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Erratum: The RNA editing enzyme APOBEC1 induces somatic mutations and a compatible mutational signature is present in esophageal adenocarcinomas

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Erratum

During the typesetting of the final version of the article [1] some data in Table 1 have been accidentally changed. These data were correct in the provisional version of the article. A correct Table 1 is as follows:

Please note that references 41 and 42 in the table legend correspond to the reference order in the original article [1].

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Reference

1. Saraconi G, Severi F, Sala C, Mattiuz G, Conticello SG: **The RNA editing enzyme APOBEC1 induces somatic mutations and a compatible mutational signature is present in esophageal adenocarcinomas.** *Genome Biology* 2014, **15**:417.

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Table 1 List of the non-clonal mutations identified in the *BCR-ABL1* fusion gene from imatinib-resistant K562 clones

Samples	Position	AA change	Codon change	Sequence context
Control	1409	E495G	G**A**A > G**G**A	
	1472	R491Q	C**G**G > C**A**G	GC**C**GG
	1503	Silent	TT**T** > TT**C**	
AID	298	G100C	**G**GC > **T**GC	GC**C**TA
	395	Silent	AA**A** > AA**G**	
	568	G190D	G**G**C > G**A**C	TG**C**CA
	633	T212A	**A**CG > **G**CG	
Rat APOBEC1	344	G155D	G**G**C > G**A**C	GG**C**CA
	607	L203M	**C**TG > **A**TG	CC**C**TG
	613	E205K	**G**AG > **A**AG	CT**C**GG
	764	K255T	A**A**G > A**C**G	
	987	Silent	GG**G** > GG**T**	TT**C**CC
Human APOBEC1	669	Silent	AG**C** > AG**T**	AG**C**CG
	758	R253H	C**G**C > C**A**C	TG**C**GC
	841*	Silent	**C**TG > **T**TG	AG**C**TG
	697	H233D	**C**AT > **G**AT	TT**C**AT
	699	H233D	CA**T** > GA**C**	
	1149*	Silent	GC**C** > GC**A**	GC**C**AT
	1245*	F415L	TT**T** > TT**G**	

The region analyzed (encompassing exon 13 of BCR and exon 9 of ABL1) includes the imatinib-binding region of the fusion gene. The asterisk indicates mutations found in the same clone. The local sequence context for the mutations at cytosines is shown. Compared to the AID-induced mutations found in previous reports (mutations in approximately 30% of the sequences) [41], [42], we found approximately one mutation in each of the clones analyzed. This is explained by the different procedures we used to select resistant clones: whereas the other studies focused on competing bulk populations of AID-transfected GFP(+) cells and control GFP(–) cells, we analyzed individual clones arising from the same number of cells plated in the presence of imatinib.