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Erratum

# Erratum to "Identification and characterization of murine IRAK-2" [Biochem. Biophys. Res. Commun. 297 (2002) 52–58]☆

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The use of a polymerase with an unexpectedly high error rate during cloning of murine IRAK-2 resulted in 11 base errors causing 9 amino acid changes in the sequence we published. The most relevant mutations were in the death domain (affecting the recruitment to MyD88) and in the kinase domain with an exchange of arginine to lysine at position 235, the putative ATP-binding site (possibly affecting the autophosphorylative capability).

We have corrected the sequence errors in the databases.

We observed the functional consequences of the amino acid exchanges. In contrast to our previous findings, the correct murine IRAK-2 when overexpressed enhances IL-1-stimulated NF-κB-dependent reporter gene expression and behaves like human IRAK-2. The correct murine IRAK-2 shows a very weak phosphorylation signal when overexpressed in HEK 293 cells and subjected to an in vitro kinase assay after immunoprecipitation. As HEK 293 cells contain endogenous IRAK-1 and IRAK-4, we cannot exclude the possibility that the weak phosphorylation signal may be due to cross-phosphorylation either by human IRAK-1 or by IRAK-4.

In summary, the murine IRAK-2 with the correct sequence behaves like human IRAK-2, murine IRAK-M, and human IRAK-M in overexpression systems and shows an extremely weak autophosphorylative capacity when compared to murine or human IRAK-1 or IRAK-4.

We apologize for the errors in our original report, and we thank Dr. M. Hardy, Trinity College, Dublin, Ireland, for drawing our attention to the mistakes.

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