acterized by the accumulation of storage products and LEA (late embryogenesis abundant) proteins with a protective role.

Viviparous mutant embryos that are deficient in ABA synthesis or lack an active *vp1* factor do not express the normal set of maturation phase proteins and should not acquire desiccation tolerance. To verify this, we applied premature desiccation to developing *vp* embryos about 25 DAP, and compared their germination capacity to sib embryos not subjected to such treatment (Durantini et al., Heredity 101:465-470, 2008). To this aim, *vp* and normal sibling embryos from a segregating ear were excised and transferred to plant cell culture vessels (Phytatray Sigma) on basal MS medium containing 2% sucrose solidified with 0.8% agar, or subjected to desiccation and a storage period of 60 days at 5°C before transfer to the same medium. For the drying treatment, mutant and normal embryos were laid between two disks of blotting paper within a sterile petri dish and incubated in an oven at 35°C for 48 hours.

At the end of the treatment, the dishes were sealed with parafilm and conserved at 5°C with silica gel at the bottom of the petri dish under the blotting paper. For the germination test, embryos were maintained in a growth chamber at 25°C with a 14/10 h light/dark photoperiod. Germination was determined after 10 days of culture. When cultured immediately after their excision, immature embryos of all mutants tested germinated with a high frequency (95-100%) like their wild-type counterparts (data not shown). On the other hand, if they were cultured following a premature dehydration treatment, only *vp1* and *vp10* maintained a partial desiccation tolerance while the other mutants lost it (Table 1, Fig. 1). In addition, *vp5* showed minimal germination, consisting of primary root protrusion without a shoot.

In contrast to the results presented in this report, White et al. (Plant Physiol. 122:1081-1088, 2000) showed that induction of GA deficiency early in seed development, either genetically or via biosynthesis inhibitors, suppressed vivipary of *vp5/vp5* mutants while maintaining desiccation tolerance. However, since we applied a different protocol to test desiccation tolerance of the viviparous mutants that did not involve the inhibitors of GA synthesis, the results obtained are not strictly comparable. Furthermore, while interpreting these data one should keep in mind that the nature and position of the molecular lesion within the gene might affect the germination test of the mutant under testing, as clearly shown by the analysis of the alleles of *vp7* (*ps1*) obtained by *Ac* insertional mutagenesis (Bai et al., Genetics 175-981-992, 2007). We also found that *vp10-374* mutants exhibiting partial desiccation tolerance are impaired, to a different extent, in their morphogenesis.

These results seem to suggest that acquisition of desiccation tolerance requires the completion of the steps between carotenoid production and the late stage of ABA biosynthesis, suggesting a link between embryo morphogenesis and desiccation tolerance which should be further investigated.

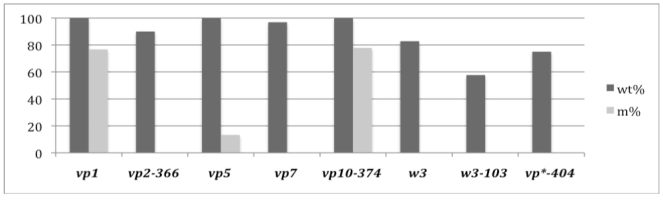

Figure 1. Effect of premature dehydration on germination capacity of wild-type (wt) and viviparous sib embryos.

Table 1. Germination percentage of homozygous *vp* mutant embryos. The germination percentage of the corresponding wild-type sibs is shown in Figure 1.

			+		m	
CODE	DAP	Mutant	No. seeds	germ %	No. seeds	germ %
07.50A-2	26	*vp1*	31	100	30	77
07.01-2	28	*vp2-366*	10	90	9	0
07.62-15	25	*vp5*	30	100	30	13
07.21-2,7	24,26	*vp7*	32	97	30	0
06.182	24	*vp10-374*	24	100	18	78
07.67-7	26	*w3*	29	83	38	0
07.06-1,2	21,24	*w3-103*	33	58	17	0
06.51-N	25	*vp*-404*	20	75	8	0

