Heart cell damage: what leads to cell death? Human embryonic stem cells reveal new insights

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The results, now published in the journal Stem Cell Reports, show that BNA is a versatile â€” and potentially important â€” gene for determining gene expression in cardiac myocytes. Once a cardiac myocyte is purified, transcription factor is attracted to the gene and begins to use BNA as an entryway into the geneâ€™s envelope.

Some cardiac myocytes express the messenger RNA of the participating gene. But not all cardiac myocytes express the collaborating messenger RNA, which is distinct from the messenger RNA of the gene. Without a translation function, the communications between genes are more affected. That means two genes, called NaC-KCATPase and NaC-NHBPase, are disrupted. In addition, nanobody proteins corresponding to both genes can be produced in the cardiac myocytes. In addition, some of the Nanobody cells can cross-react with NaC-HCATPase and NaC-NHBPase molecules, constituting a complex of NaC-GCATPase-NaC-HCATPase and Ni-NHBPase-Nans-GCATpase. BNA is the intermediate site where these complicated groups of proteins assemble. BNA is usually found in the mitochondria (and â€œmuâ€) adjacent to the CEF protein, and the researchers found that it played a crucial role in inducing the expression of the NaC-KCATPase-NaC-NHBPase complex in the cell. BNA can also inhibit the production of NaC-HCATPase and/or NaC-HCATPase-Nans-GCATpase. The researchers say that this finding could not only yield new insights into the processes involved in cardiac contraction (and conversely, the processes involved in cardiac myocytesâ€™ recovery from contraction during cardiac arrest), but also point the way to therapeutic interventions. Interleukin-1 is used as a marker for cardiac cell death. â€œIf the mitochondrial and the NiFAN and NiZW factors can be inhibited, the failure of cardiac cells will be prevented and even reversed,â€ said Stanfordâ€™s Imelda Okuma, who helped author the paper. â€œIn the future, based on the current findings, the technique could be used to develop therapeutics for heart failure, such as treatments to prevent cell death or overcome the resulting inflammation.â€ Additional Stanford co-authors include Gennaro De Martino and Shiham Gammal of the Department of Cell Biology and Anatol Svendsen of the School of Medicine. The work was supported by the Medical College of Wisconsin, European Union 7th Framework Program (FP7), NIH 1R01 HL086081-01, Stanford Cardiovascular Health Network Scholar Award (5T32HL100735-01), the National Heart, Lung, and Blood Institute (HL-118610 and HL-135204), and the Ericsson Medical Research Institute (RR-4-001). For more on stem cell research, visit stanspermune.stanford.edu. More information on stem cell biology and stem cell research can be found at stemcellbiology.stanford.edu.

A Fire Hydrant In The Middle Of A Forest