FEATURES

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Involvement of ALAD-20S Proteasome Complexes in Ubiquitination and Acetylation of Proteasomal α2 Subunits

Sara M. Schmitt, Christine Neslund-Dudas, Min Shen, Cindy Cui, Bharati Mitra, and Q. Ping Dou

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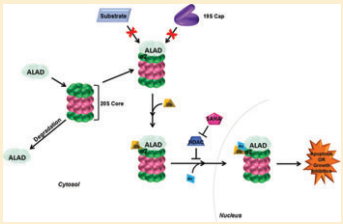
The ubiquitin-proteasome pathway has gained attention as a potential chemotherapeutic target, owing to its importance in the maintenance of protein homeostasis and the observation that cancer cells are more dependent on this pathway than normal cells. Additionally, inhibition of histone deacetylases (HDACs) by their inhibitors like Vorinostat (SAHA) has also proven a useful strategy in cancer therapy and the concomitant use of proteasome and HDAC inhibitors has been shown to be superior to either treatment alone. It has also been reported that delta-aminolevulinic acid dehydratase (ALAD) is a proteasome-associated protein, and may function as an endogenous proteasome inhibitor. While the role of ALAD in the heme biosynthetic pathway is well characterized, little is known about its interaction with, and the mechanism by which it inhibits, the proteasome. In the present study, this ALAD-proteasome complex was further characterized in cultured prostate cancer cells and the effects of SAHA treatment on the regulation of ALAD were investigated.



Regulation of Adipose Tissue Stem Cells Angiogenic Potential by Tumor Necrosis Factor-Alpha

Ekaterina S. Zubkova, Irina B. Beloglazova, Pavel I. Makarevich, Maria A. Boldyreva, Olga Yu. Sukhareva, Marina V. Shestakova, Konstantin V. Dergilev, Yelena V. Parfyonova, and Mikhail Yu. Menshikov

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Tissue regeneration requires coordinated "teamwork" of growth factors, proteases, progenitor and immune cells producing inflammatory cytokines. Mesenchymal stem cells (MSC) might play a pivotal role by substituting cells or by secretion of growth factors or cytokines, and attraction of progenitor and inflammatory cells, which participate in initial stages of tissue repair. Due to obvious impact of inflammation on regeneration it seems promising to explore whether inflammatory factors could influence proangiogenic abilities of MSC. In this study we investigated effects of TNF-α on activity of adipose-derived stem cells (ADSC). We found that treatment with TNF-α enhances ADSC proliferation, F-actin microfilament assembly, increases cell motility and migration through extracellular matrix.