

Please read carefully the text, observe attentively the figures, and provide your answers with clear, precise, and complete sentences, meaning with subject, verb, and object. Critical values of the Chi-square distribution and a table of genetic code are provided at the end of the questions.

- (20 points) There are two pure-breeding lines of lentils: one with white seed color and the other with brown seed color. All of the F1 plants from the cross of these two pure-breeding lines have tan seed color. From a single self-pollinated F1 plants, the F2 progenies set seeds with three different colors – white, brown, and tan. For 160 F2 progenies, the counts of white, tan, and brown colors equal to 42, 82, and 36, respectively. While the F3 progenies derived from all of the white-seed F2 individuals keep the white seed color, some of the F3 progenies derived from the brown-seed F2 individuals have both brown and white color seeds. Please describe the inheritance model of the seed color in this cross of lentils. You need to give proper symbols, their genetic functions, and their attached phenotypes, and to give reasonable explanations of your model based on the Mendel's principle of inheritance and the test of Chi-square statistics.
- (15 points) Harold Flor is the scientist who built the gene-for-gene theory for diseases resistance using flax and rust (*Melampsora lini*). During his investigations, he screened a great number of flax varieties that were resistant to different rust races. For the varieties that revealed resistance, he used 12 self-pollinated progenies to confirm the resistance. Given the situation that the disease resistance is monogenic inheritance, please figure out whether Flor's strategy is workable to confirm the disease resistance. Was 12 self-pollinated progenies too many or insufficient for the confirmation test?
- (15 points) In maize trisomics,  $n+1$  pollen is not viable. If a dominant allele at the *B* locus produces purple color instead of the recessive phenotype bronze and a *Bbb* trisomic plant is pollinated by a *BBb* plant, what proportion of the progeny produced will be trisomic and have a bronze phenotype?
- Based on excerpts of the abstract and results from Garcia et al. (2017) Maize defective kernel mutant generated by insertion of a Ds element in a gene encoding a highly conserved TTI2 cochaperone. PNAS 114:5165, please answer related questions.

[Excerpts of the abstract]

We have used the newly engineered transposable element *Dsg* to tag a gene that gives rise to a defective kernel (*dek*) phenotype. *Dsg* requires the autonomous element *Ac* for transposition. Upon excision, it leaves a short DNA footprint that can create in-frame and frameshift insertions in coding sequences. Therefore, we could create alleles of the tagged gene that confirmed causation of the *dek* phenotype by the *Dsg* insertion.

- (10 points) The authors used the well-known *Ac/Ds* transposable elements to create mutants for genetic analysis. Please describe what *Ac* and *Ds* is, respectively, and how they work together to create genomic variation, including the possible mechanisms creating frameshift insertions.
- (4 points) With the help of a diagram, explain what a frameshift mutation is.
- dek* mutant shows visible kernel defectiveness compared to wild-type maize (Figure 1). The *Ac/Ds* system used by the authors allowed them to identify the mutated genes, GRMZM2G048851, which encoded a TTI2 protein (Figure 2A). TTI2 interacts with TEL2 and TTI1 to form a protein complex that regulates the cellular levels of a kinase complex, formed by ZmTOR and ZmATM.

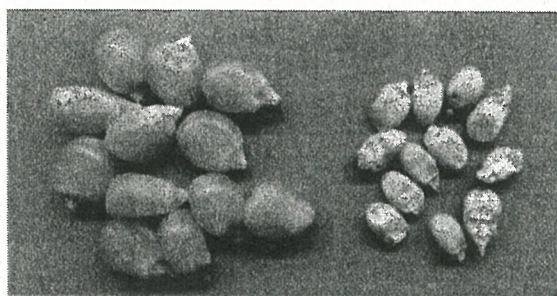


Figure 1. Wild-type maize kernels (left) vs. kernels showing *dek* phenotype (right).

