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CORRECTION

Correction: Targeted gene correction in the mdx mouse using short DNA fragments: towards application with bone marrow-derived cells for autologous remodeling of dystrophic muscle

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Figure 2 in the original manuscript contained two referential panels (B and C) that had been modified from a previous publication by the authors (Hum Mol Genet, 2001, 12:629–642). These panels were superfluous to the specific context of the data published in the manuscript and did not serve any purpose other than being illustrative of general methodology and were therefore redundant to the scientific substance of discovery being communicated in the publication. Panels C and D from the original publication, along with any textual reference to them in the Fig. 2 Legend, have thus been removed. The Fig. 2 (and modified Figure Legend) in the original communication should be replaced with those provided herein.

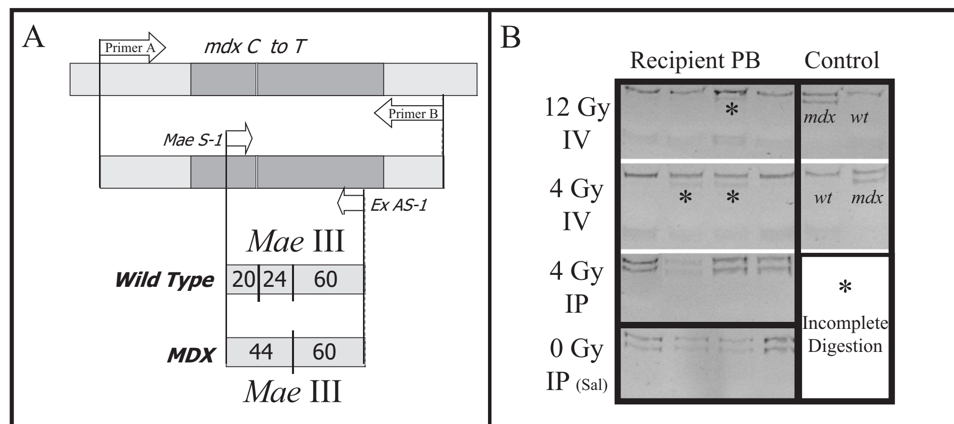

Fig. 2 Detection and quantification of *wt dys* loci amongst *mdx dys* loci. (A). Strategy for quantification of *wt dys* locus and transcript. DNA and RNA extracted from blood and muscle are subject to primary PCR using primers 1 and 2. Primers 1 and 2 are differently designed for DNA and RNA protocols. For amplification of DNA from blood and muscle, primers 1 and 2 consist of neighbouring intronic sequence, whilst for amplification of RNA message from muscle, sequence from neighbouring exons were used[8]. DNA sequence was amplified directly, whilst RNA sequence was obtained by poly-T primed reverse transcription (Promega). After purification of primary PCR products using reverse phase chromatography (Qiagen), a second PCR was performed on 50 ng of the amplification product, using the *Mae* S-1 and *Ex* AS-1 primers as shown with the inclusion of aP^{32}-dCTP radiolabel. The *Mae* S-1 primer is 3′ end-modified to result in *Mae* III restriction site acquisition in the presence of the *wt* nucleotide at the *mdx dys* locus[4]. In the absence of *wt* locus, the secondary PCR product (1 to 2 μg) is cleaved overnight at 55°C into two fragments, 60 and 44 bp in length at an existing *Mae* III site. In the presence of the *wt* nucleotide, the 44 bp fragment is further cleaved to give two fragments 20 and 24 bp long (Fig 1B, controls). These fragments are then resolved on a 20% acrylamide gel and visualized by phosphorimager screen exposure. **(B)**. Quantification of *wt dys* locus in peripheral blood of *mdx* mice transplanted with *wt* BM cells. DNA was extracted from the blood obtained at 6 months post-injection from the recipient *mdx* mice. The extent of *wt dys* gene was evaluated using methodology described elsewhere[4]. Incomplete digestion is shown by asterisk (*). Data from 4 mice from each group of 5 are shown. Both IV groups showed ~100% *wt dys* locus in the PB after 6 months, indicating full BM remodelling with *wt* BM derived from donor cells. The 4Gy/IP group did not display *wt* locus within the resolution of the PCR-RFLP *wt dys* locus detection method employed here. The level of *wt dys* locus in the BM of these mice was therefore less than the absolute detection capacity (<0.5% of loci) of this method. The 0 Gy /Saline-IP group are included as controls.