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Autophagy-mediated defense response and mitochondrial remodeling in mouse mesenchymal stromal cells subjected to bacterial challenge

Nikolai V. Gorbunov
The Henry M. Jackson Foundation for the Advancement of Military Medicine, Inc., USA

Mesenchymal cell lineages are major constituents of bone marrow stroma, which plays a crucial role in homeostasis of the marrow-blood barrier under normal and pathophysiological conditions such as sepsis. In systemic infections the mesenchymal stromal cells (MSCs) affected by circulating pathogen factors can trigger a cascade of adaptive stress-response mechanisms, and thus, maintain the barrier integrity. The data presented in this communication demonstrate that in vitro challenge of MSCs, i.e., colony-forming unit fibroblasts (CFU-F), with *Escherichia coli* and *Staphylococcus epidermidis* induced up-regulation of a complex antibacterial defense response and bacterial phagocytosis mediated by the autophagy mechanisms. The observed up-regulation of autophagy was characterized by increase in (i) expression of ATG5 and ATG8/LC3 autophagy proteins; (ii) amounts of LC3-puncta; (iii) autophagic flux; (iv) autolysosomal degradation of the phagocytized bacteria; and (v) autophagy-mediated secretory activity. The MSC response to the bacteria-produced stress was associated with induction of Pink1 and Parkin proteins, increase in translocation of Drp1, Pink1, and Parkin proteins toward mitochondria, and remodeling of mitochondrial network via fission and fusion events. The following mitophagy and biodegradation of aberrant mitochondria segregated in fission was mediated by Pink1/Parkin-dependent signaling mechanism. The presented data suggest that bone marrow MSCs can elicit autophagy-mediated antibacterial innate defense and stress-adaptive mechanisms in order to maintain integrity of stromal barrier under septic conditions.

Biography

Nikolai V. Gorbunov is Research Associate Professor at the Henry M. Jackson Foundation for the Advancement of Military Medicine Inc. He received his Ph.D. from the Russian Academy Sciences in St. Petersburg, Russia. He then completed a two-year postdoctoral training in Biomedicine at the Research Center of the Consorzio "Mario Negri" Sud (Italy) and a DOE training program at the University of Pittsburgh, Pittsburgh, Pennsylvania. In 1994 and 2000 he was a recipient of the National Research Council Fellowship Awards at the Walter Reed Army Institute of Research (WRAIR). He has a long-term interest is the tissue barrier homeostasis in traumatic injury, inflammation, stress, and immunosuppression. Working on models of blast-related injury, he proposed a mechanism of inflammatory response to the shock wave - induced pulmonary trauma and defined the role of cell redox signaling in the related pulmonary microvascular remodeling. His recent research has been focused on role of autophagy in defense response of tissue barriers to radiation-induced injury and associated bacterial sepsis.

nikolaiv.gorbunov@gmail.com