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Modification of the ribosome as part of the adaptive response to oxidative stress in yeast.

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Living organisms are constantly exposed to a variety of environmental and internal stressors that are detrimental to their cellular physiology and viability. One such condition, oxidative stress, is caused by abnormal amounts of Reactive Oxygen Species (ROS) that can lead to damage to proteins, nucleic acids, and lipids. Although the mechanisms to neutralize ROS have been widely studied, the understanding of ROS-mediated signaling for these mechanisms is rather incomplete and sparse. We have uncovered a previously undescribed phenomenon of yeast ribosomes to respond to elevated levels of ROS through a

site-specific endonucleolytic cleavage of the 25S rRNA in the c-loop of the expansion segment 7 (ES7c) regions. ES7c is a putative regulatory region located on the surface of the 60S ribosomal subunit. This cleavage was detected at the early stages of stress and is not a part of cell death. Additionally, we have seen that low level exposure to oxidative stressors can lead to a protective effect against this cleavage when cells are exposed to higher levels of the stressor. We conclude that ES7c cleavage represents an early and sensitive marker of increased ROS levels in yeast cells and propose that changes in ribosomes may signify an adaptive response to oxidative stress.