

Supplemental Table 1 : Updated classification of *ago1* alleles

Line	Allele	Mutagen	Ecotype	Mutation
nab1	<i>ago1-1</i> ^{a*}	T-DNA	Col-2	
dlw2	<i>ago1-2</i> ^{a*}	T-DNA	WS	
e052	<i>ago1-3</i> ^{a*}	EMS	Col-0	G42Stop
e436	<i>ago1-4</i> ^{a*}	EMS	Col-0	
e553	<i>ago1-5</i> ^{a*}	EMS	Col-0	22bp deletion C2726-G2747
e691	<i>ago1-6</i> ^{a*}	EMS	Col-0	
abf27	<i>ago1-7</i> ^b	T-DNA	WS ^b	
	<i>ago1-8</i> ^b	EMS	Ld ^b	
	<i>ago1-9</i> ^c	EMS	Ld ^c	2bp deletion C1154-T1155
	<i>ago1-10</i> ^d	EMS	Ld ^g	1bp deleted A1170
	<i>ago1-11</i> ^e	EMS	Ld ^f	
	<i>ago1-12</i> ^d	EMS	Ld ^g	H763L
e1099	<i>ago1-13</i> ^{f*}	EMS	Col-0	Q537stop
e1387	<i>ago1-14</i> ^{f*}	EMS	Col-0	Q885Stop
6w	<i>ago1-15</i> ^{f*}	EMS	Col-0	
7r	<i>ago1-16</i> ^{f*}	EMS	Col-0	
8t	<i>ago1-17</i> ^{f*}	EMS	Col-0	
14b	<i>ago1-18</i> ^{f*}	EMS	Col-0	P493S
acx7	<i>ago1-19</i> ^{f*}	T-DNA	WS	
dsv1	<i>ago1-20</i> ^{f*}	T-DNA	WS	
dyp79	<i>ago1-21</i> ^{f*}	T-DNA	WS	
23-1	<i>ago1-22</i> ^g	EMS	Col-0	
46-3	<i>ago1-23</i> ^g	EMS	Col-0	
60-1	<i>ago1-24</i> ^g	EMS	Col-0	L571F
19-3	<i>ago1-25</i> ^h	EMS	Col-0	G758S
33-2	<i>ago1-26</i> ^h	EMS	Col-0	P838S
69-4	<i>ago1-27</i> ^h	EMS	Col-0	A992S
e965	<i>ago1-28</i> ^{f*}	EMS	Col-0	
e1174	<i>ago1-29</i> ^{f*}	EMS	Col-0	
dwy4	<i>ago1-30</i> ^{f*}	T-DNA	WS	
eai70	<i>ago1-31</i> ^f	T-DNA	WS	
13-5	<i>ago1-32</i> ^f	EMS	Col-0	
6-5	<i>ago1-33</i> ^f	EMS	Col-0	
450-3	<i>ago1-34</i> ^f	FNM	Col-0	
450-16	<i>ago1-35</i> ^f	FNM	Col-0	

a - Bohmert *et al.* (1998), b - Lynn *et al.* (1999), c - Lippman *et al.* (2003),
d - Kidner *et al.* (2004), e - Kidner *et al.* (2003), f- Sorin *et al.* this issue, g
- Fagard *et al.* (2000), h - Morel *et al.* (2002)

* analysed on western blot (supplemental fig1)

In a forward genetic screen of EMS mutagenized lines and T-DNA insertion lines we identified 21 *argonaute1* alleles showing a strong phenotype (Camus 1999). Some of these alleles had to be renamed, because of the recent publication of new alleles (Kidner and Martienssen 2003; Kidner and Martienssen 2004). An updated nomenclature for our *ago1* alleles and their origin is provided here. The first six alleles (*ago1-1* to *ago1-6*) were described in (Bohmert et al. 1998). Fourteen of the other alleles, which showed a similar, strong phenotype, were considered as strong alleles. The allele *ago1-18* gave rise to a slightly weaker phenotype. The mutation was identified in five of the EMS alleles. These include our reference mutant *ago1-3*, *ago1-18* that had the WT size protein, and three alleles showing truncated proteins (STable1). The *ago1-3* mutation induces an early STOP codon in position G42, confirming that it is a null allele. The truncated proteins are likely to be inactive as they certainly lack the conserved PIWI and/or PAZ domain as confirmed by the sequence analysis for three of the alleles. The *ago1-18* sequence showed a single amino-acid change (P493S). Interestingly this is the exact same mutation as observed in the allele *zll1-16* of the *ZWILLE/PINHEAD* gene (Moussian et al. 1998), and that also leads to a milder phenotype (Moussian personal communication). Although *ago1-18* had a weaker phenotype than the null alleles, it was still stronger than that of the hypomorphic *ago1* mutants described by Morel et al. (2002) highlighting the importance of that particular amino-acid for the protein functionality. The *ago1-5* sequence showed a frame-shift 22 bp deletion that created a STOP codon leading to the deletion of the C-terminus part of the protein including part of the PIWI domain.

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