+ and m refer to wild-type and viviparous siblings

Another case of second site non-complementation

--Galbiati, M; Gavazzi, G

We observed another case of second site non-complementation (SSNC) while analyzing the complementation pattern of *d11**, a dwarf mutant inherited as a monogenic trait. Homozygous *d11** plants, which are drastically reduced in stature, produce andromonecious ears with normal seed set and exhibit a significant increase in their elongation if grown in the presence of 10 µM GA (Galbiati et al., Maydica 47:169-180, 2002). The pattern of complementation of this mutant with recessive *d* mutants reported in the literature (*d1*, *d2*, *d3*, *d5* and *an1*) is unexpected since it indicates that the mutant complements *d3* and *an1* but fails to complement *d1, d2* and *d5*. By further testing each of the double mutants in the F2 generation, we confirmed allelism of *d11** with *d1* and *d5*, a surprising result. On the other hand, the selfed progeny of heterozygous *d11**/+ *d2*/+ dihybrids produceed ears with a 9 to 7 segregation of normal versus dwarfs, a segregation expected if *d2* and *d11** define two separate genes. We hypothesize that the contrasting results observed in the F1 and F2 generation could indicate an interaction of the gene products of two genes as described in our previous report. While germinating seeds of the 9:7 segregating ears, we noticed that a minority of the dwarf seedlings had a more pronounced reduction in their elongation. A similar observation also applied to the F2 progeny of *d11**/+ *an1*/+ parents. In the F2 progeny of these heterozygous double mutants, one-seventh of the dwarfs should be homozygous double mutants and should yield, assuming an additive effect of the two mutations, seedlings with a higher reduction in their length than single gene mutants. This is exactly what we observed and can be taken as evidence that the two genes have an additive effect (Table 1, Figure 1).

Table 1. Stature of wild type, dwarf and severe dwarf seedlings and frequency of the severe dwarf phenotype in the selfed progeny of different double heterozygous combinations. The *d*10/an1* double mutant is included as another example of the detection of severe dwarfs in the F2.

Double heterozygote constitution (F2)	Seedling elongation (cm)			Frequency of severe dwarfs (%)		P-value
	wild type	dwarfs	severe dwarfs	observed	expected	
*d*11* x *d2*	18.2	7.2	4.4	18.7	14.3	0.15
*d*11* x *an1*	22.9	9.4	6.1	15.6	14.3	0.56
*d*10* x *an1*	16.6	8.9	4.5	17.4	14.3	0.38

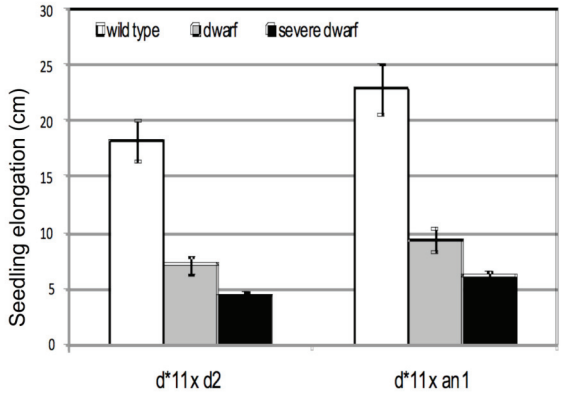

Figure 1. Stature of wild type, dwarf and severe dwarf seedlings in the selfed progeny of *d2/d*11* and *an1/d*11* double heterozygotes. Seedling elongation was determined at day ten after germination by measuring the distance between the scutellar node and the tip of the last leaf. Each value represents the mean (± std. dev) of three independent experiments.

These results seem to suggest that *d11** and *d2* or *an1* affect different biological pathways, both of which contribute independently to seedling elongation. It remains to be explained how *d11** shows non-complementation with *d1* and *d5,* two genes located on different chromosomes and controlling separate steps in GA biosynthesis.

Characterization of a dominant mutation of the *Dwarf8* gene

--Pilu, R; Cassani, E; Bertolini, E; Landoni, M; Gavina, D; Villa, D; Cerino Badone, F; Sirizzotti, A; Casella, L; Lago, C

We have isolated and characterized a new spontaneous dominant dwarf mutation that at maturity shows delayed flowering and reduced stature, ranging from 60-70% in a W23 NIL (Fig. 1A) to a 40-45% in a W23 X B73 F1 hybrid (Fig. 1B), caused by a reduced internode length. This mutant also shows thick broad leaves, that are 25-30% larger than wild type (Fig. 1C), a strong gene dosage effect on phenotype and a less severe phenotype in comparison with the *D8-1* dominant mutant, as shown in Fig. 1D. The dwarf phenotype is also easily detectable in the first stage of plant growth (Fig. 1E) and at maturity shows a tendency to produce tillers (Fig. 1F). In addition, the dwarf mutant is altered in its floral development. In fact, stamens are present in the terminal flowers of the ears (andromonoecious ear); however, they are sterile (Fig. 1G). The dimensions of the silks and anthers in the inflorescence are bigger by about 40% and 17%, respectively, compared to the wild type (Fig. 1H, I). The genetic analysis performed to understand the inheritance of this dwarf mutation demonstrated a monogenic dominant inheritance of this trait, and the map position was established on the long arm of chromosome 1. The results obtained from this analysis showed that *D*-1023* maps where *D8-ref* was located, and thus the mutation was renamed *D8-1023*.

