Erratum

Key Protein Alterations Associated with Hyperdynamic Cardiac Function: Insights Based on Proteomic Analysis of the Protein Phosphatase 1 Inhibitor-1 Overexpressing Hearts

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As the result of an error during the preparation of the January-February, 2007, issue of the HJC, the above article was incorrectly published as a "Review Article", rather than under the correct heading "Original Research". The abstract submitted with the manuscript follows.

Transgenesis based on organ specific gene expression has provided the basis to elucidate the functional role of proteins for the past 15 years. Using this technology, we showed that inhibition of the protein phosphatase 1, by its constitutively active inhibitor-1, significantly increases cardiac contractility and calcium handling. To uncover protein changes accompanying the chronic increases in cardiac function of these transgenic hearts, we analyzed the cardiac proteome. Interestingly, we found significant increases in the levels of 6 proteins involved in metabolism, calcium binding and scavenging of oxido-reductive stress. These proteins were identified as: hydroxyacyl CoA dehydrogenase II, alpha subunit of the mitochondrial proton ATPase, peroxiredoxin 2, a novel EF-hand containing protein-2, annexin 5, and a previously uncharacterized cDNA. Thus, long-term cardiac specific overexpression of the protein phosphatase 1 inhibitor-1 and the associated increases in cardiac contractility appear to herald changes in a rather small number of proteins, which may reflect important compensatory adaptations in a hyperdynamic heart.