Erratum

Erratum to *EGFR* exon 20 insertion mutations and *ERBB2* mutations in lung cancer: a narrative review on approved targeted therapies from oral kinase inhibitors to antibody-drug conjugates

Editorial Office

Translational Lung Cancer Research

Correspondence to: Editorial Office. Translational Lung Cancer Research. Email: editor@tlcr.org.

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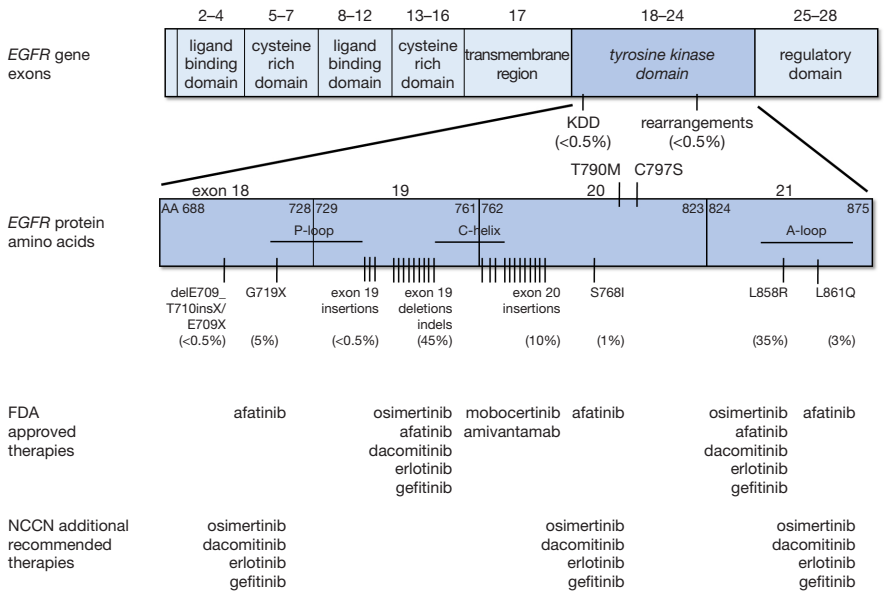
Erratum to: Transl Lung Cancer Res 2023;12:1590-610.

In the July 2023 issue of *Translational Lung Cancer Research*, the article "*EGFR* exon 20 insertion mutations and *ERBB2* mutations in lung cancer: a narrative review on approved targeted therapies from oral kinase inhibitors to antibody-drug conjugates" authored by Dr. Sentana-Lledo *et al*. (1) was published with some minor errors in *Figure 2* and its figure legend. The data "0.5%" under "G719X" should be corrected as "5%". In the figure legend, the sentence "EGFR-S768I is placed out of position for better representation" should be added.

The whole *Figure 2* should be corrected as:

A



B

	preclinical sensitivity + selectivity pattern against *EGFR*-WT of EGFR inhibitors			
EGFR mutant subtype	1st gen EGFR TKI (gefitinib, erlotinib)	2nd gen EGFR TKI (dacomitinib, afatinib)	3rd gen EGFR TKI (osimertinib)	exon 20 insertion active EGFR TKI (mobocertinib)
Most common mutations				
exon 19 deletions/exon 19 indels	+++	+++	+++	++
exon 21 L858R	+++	+++	+++	++
Less common mutations				
exon 18 G719X	+	++	+	+
exon 21 L861Q	++	++	++	+
exon 20 S768I	+	+	+	+
exon 19 insertions/ K745_E746insIPVAIK	++	++	++	+
Exon 20 insertion mutations				
exon 20 insertions most prevalent (A767_V769dupASV, D770_N771insSVD, H773_V774insH)	−−−	−−−	−	+
exon 20 D770 > GY	−−−	++	−	+
exon 20 A763_Y764insFQEA	++	++	++	+
Resistance mutations				
exon 19 indel or L858R + T790M	−−−	−−−	+++	−−
exon 19 indel or L858R + C797S	+++	−−	−−−	−−−
exon 19 indel or L858R + T790M + C797S	−−−	−−−	−−−	−−−

Figure 2 Subtypes of *EGFR* mutations with a focus on preclinical patterns of response/resistance to EGFR TKIs. (A) Representation of the EGFR protein by key gene numbers, overlaid with clinically-relevant types of mutations mostly centered within the kinase domain. The prevalence of these mutation subtypes are indicated by exon location. The frequency of *EGFR* mutations was obtained from (17-22,49-56). EGFR-S768I is placed out of position for better representation. (B) Summary of preclinical models driven by selected *EGFR* mutations paired with the *in vitro* sensitivity and also *in vitro* selectivity pattern against *EGFR* WT of the diversity of approved EGFR TKIs. Data was extrapolated from (23,50,57-62) and unpublished data from the authors' translational thoracic oncology laboratory. The degree of sensitivity and resistance is indicated by number of + (sensitive/selective) or – (resistant/non-selective) signs as extrapolated from preclinical studies. Please, refer to aforementioned references for each individual half maximal inhibitory concentration (IC_{50}) for preclinical proliferation assays. EGFR, epidermal growth factor receptor; ERBB2, erb-b2 receptor tyrosine kinase 2 (also known as HER2, human epidermal growth factor receptor-2); WT, wild-type; TKIs, tyrosine kinase inhibitors.

The authors regret for the errors and confirm that they would not change the results or conclusion of the article.

Click here to view the updated version of the article.

References

1. Sentana-Lledo D, Academia E, Viray H, et al. EGFR exon 20 insertion mutations and ERBB2 mutations in lung cancer: a narrative review on approved targeted therapies from oral kinase inhibitors to antibody-drug conjugates. Transl Lung Cancer Res 2023;12:1590-610.

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