The antiapoptotic protein clusterin protects cardiomyocytes against ischemia-induced cell death

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TO THE EDITOR: With great interest we have read the recent article by Singla and McDonald (3) showing the release of clusterin from embryonic stem cells and documenting the conditioned protective effect of culture medium containing clusterin and other antiapoptotic factors on rat cardiomyoblast cell line H9c2 exposed to H2O2. In their discussion, they state that these antiapoptotic factors, clusterin included, are antiapoptotic in cancer cells, neuroblastoma cells, PC12 cells, and endothelial cells but that such activity has not been demonstrated in isolated cardiomyocytes or in H9c2 cells. We would like to point out that we have shown previously that exogenous secretory clusterin added to H9c2 cells or isolated ventricular rat cardiomyocytes protected both these cells against ischemia-induced cell death as measured by morphological criteria and cell damage markers annexin V and propidium iodide using flow cytometry and fluorescent microscopy (1).

The interesting data of Singla and McDonald (3) support the hypothesis that clusterin is potentially beneficiary in the reperfused myocardium after acute myocardial infarction. The manner(s) in which clusterin protects cardiomyocytes in the reperfused myocardium is not yet clear. It is, of course, known that clusterin colocalizes with complement on ischemic cardiomyocytes (1, 4) and that clusterin is a potent complement inhibitor (2). We have shown that secreted clusterin, in addition, is able to protect cardiomyocytes against ischemic death independent of complement (1). The experiments of Singla and McDonald and our previous results thus suggest that clusterin secreted from embryonic stem cells may protect cardiomyocytes in the reperfused myocardium after acute myocardial infarction.

REFERENCES