattsson // New Phytol. – 2013. – Vol. 197, № 3. – P. 737–750.
30. The *Arabidopsis thaliana* STYLISH1 protein acts as a transcriptional activator regulating auxin biosynthesis / D. M. Eklund, V. Ståldal, I. Valsecchi [et al.] // Plant Cell. – 2010. – Vol. 22, № 2. – P. 349–363.
31. Chapter 14 Barley diversity – an outlook / K. Sato, R. von Bothmer, T. van Hintum, H. Knüpffer // Diversity in barley (*Hordeum vulgare*). – 2003. – Vol. 7. – P. 269–278.
32. Genetics of Mutations Affecting the Development of a Barley Floral Bract / C. Pozzi, P. Faccioli, V. Terzi [et al.] // Genetics. – 2000. – Vol. 154, № 3. – P. 1335–1346.
33. Identification of the genes promoting awnedness in the *Triticum aestivum/Aegilops umbellulata* introgressive line / M. Z. Antonyuk, D. O. Prokopyk, V. S. Martynenko, T. K. Ternovska // Cytol. Genet. – 2012. – Vol. 46, № 3. – P. 136–143.
34. Sood S. Molecular characterization of threshability genes in wheat / S. Sood // Comp. Gen. Pharmacol. – 2008.
35. Friebe B. R. Development and indentification of a complete set of *Triticum aestivum – Aegilops geniculata* chromosome addition lines / B. R. Friebe, N. A. Tuleen, B. S. Gill // Genome. – 1999. – Vol. 42, № 3. – P. 374–380.
36. Dvorak J. Homoeology between *Agropyron elongatum* chromosomes and *Triticum aestivum* chromosomes / J. Dvorak // Can. J. Genet. Cytol. – 1980. – Vol. 22, № 2. – P. 237–259.
37. Catalogue of gene symbols for wheat / R. McIntosh, Y. Yamazaki, J. Dubcovsky [et al.] // 11th Int. Wheat Genet. Symp. – Australia, Brisbane, 2008. – P. 1–197.
38. Watkins A. E. Variation and genetics of the awn in *Triticum* / A. E. Watkins, S. Ellerton // J. Genet. – 1940. – Vol. 40, № 1–2. – P. 243–270.
39. Gramene: A comparative resource for plants [Electronic resource]. – Mode of access: www.gramene.org.

Наваліхіна А. Г., Антонюк М. З., Терновська Т. К.

ГЕНЕТИЧНЕ ПІДҐРУНТЯ РОЗВИТКУ ОСТЕЙ У ЗЛАКОВИХ

Такі представники злаків, як рис, ячмінь, овес і пшениця, мають довгі стрільчасті видовження леми – ості. Генетичні механізми контролю розвитку цих утворень досі залишаються мало дослідженими. У статті наведено огляд літератури щодо контролю остистості в трьох представників злаків: рису, ячменю та пшениці. Спорідненість цих видів та еволюційна консервативність механізмів розвитку дозволяє застосовувати принцип синтенії для перенесення наявних даних щодо рису і ячменю на пшеницю. Таким чином, стає можливим з'ясування механізмів розвитку остей та відкриття відповідних генів у пшениці, що є важливим і необхідним завданням для спеціальної генетики пшениці.

Ключові слова: злакові, пшениця, ості, генетичний контроль остистості, гомеотичні гени.

Матеріал надійшов 05.10.2015