*Indian J. Genet*., 76(4): 512-514 (2016)
DOI: 10.5958/0975-6906.2016.00069.9

# Multiple allelism at *Er* locus for powdery mildew resistance in pea (*Pisum sativum* L.)

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(Received: November 2016; Revised: November 2016; Accepted: November 2016)

**Abstract**

**Several sources of powdery mildew resistance in pea have been identified in natural germplasm as well as in landraces and cultivated varieties. Genetic studies revealed that all such cases are recessive mutations in a single gene called *Er.* Many carriers of the powdery mildew resistance trait differ in their phenotype and expression. This indicated existence of multiple alleles at the locus controlling resistance to powdery mildew. Molecular analysis revealed differences in the nature of changes induced in nature or during mutagenesis. A total of nine molecular mutations have been characterized. Many more alleles of different origin are awaiting similar characterization.**

**Key words:** Pea, *Pisum sativum*, powdery mildew resistance, multiple allelism, molecular characterization

The powdery mildew is possibly the most widespread and devastating disease of pea. Depending on the genotype and growing conditions (mainly humidity and temperature), it causes severe losses in grain yield and quality of produce. The first, and so far only, gene for powdery mildew resistance (PMR) was reported in a germplasm strain of Peruvian origin by Harland (1948). It was monogenic recessive and caused by the gene *Er* (Erysiphe resistance), the dominant allele *Er* making the plant susceptible and its recessive allele *er* causing near-perfect resistance. Subsequently, Heringa et al. (1969) reported resistance in three strains of pea (Mexique 4, Stratagem [JI 2302] and SVP 942), which were weaker in causing PMR, hence presumed to be a different gene. It was therefore named *Er2* and the gene discovered by Harland was renamed *Er1*. The digenic control of PMR was not validated through complementation test for allelism. It was generally accepted as a fact. In a few studies, attempts were made to determine allelic relationship between *Er1* and *Er2*, but with conflicting conclusions. The gene in Mexique 4 was demonstrated to be *er1* (same as reported by Harland) and Stratagem is also now maintained as type line of *er1*. One of the strains in the SVP series (SVP 941 [JI 2480]) is still maintained as type line for *er2*.

Although the exixtence of *er2* was accepted as *fait accompli*, attempts to verify its status as an independent genetic unit yielded variable results. Kumar and Singh (1981) reported $F_2$ segregation in the digenic ratio of 15 (R) : 1 (S) in the crosses between donor strains of *er1* and *er2*. From similar crosses, Tiwari et al. (1997) reported segregation in the ratio of 9 : 7. Gupta et al. (1995) concluded the PMR trait to be polygenic. Without any experimental evidence and additional confirmation, Katoch et al. (2010) reported *er2* as an indipenedent gene and mapped it in LGIII. Our results (Sharma and Kala, 2012) from coplementation test between *er1* (Stratagem) and *er2* (SVP 951) unequivocally proved that they are non-complementing alleles of the same gene first reported by Harland.

The reason for contradictory and erroneous conclusions about *er1* and *er2* lies in inaccurate identification of the genetically resistant and susceptible plants in a segregating population. The fact that infection (fungal growth) does not always lead to disease (tissue necrosis) was not taken into account. Another major criterion to differentiate between gene-caused susceptibility and resistance is that in the genetically resistant plants (*erer*), the fungal infection remains confined to the foliage (leaves and

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Published by the Indian Society of Genetics & Plant Breeding, F2, First Floor, NASC Complex, PB#11312, IARI, New Delhi 110 012
Online management by indianjournals.com; http://epubs.icar.org.in/journal/index.php/IJGPB

stipules) and does not spread to the stem, peduncles, and pods (Sharma, 2003, 2015).

**Multiple allelism at the Er locus**

Recessive PMR mutations have been reported in many genotypes. These include germplasm accessions, landraces, cultivars developed from recombination breeding, and induced mutations. Conventionally, mutations with similar phenotypes in different genotypes are accepted as different alleles with a common denominator (e.g. *er1*) accompanied by a suffix to indicate the source of identification – a symbol representing the parent genotype or name of an induced mutation. Unless the new alleles are independently discovered in unrelated genotypes, at different times or different places, or different induced mutations, the possibility always remains that the same allele is given different names in different genotypes in the absence of knowledge about the molecular change in the gene. This is more likely to happen in the derivatives of hybridization experiments.

