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Zsuzsa Szondy

University of Debrecen, /Department of Basic Medical Sciences,
Faculty of Dentistry, Debrecen, Hungary

Adenosine receptors harmonize the degree of inflammation and the consequent tissue repair with the degree of injury

Abstract: Skeletal muscle regeneration is a complex process orchestrated by multiple interacting steps. An increasing number of reports indicate that inflammatory responses play a central role in linking initial muscle injury responses to timely muscle regeneration following injury. Adenosine has been known for a long time as an endogenously produced anti-inflammatory molecule that is generated from ATP released from the dying cells during tissue injury. It mediates its physiological effects via four types of adenosine receptors that respond to different concentrations of adenosine. From these, adenosine A3 receptors (A3Rs) are not expressed by the skeletal muscle, but are present on the surface of various inflammatory cells. In the present paper, the effect of the loss of A3Rs was investigated on the regeneration of the tibialis anterior (TA) muscle in mice following cardiotoxin-induced injury. Here we report that regeneration of the skeletal muscle from A3R^{-/-} mice is characterized by a stronger initial inflammatory response resulting in a larger number of transmigrating inflammatory cells to the injury site, faster clearance of cell debris, enhanced proliferation and faster differentiation of the satellite cells, and increased fusion of the generated myoblasts. This leads to accelerated skeletal muscle tissue repair and formation of larger myofibers. Bone marrow transplantation experiments revealed that the increased response of the tissue-resident cells to tissue injury is responsible for the observed phenomenon. Interestingly, similar results can be obtained by injecting an adenosine A2B receptor agonist, while opposite in A2AR^{-/-} mice. Altogether our data indicate that A3Rs are negative, while A2Rs are positive regulators of the injury-related regenerative inflammation, and consequently also that of the muscle fiber growth in the TA muscle.

Keywords: skeletal muscle, cardiotoxin-induced injury, adenosine receptors, tissue repair

Biography: Zsuzsa Szondy received her MD in 1983 in the University of Medical School of Debrecen. She continued her studies in Indiana and in Oxford University to receive her PhD in 1990. Since then she is employed by the University of Debrecen, Hungary, where she became a full professor in 2003. Her scientific interest is efferocytosis and its biological importance in the thymus, in the skeletal muscle and in the adipose tissue. She published around 90 papers. She was awarded with the prizes "Women in Science" (l'Oréal and UNESCO), Master Teacher (Ministry of Education, Hungary), and was elected to be member of the ECDO Academy.