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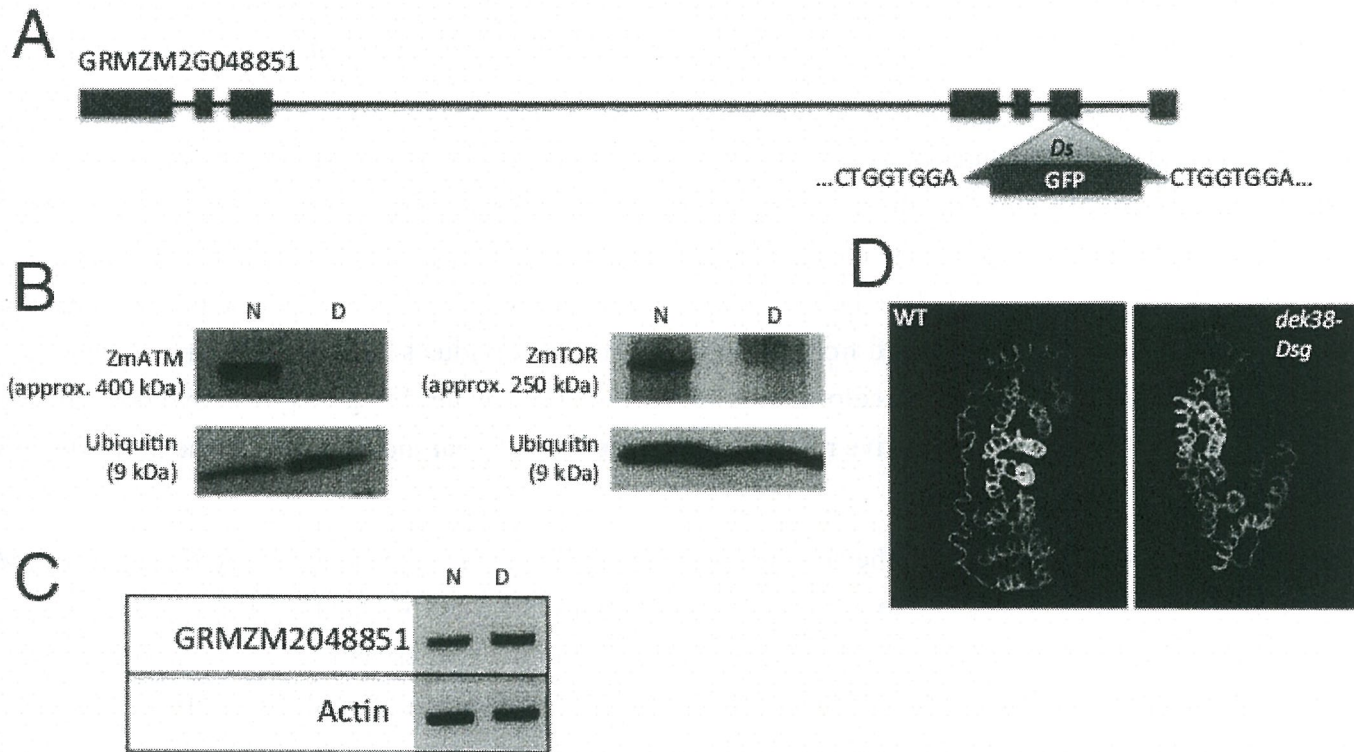


Figure 2. (A) The *Ds* is inserted in GRMZM2G048851. (B) Western blots comparing ZmATM and ZmTOR protein levels between normal (N) and *dek* (D) kernels. (C) Gene expression of GRMZM2048851 between wild-type and *dek* using mRNA extracted from immature seeds. (D) Predicted protein folding of ZmTTI2 in wild-type (WT) and mutated *dek38-Dsg* alleles.

- (a). (4 points) What was the purpose to show ubiquitin and actin in Figure 2B and Figure 2C, respectively?
  - (b). (25 points) Please describe what you see in Figure 2B, 2C, and 2D. Combining your observation of these sub-figures with information provided, explain the possible molecular scenario in *dek* mutant at transcriptional- and protein-level.
5. (7 points) Genome editing is a way to modify genomic sequence in the last few years. This approach introduce double-strand breaks (DSB) at precise sites in the genome, allowing targeted modifications to occur when DSB are repaired by non-homologous end joining (NHEJ) or homology-dependent repair (HDR). Based on what you know about DSB repair mechanism and Figure 3, please provide which repair pathway occur more often in plants, and potential outcomes of genome editing.

