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Corrigendum

IDO2 is critical for IDO1-mediated T-cell regulation and exerts a non-redundant function in inflammation

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The authors wish to correct an error discovered by one of us (L.M.F.M.) in the structure of the recombinant *Ido2* genetic construct used to create the *Ido2* mutant mouse analyzed, however, this correction does not impact its validity as a loss-of-function strain or therefore any conclusions of the study. As illustrated below in the revised Fig. 1, the location of the two loxP sites employed for Cre-mediated excision are located within exons 9 and 10 in the *Ido2* gene, rather than upstream of exon 9 and

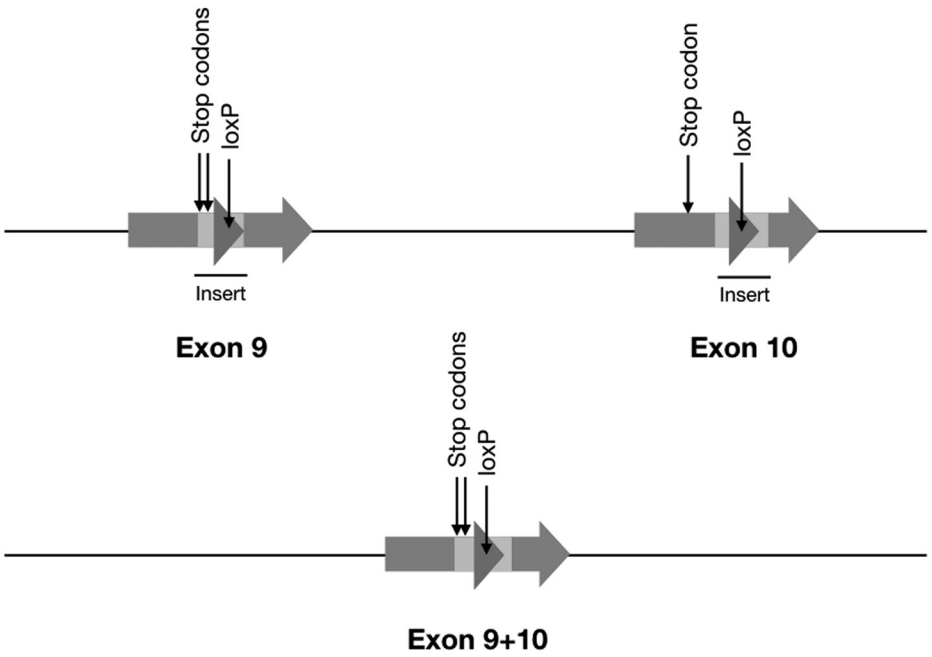

Fig. 1. Corrected genetic structure of *Ido2* mutant.

downstream of exon 10 as reported (due to an error in selection of this construct for use for unknown reasons). In this construct, stop codons are generated for Ido2 translation by frameshift readthroughs at the loxP cassette which was inserted into exon 9 (top line of the figure). Accordingly, targeted insertion into the mouse genome created a constitutive loss-of-function allele without further utility as a *cre*-inducible loss-of-function allele. In breeding the parental strain to EII-*cre* mice, to induce *cre*-mediated recombination, we generated a systemic deletion mutant lacking the exon 9–10 segment (bottom line in the figure). Thus, a valid loss-of-function mutant was created, albeit one derived from a mutant rather than wild-type parent. The corrected genetic construct eliminates uses for *cre*-inducible deletion, e.g. to generate tissue-specific mutants, but the strain that was created nevertheless remains valid and useful as a systemic deletion mutant.