Embryonic heart exhibits impressive regenerative capacity

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A new study demonstrates that the embryonic mouse heart has an astounding capacity to regenerate, a phenomenon previously observed only in non-mammalian species. The research, published by Cell Press in the October 14th issue of the journal *Developmental Cell*, describes the previously unrecognized potential of the embryonic heart to replace diseased tissue through compensatory proliferation of healthy cells.

Disorders of the mitochondria, a cell structure required for energy production, are one of the leading causes of fatal early onset cardiomyopathies. To investigate how mutations that interfere with mitochondrial function impact the heart during development, Professor Timothy C. Cox (from the University of Washington in Seattle) and colleagues used a heart-specific knockout approach in mice to inactivate a gene crucial for normal mitochondrial function. Their experimental methods established embryonic female mice with mosaic hearts composed of mixed cell populations: half normal and half "diseased" (lacking the gene). However, surprisingly, at birth the diseased cells represented only about 10% of the cardiac tissue.

The authors went on to show that increased proliferation of healthy heart cells was responsible for this change and led to a fully functional heart. Nevertheless, despite normal cardiac function early in life, over 40% of adult mice prematurely developed cardiac pathologies which may indicate a hitherto unsuspected embryological origin for early onset cardiac disease in humans.

"Our findings reveal an impressive regenerative capacity of the fetal heart that can compensate for an effective loss of half of the cardiac tissue," concludes Professor Cox. "To the best of our knowledge, this represents the first in vivo demonstration of selection against diseased tissue during embryonic heart development." The work also suggests that some cell populations within the heart are better able to regenerate than others and that those others are likely to be the source of later pathology.

Source: Cell Press