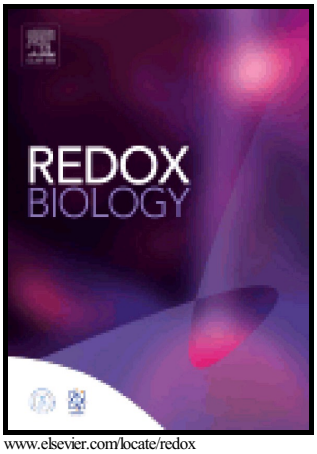


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# Proteostasis, Oxidative Stress and Aging

Ioanna Korovila[1], Martín Hugo[1], José Pedro Castro[1,2,5,6], Daniela Weber[1,4], Annika Höhn[1,2], Tilman Grune[1,2,3,4], Tobias Jung[1,3]

[1]Department of Molecular Toxicology, German Institute of Human Nutrition Potsdam-Rehbruecke (DIfE), 14558 Nuthetal, Germany
[2]German Center for Diabetes Research (DZD), 85764 Muenchen-Neuherberg, Germany
[3]German Center for Cardiovascular Research (DZHK), 10117 Berlin, Germany
[4]NutriAct – Competence Cluster Nutrition Research Berlin-Potsdam, 14558 Nuthetal, Germany
[5]Faculty of Medicine, Department of Biomedicine, University of Porto, 4200-319, Portugal
[6]Institute for Innovation and Health Research (I3S), Aging and Stress Group, R. Alfredo Allen, 4200-135 Porto, Portugal



## Abstract

The production of reactive species is an inevitable by-product of metabolism and thus, life itself. Since reactive species are able to damage cellular structures, especially proteins, as the most abundant macromolecule of mammalian cells, systems are necessary which regulate and preserve a functional cellular protein pool, in a process termed “proteostasis”. Not only the mammalian protein pool is subject of a constant turnover, organelles are also degraded and rebuild. The most important systems for these removal processes are the “ubiquitin-proteasomal system” (UPS), the central proteolytic machinery of mammalian cells, mainly responsible for proteostasis, as well as the “autophagy-lysosomal system”, which mediates the turnover of organelles and large aggregates.

Many age-related pathologies and the aging process itself are accompanied by a dysregulation of UPS, autophagy and the cross-talk between both systems. This review will describe the sources and effects of oxidative stress, preservation of cellular protein- and organelle-homeostasis and the effects of aging on proteostasis in mammalian cells.

**Keywords**

Redox shift, oxidative stress, proteasome, autophagy, lysosome

## Introduction

One of the main “primary” free radicals in mammalian cells is the superoxide radical anion ($O_2^{\bullet -}$), resulting from electrons taken up by molecular oxygen. In a broad variety of secondary/further reactions,