The novel mutant allele was cloned and the alignment with *d8*(+) wild type alleles present in the database has shown a molecular lesion: an insertion of 3bp within the VHYNP domain, located in the 5' of the gene near the DELLA domain, which is responsible for the GA response (Fig. 2). This finding represents the first evidence of a dominant dwarfing mutation that does not involve the DELLA domain but is in the not yet well-characterized VHYNP domain, which is involved in protein degradation. We have found a new and interesting phenotype and we suggest a possible future modification of the VHYNP domain of the *D8* gene to modulate plant growth and to shorten excessively tall germplasm, with the aim of improving crop production.

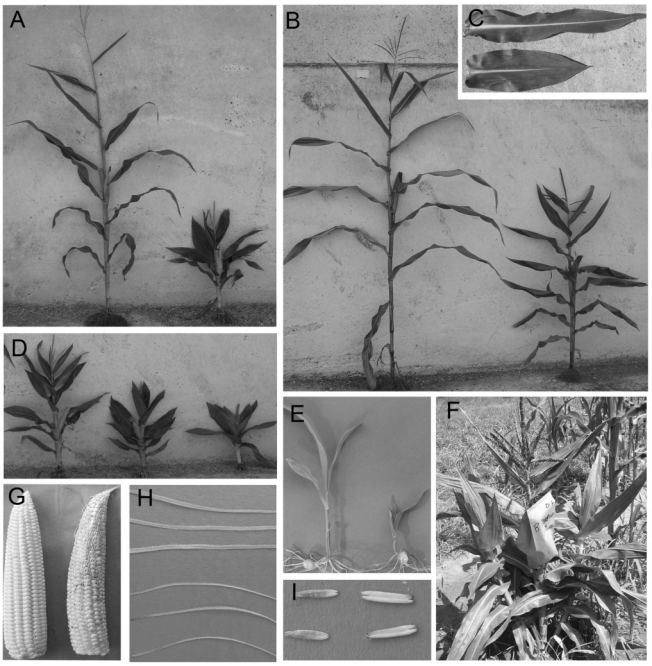

Figure 1. Phenotype of the new dwarf mutant: (A) wild type (left) and *D*-1023/+* mutant (right) whole plants at maturity in a W23 near-isogenic line; (B) wild type (left) and *D*-1023/+* mutant (right) whole plants at maturity in a W23 X B73 F1 hybrid genetic background; (C) leaves, wild type above and mutant below in a W23 near isogenic line; (D) from left to right *D*-1023/+*, *D*-1023/D*-1023*, *D8-1/+* whole plants in a W23 genetic background; (E) wild type seedling (left) and dwarf (right); (F) dwarf tillering growth habit in a B73 near isogenic line; (G) wild type ear (left) and dwarf anthered ear (right); (H) mutant silks (above) and wild type silks (below); (I) wild type anthers (left) and mutant anthers (right).

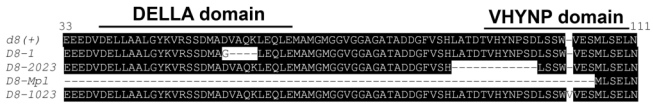

Figure 2. Partial alignment between the *d8* wild type allele and predicted proteins encoded by dominant mutant alleles. The wild type *d8* allele is compared with *D8-1*, *D8-2023*, *D8-Mpl* and *D8-1023* dominant mutant allele-encoded proteins with mutant N-terminals. Differences between wild type and mutant sequences (deletions, insertions and substitutions) are highlighted in white, and the previously identified highly conserved DELLA and VHYNP domains are shown.

MONTECILLO, MEXICO
Colegio de Postgraduados

Double kernel fruitcases found in teosinte populations

--Kato Y., TA

Teosinte fruitcases traditionally are known to have only one developed spikelet each because the second one is suppressed during the ontogeny of the female inflorescence. According to Galinat (Corn and Corn Improvement, G. F. Sprague (ed.), pp. 1-47, 1988) the primary step for evolving teosinte to maize was the reactivation of the second spikelet to form the necessary link between these two plant taxa and he states that "The pairing of fe-