



Function of Sirtuins in Stem Cell Differentiation

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DESCRIPTION

Sirtuins perform as radars of environmental stimuli and organize the stress response of cells, deregulation of these proteins is associated with cancer. Also, some sirtuins appear to be involved in cell senescence and aging, although some of the experimental models reported are controversial.

Sirtuins are involved in several cellular functions and can target an inclusive array of proteins, both histone and nonhistone. SIRT1 is the most closely related and investigated homolog of the yeast *Sir2* gene. Many biological functions have been associated with it, although its key function seems to be linked to gene silencing through heterochromatin formation. SIRT1 specially deacetylates lysine 6 of histone H4 (H4K6) and lysines 9 and 56 of histone H3 (H3K9 and H3K56), promoting the construction of facultative heterochromatin. Additionally, SIRT1 can acetylate and convert histone H1 to the chromatin, increasing local compaction. In addition, it can deacetylate other nonhistone targets that also contribute to heterochromatin formation, such as the histone methyltransferase Suv39h1, thereby promoting dimethylation and trimethylation of lysine 9 in histone H3 and contributing to heterochromatin formation as the transition from acetylation to methylation in H3K9 spreads over gene-coding regions. SIRT1 plays a role in gene expression as well as cellular homeostasis in response to stress, SIRT1 can acetylate proteins such as FOXO, p53, and the transcription factor NF- κ B, all involved in cell cycle progression, DNA repair, and apoptosis.

Sirtuins are very preserved NAD⁺-dependent enzymes that are able to remove a wide range of lipid lysine acyl-groups from protein substrates in a NAD⁺-dependent manner. These NAD⁺-dependent actions enable sirtuins to monitor cellular energy status and modulate gene transcription, genome stability, and energy metabolism in response to environmental signals. So, sirtuins are vital for cell survival, stress resistance, proliferation,

and differentiation. In recent years, sirtuins are gradually familiar as central regulators of stem cell biology in addition to their well-known roles in metabolism and aging.

SIRT2 is a cytoplasmic tubulin deacetylase that can confine in the nucleus during G2/M transition. It is a protein that has been well conserved throughout evolution, and its chief function is related to cell cycle regulation. It can deacetylate microtubules and chromatin *in vivo*. Acetylation in α -tubulin was stated to make steady microtubules, although SIRT2^{-/-} knockout mice do not show clear defects in microtubule organization, and consequently, the role of SIRT2 in microtubule function and organization remains indistinct. In chromatin, SIRT2 deacetylates H4K16 at the global level to upsurge chromatin compaction during G2/M transition; the molecular mechanism that initiates this method is, however, unknown.

Genomic stability, DNA repair, and gene silencing have all been linked to SIRT6 function. It can deacetylate histones in gene promoters and telomere chromatin in H3K9Ac, a residue in the histone tail, and H3K56, a residue in the core of histone H3, contributing to heterochromatin formation and telomere stability.

CONCLUSION

Not much is known about the remaining sirtuins: SIRT4 is a mitochondrial sirtuin without deacetylase activity and is deceptively involved in the insulin metabolism of pancreatic β cells. SIRT5 targets cytochrome C and carbamoyl phosphate synthase 1 in mitochondria. Amusingly, it has been detected, both *in vitro* and *in vivo*, that SIRT5 has a very strong demalonylase and desuccinylase activity, which suggests additional functions beyond lysine deacetylation. Lastly, SIRT7 is a protein present in the nucleolus that interacts with and triggers RNA polymerase I but does not deacetylate Pol I, and its molecular mechanism is presently unknown.

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