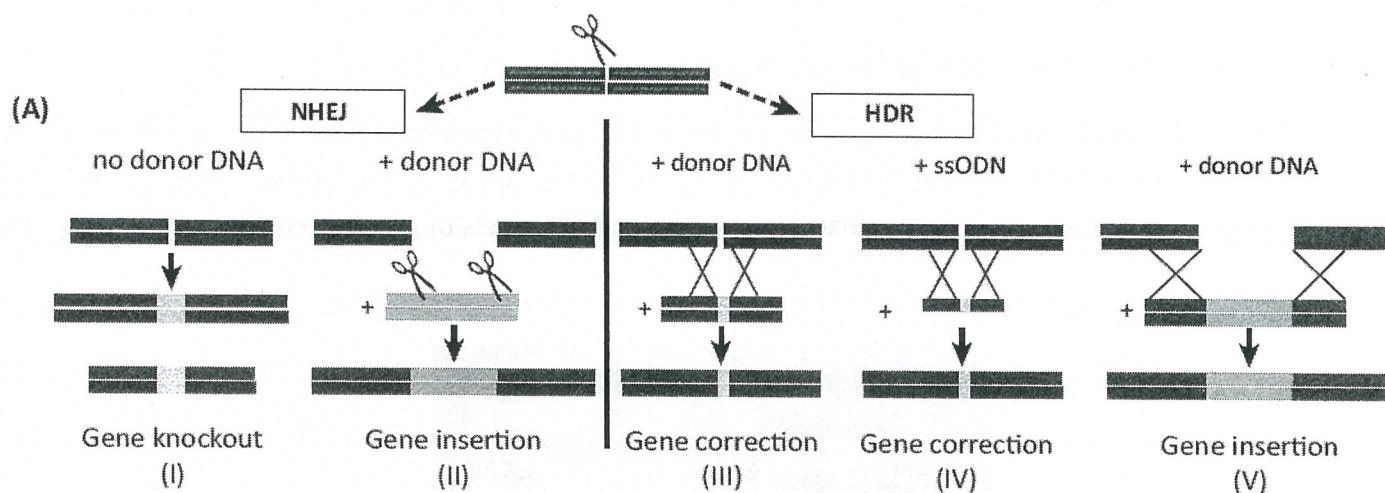


Figure 3. Potential outcomes of genome editing (modified from Figure 1A of Zhu et al. (2017) Trends Plant Sci 22:38). ssODN, single-stranded oligodeoxynucleotide.

Supplementary tables:

Critical values of the  $\chi^2$  distribution

df	1	2	3	4
P=0.05	3.841	5.991	7.815	9.488

Genetic codes (from Figure 9-8, Griffith et al. (2005) An introduction to genetic analysis. 8<sup>th</sup> ed.)

		Second letter					
First letter	U	UUU } Phe	UCU } Ser	UAU } Tyr	UGU } Cys		Third letter
		UUC } Phe	UCC } Ser	UAC } Tyr	UGC } Cys		
		UUA } Leu	UCA } Ser	UAA } Stop	UGA } Stop		
		UUG } Leu	UCG } Ser	UAG } Stop	UGG } Trp		
C	CUU } Leu	CCU } Pro	CAU } His	CGU } Arg			
		CUC } Leu	CCC } Pro	CAC } His			CGC } Arg
		CUA } Leu	CCA } Pro	CAA } Gln			CGA } Arg
		CUG } Leu	CCG } Pro	CAG } Gln			CGG } Arg
A	AUU } Ile	ACU } Thr	AAU } Asn	AGU } Ser			
		AUC } Ile	ACC } Thr	AAC } Asn			AGC } Ser
		AUA } Ile	ACA } Thr	AAA } Lys			AGA } Arg
		AUG } Met	ACG } Thr	AAG } Lys			AGG } Arg
G	GUU } Val	GCU } Ala	GAU } Asp	GGU } Gly			
		GUC } Val	GCC } Ala	GAC } Asp			GGC } Gly
		GUA } Val	GCA } Ala	GAA } Glu			GGA } Gly
		GUG } Val	GCG } Ala	GAG } Glu			GGG } Gly

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