Can we protect the heart from heart attack damage?

The trouble with heart muscle tissue is that once it's dead…it's dead! During a heart attack, the area of the heart that has been deprived of blood, and therefore oxygen, can become permanently damaged. However, heart cells can "resist" damage caused by reduced oxygen although how this works is not well understood. One molecule that seems to be important in this process is adenosine. Adenosine moves in and out of cells through specialized gateway proteins in the membrane known as transporters. Since adenosine seems to help heart cells resist stresses like lack of oxygen, we theorized that the transport proteins would also be important in overall heart cell health. Therefore, we investigated the role of one transport protein known as ENT1, in heart physiology, using a mouse "knockout" model where the ENT1 protein has been genetically removed. We mimicked a heart attack (through occlusion of the coronary artery) in both normal and knockout mice and found that these knockout mouse hearts showed significantly LESS cellular damage compared to normal mouse hearts with the ENT1 protein. We believe there is less damage in the knockout mouse heart because adenosine cannot be taken up into cells via ENT1 and therefore stays outside of cells for longer, allowing it to signal through adenosine receptors to activate cellular response mechanisms that protect the cell from stress.

In conclusion, our data demonstrate that ENT1 plays an essential role in mediating the effects of adenosine cardioprotection and this research model could lead to future studies aimed at the development of drugs targeted to inhibit ENT1 protein and help protect the heart from damage during a heart attack.

Rose JB, Naydenova Z, Bang A, Eguchi M, Sweeney G, Choi DS, Hammond JR, **Coe IR**. Equilibrative Nucleoside Transporter 1 (ENT1) plays an essential role in cardioprotection. Am J Physiol Heart Circ Physiol 298(3): H771–H777, 2010.

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