In such a situation, the true identity or difference between phenotypically similar, or even identical, alleles of a given gene can be established with absolute certainty on the basis of molecular change induced in each individual case (Sun et al. 2016).

The multiple alleles of *Er* gene reported and analysed so far can be summarized as follows:

***er1:*** The first PMR allele, a spontaneous mutation, reported by Harland (1948). It is a very strong allele, imparts strong and reliable disease resistance in all regions of the world, and has been almost exclusively used in breeding programmes. Among the commercial varieties, it was first detected in var. Mexique 4 and, possibly, spread to the cultivars Tara (Canada), Cooper (Denmark) and Yunwan 8 (China). It carries a C → G transversion at position 680 of the *Er* (PsMLO1) gene, which creates UAG stop codon, leading to premature chain termination.

This allele is likely to be present in the largest number of mildew resistant varieties developed all over the world. India, possibly, is the only country where the PMR alleles have been used most effectively in developing resistant commercial varieties. The present situation is that only PMR varieties of field pea are under cultivation. Production of certified seed of PMS varieties is prohibited. Most of them have gone extinct. Such a universal exploitation of recessive allele(s) of one gene with mass impact is not known elsewhere. After nearly 30 years of PMR research, the powdery mildew disease has been almost completely wiped out in India. Based on the nature of molecular change in this mutation, this allele has been renamed as *er1-1* (Sun et al. (2016).

***er1-2*****:** This allele resulted from a combination of one 129-bp long deletion between positions 1171 and 1299, a 155-bp long insertion, and another 220-bp long insertion at position 1263 in the coding sequence of PsMLO1 gene (Humphrey et al. 2011; Sun et al. 2015; Wang et al. 2015), leading to frameshift mutations. This means three different molecular events have been clubbed into a single allele. Ideally, these should be distinguished as thre different alleles, for example, *er1-2*, *er1-3* and *er1-4*. In that case, the subsequent alleles of er1 gene will have to be renumbered accordingly. According to Sun et al. (2016), the present *er1-2* allele is responsible for disease resistance in the vaieties/ lines Yunwan 21, Yunwan 23, Stratagem (JI 2302), Franklin, R013/02, orian, Nadir, X 2009 and Xucal 1.

**Nine recessive alleles** of *Er1* gene have been identified and characterized so far: *er1-1*, *er1-2*, *er1-3*, *er1-4*, *er1-5*, *er1-6*, *er1-7* and two chemically induced mutations – *er1* mut1 and *er1* mut2 (Santo et al. 2013). The recently identified allele, *er1-7*, in PMR variety DDR 11 (developed at the Indian Agricultural Research Institute, New Delhi, India) carries a 10-bp long deletion in exon 1 of PsMLO1 (Sun et al. 2013).

**Er alleles identified in other pea cultivars and Pisum species**

As mentioned above, the mutation identified as an independent gene, *er1*, by Heringa is now confirmed as an allele of *Er1.* It can still be maintained with the symbol *er2*. Sharma (2003b) reported a PMR strain of *Pisum arvense*. Its genetics is yet to be worked out. However, it is proposed to be named as erA where A stands for *arvense*. Another PMR strain, called Peru II of *Pisum sativum* (kindly provided by Prof. N. Weeden), was tested for er allelism, and was found to be allelic to *Er1* gene. This allele is proposed to be named as *erP* (P for Peru II). Such a large number of independently induced PMR mutations, and all of them belonging to the *Er1* gene, leaves no doubt that

powdery mildew resistance in cultivated peas is governed by a single gene. The other source of PMR is available in *Pisum fulvum*. This gene causes disease resistance in dominant state, hence named *Er3.* It has been transferred in *Pisum sativum* background, and its breeding value needs to be determined.

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