Erratum


PDZ interaction site in ephrinB2 is required for the remodeling of lymphatic vasculature
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In our above-mentioned paper, we have detected a sequence error in the gene targeting construct used to generate mutant mice expressing a tyrosine phosphorylation-deficient ephrinB2 protein (ephrinB2^{5F}). The presence of this error in the knock-in mouse line was confirmed using tail biopsy genomic DNA. The observed erroneous codon respecified Tyr 333 to leucine, rather than to phenylalanine as reported.

Subsequent to detection of this error, the full ORF of ephrinB2 obtained from tail biopsy genomic DNA of ephrinB2^{5F} and ephrinB2^{5Y} knock-in mice was sequenced. No additional differences from the reported sequences were found.

The actual C-terminal amino acid sequence of ephrinB2 specified by this knock-in allele, which we propose renaming as ephrinB2^{5Y}, in comparison to the wild-type sequence and the sequence reported in our above-mentioned paper is shown here, with \text{333}^{\text{Tyr}} \rightarrow \text{333}^{\text{Leu}} highlighted in blue:

\[
\begin{align*}
\text{YPFephrinB2}^{\text{wt}}: & \quad \text{YKVS}^{\text{307}} \text{EKP}^{\text{307}} \text{VS}^{\text{307}} \text{HP}^{\text{307}} \text{V}^{\text{307}} \text{IF}^{\text{307}} \text{VP}^{\text{307}} \text{EQ}^{\text{307}} \text{MP}^{\text{307}} \text{PQ}^{\text{307}} \text{PA}^{\text{307}} \text{N}^{\text{307}} \text{YY}^{\text{307}} \text{KV}^{*} \\
\text{YPFephrinB2}^{\text{5F}}: & \quad \text{FKVS}^{\text{307}} \text{EKP}^{\text{307}} \text{VS}^{\text{307}} \text{HP}^{\text{307}} \text{V}^{\text{307}} \text{IF}^{\text{307}} \text{VP}^{\text{307}} \text{EQ}^{\text{307}} \text{MP}^{\text{307}} \text{PQ}^{\text{307}} \text{PA}^{\text{307}} \text{N}^{\text{307}} \text{IF}^{\text{307}} \text{KV}^{*} \\
\text{YPFephrinB2}^{\text{5Y}}: & \quad \text{FKVS}^{\text{307}} \text{EKP}^{\text{307}} \text{VS}^{\text{307}} \text{HP}^{\text{307}} \text{V}^{\text{307}} \text{IF}^{\text{307}} \text{VP}^{\text{307}} \text{EQ}^{\text{307}} \text{MP}^{\text{307}} \text{PQ}^{\text{307}} \text{PA}^{\text{307}} \text{N}^{\text{307}} \text{LF}^{\text{307}} \text{KV}^{*}
\end{align*}
\]

Our study concluded that ephrinB2 is an essential regulator of lymphatic remodeling, and that interaction with PDZ domain effectors is required to mediate these functions, while phosphotyrosine-dependent signaling is dispensable for vascular development. Since the actual \text{333}^{\text{Tyr}} \rightarrow \text{Leu} mutation also prevents tyrosine phosphorylation at position 333 of ephrinB2, the error reported here does not change these conclusions. However, the \text{333}^{\text{Tyr}} \rightarrow \text{Leu} substitution occurs within the sequence motif recognized by PDZ domain-containing proteins. Structural evidence (Hung and Sheng 2002) suggests that the amino acid at this position could contribute to PDZ-dependent signaling, raising the possibility that ephrinB2^{5Y/5Y} mice have compromised PDZ-dependent signaling in addition to an abrogation of ephrinB2 tyrosine phosphorylation. The normal appearance of the ephrinB2^{5Y/5Y} mutant mice, in contrast to the severe deficiencies seen in ephrinB2^{5Y/5Y} mutant mice, suggests that the additional impact on PDZ-dependent signaling consequent to this sequence error is not severe. However, we cannot exclude that the mild lymphatic phenotype (abnormal drainage) observed in the ephrinB2^{5Y/5Y} mice (Fig. 4E in our above-mentioned paper) is due to reduced signaling via PDZ domain-dependent binding rather than deficiency in phosphotyrosine signaling.

References
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*Genes Dev.* 2006, **20**:

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