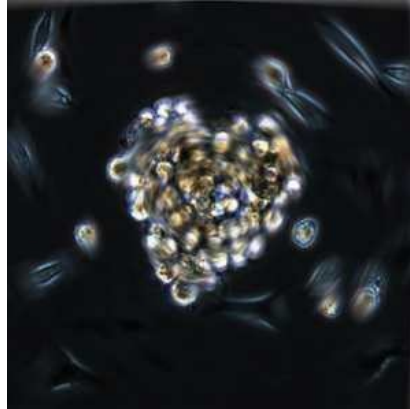




Rejuvenating the Heart



Heart cells have the ability to regenerate themselves and proliferate into new as young cells degrade with age. In a new and simplified model described by Mark Sussman, chief research scientist at the San Diego State University Heart Institute, the heart seems to be quite resistant to developing cancerous cells.

Heart cancer is almost unheard of, explains Sussman, mainly because heart cells are extremely careful when it comes to proliferating. However, this meticulousness ends up making heart disease an intractable problem. As cells continue to burn out over time, their ability to repair themselves and generate fresh replacements gets worse. Thus, as we grow older, we start to experience age-related heart disease because our cardiac cells are unable to properly divide into new cells.

Sussman and his colleagues recently published a paper in the *Journal of Biological Chemistry* where they talk about their new research which is based on using the proliferative and survival properties of cancer-prone cells to rejuvenate cardiac progenitor cells.

They explored the results of an enzyme Pim which is believed to be associated with growth and survival of certain types of cancer cells, and caused it to be overexpressed in cardiac progenitor cells in mice. Pim when teamed up with another gene, Myc, causes tumours but the Pim/Myc combination does not have the same effect in heart progenitor cells which means that they can be tweaked to overexpress the PIM1 gene without raising the risk of cancer.

Sussman's team did that by modifying mouse heart progenitor cells to overexpress PIM1 in specific locations within the cell, targeting specific locations with more of the critical Pim enzyme in hopes that it would protect against ageing-related heart disease. The results showed that compared to the control group, mice with overexpressed PIM1 lived longer and showed stronger cell proliferation. However, the results were different depending on where in the cell the gene was overexpressed.

If the researchers caused PIM1 to be overexpressed in the progenitor cell's nucleus, they saw increased proliferation into new cells. If they overexpressed the gene in a different region of the cell, the mitochondria, they found that the enzyme inhibited the cell's natural self-destruct signals, causing them to live longer. While one technique enhanced cell division, the other warded off cell death.

The results were replicated with human tissue from people whose hearts have failed and who are living on a ventricular assist device that pumps their blood for them.

"We're trying to dial back the clock to when their cells had more regenerative potential," Sussman said. "By understanding how and where Pim affects these cells, we can create specialized Pim molecules that get you all

the benefits of youthfulness without the risk of cancer."

Source: [San Diego State University](#)

Image Credit: Mark Sussman

Published on : Mon, 3 Aug 2015