Corrections

Novel Phosphorylation Target in the Serum Response Factor MADS Box Regulates α-Actin Transcription, by Dinakar Iyer, Narasimhaswamy Belaguli, Martin Flück, Brian G. Rowan, Lei Wei, Nancy L. Weigel, Frank W. Booth, Henry F. Epstein, Robert J. Schwartz, and Ashok Balasubramanyam,* Volume 42, Number 24, June 24, 2003, pages 7477−7486.

Page 7484. With regard to Figure 7, we have discovered a sequence error in the construct SRFT159D, the phosphomimetic mutant of SRF at amino acid T159. We re-made this construct, confirmed the entire sequence, including the desired SRFT159D mutation, and repeated the experiments described in Figure 7. We discovered that SRFT159D actually binds very weakly to the cardiac actin promoter, to an extent much lower than that indicated in the original figure. However, the transcription (luciferase) assays of cardiac actin promoter activity showed results very similar to those in the original figure.

In summary, the results displayed in Figure 7 should be corrected as follows: For panel A, no change in the luciferase activity assay results is necessary. For panel B, aspartic acid substitution of threonine 159 markedly weakens binding of SRF to the cardiac actin promoter.

The corrected text interpreting Figure 7 follows:

The aspartic acid substitution of threonine 159 weakened the interactions of SRF with the cardiac α-actin promoter significantly compared to wt SRF. Modulation of SRF−DNA binding characteristics is unlikely to be the mechanism by which threonine 159 phosphorylation enhances α-actin gene transcription.

Thus, the overall conclusion from the data of Figure 7 does not change from the original text. We are investigating the mechanism by which SRFT159D weakens SRF−DNA binding but still accelerates α-actin gene transcription.

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