MEDICAL SCIENCES

Correction for "Laminin-111 protein therapy prevents muscle disease in the mdx mouse model for Duchenne muscular dystrophy," by Jachinta E. Rooney, Praveen B. Gurpur, and Dean J. Burkin, which appeared in issue 19, May 12, 2009, of *Proc Natl* Acad Sci USA (106:7991-7996; first published April 28, 2009; 10.1073/pnas.0811599106).

The authors note that in preparing Fig. 3A, an image from Fig. 6A was inadvertently inserted. This error does not affect the conclusions of the article. The corrected figure and its legend appear below.

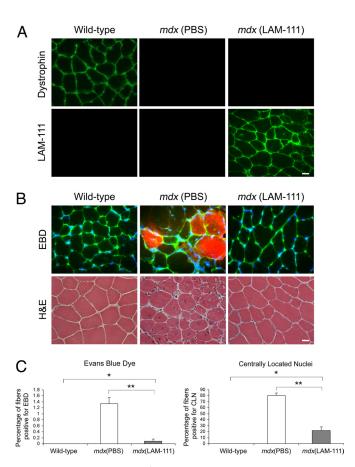


Fig. 3. Intramuscular injection of laminin-111 prevents muscle disease in *mdx* mice. (A) Immunofluorescence of the TA muscles of control and laminin-111-treated mice confirms the absence of dystrophin in mdx muscle treated with LAM-111 or PBS. Laminin-111 was not present in wild-type or PBS-injected mdx muscle, but it was detected in the extracellular matrix of laminin-111-injected mdx muscle. (Scale bar: 10 μm.) (B) EBD uptake reveals that mdx muscle injected with laminin-111 exhibits reduced EBD uptake compared with control. (Scale bar: 10 μm.) H&E staining reveals that mdx muscle treated with laminin-111 contains few muscle fibers with centrally located nuclei and mononuclear cell infiltrate compared with control. (C) Quantitation reveals wild-type and mdx muscle treated with laminin-111 contained significantly fewer EBD-positive fibers and myofibers with centrally located nuclei compared with control. *, P < 0.05; **, P < 0.001; n = 5 mice per group.

www.pnas.org/cgi/doi/10.1073/pnas.0908571106

APPLIED BIOLOGICAL SCIENCES, ENGINEERING

Correction for "A modular and extensible RNA-based generegulatory platform for engineering cellular function," by Maung Nyan Win and Christina D. Smolke, which appeared in issue 36, September 4, 2007, of Proc Natl Acad Sci USA (104:14283-14288; first published August 20, 2007; 10.1073/ pnas.0703961104).

The authors note that on page 14284, right column, starting on line 28 of the second full paragraph, "An ≈25-fold increase in target expression levels at 5 mM theophylline, relative to those in the absence of effector, was observed in L2bulge1 (Fig. 2C and SI Fig. 8). In contrast, an \approx 18-fold reduction in expression levels at 5 mM theophylline, relative to those in the absence of effector, was observed in L2bulgeOff1 (Fig. 2D and SI Fig. 8)" appeared incorrectly. The text should instead read: "An increase in target expression levels (induction in fold ≈25) at 5 mM theophylline, relative to those in the absence of effector, was observed in L2bulge1 (Fig. 2C and SI Fig. 8). In contrast, a reduction in expression levels (repression in fold ≈18) at 5 mM theophylline, relative to those in the absence of effector, was observed in L2bulgeOff1 (Fig. 2D and SI Fig. 8)." In addition, the authors note that all switch dynamic range data (Figs. 2-6) reported as induction or repression or normalized gene expression in fold reflect the definitions described in SI Text (Materials and Methods). These errors do not affect the conclusions of the article.

www.pnas.org/cgi/doi/10.1073/pnas.0908785106

Correction for "DNA-binding specificity and in vivo targets of Caenorhabditis elegans nuclear factor I," by Christina M. Whittle, Elena Lazakovitch, Richard M. Gronostajski, and Jason D. Lieb, which appeared in issue 29, July 21, 2009, of *Proc Natl Acad Sci* USA (106:12049–12054; first published July 7, 2009; 10.1073/ pnas.0812894106).

The authors note that due to a printer's error, on page 12053, left column, the first line of the second full paragraph, "Despite the specific nature of the motif over-representation, few stringent criteria for peak definition" should instead read "Despite the specific nature of the motif over-representation, few in vivo targets were identified. As demonstrated in the text, the low number of in vivo NFI-1 targets cannot be explained by overly stringent criteria for peak definition." This error does not affect the conclusions of the article.

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