SOD citations

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# Expanding roles of superoxide dismutases in cell regulation and cancer

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## Highlights

Superoxide dismutases (SODs) have important regulatory functions in metabolism, signalling and transcription.

SODs are crucial for cancer cell growth, proliferation, survival and metastasis.

SODs are potential therapeutic targets for drug and radiation therapy for human cancer.

Reactive oxygen species (ROS) have important roles in normal physiology and diseases, particularly cancer. Under normal physiological conditions, they participate in redox reactions and serve as second messengers for regulatory functions. Owing to aberrant metabolism, cancer cells accumulate excessive ROS, thus requiring a robustly active antioxidant system to prevent cellular damage. Superoxide dismutases (SODs) are enzymes that catalyze the removal of superoxide free radicals. There are three distinct members of this metalloenzyme family in mammals: SOD1 (Cu/ZnSOD), SOD2 (MnSOD) and SOD3 (ecSOD). SODs are increasingly recognized for their regulatory functions in growth, metabolism and oxidative stress responses, which are also crucial for cancer development and survival. Growing evidence shows that SODs are also potentially useful anticancer drug targets. This review will focus on recent research of SODs in cellular regulation, with emphasis on their roles in cancer biology and therapy.

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# Extracellular superoxide dismutase and its role in cancer

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## Highlights

EcSOD is significantly downregulated across a vast majority of cancers.

Overexpression of EcSOD resulted in tumor suppressive effects.•

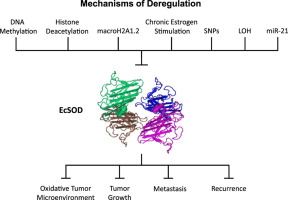
Low EcSOD expression confers poor prognosis in cancer patients.•

Deregulation of EcSOD is associated with both genetic and epigenetic mechanisms.

## Abstract

[Reactive oxygen species](https://www.sciencedirect.com/topics/medicine-and-dentistry/reactive-oxygen-species) (ROS) are increasingly recognized as critical determinants of [cellular signaling](https://www.sciencedirect.com/topics/medicine-and-dentistry/signal-transduction) and a strict balance of ROS levels must be maintained to ensure proper cellular function and survival. Notably, ROS is increased in cancer cells. The superoxide dismutase family plays an essential physiological role in mitigating deleterious effects of ROS. Due to the compartmentalization of ROS signaling, EcSOD, the only superoxide dismutase in the [extracellular space](https://www.sciencedirect.com/topics/medicine-and-dentistry/extracellular-space), has unique characteristics and functions in cellular signal transduction. In comparison to the other two intracellular SODs, EcSOD is a relatively new comer in terms of its tumor suppressive role in cancer and the mechanisms involved are less well understood. Nevertheless, the degree of differential expression of this extracellular antioxidant in cancer versus normal cells/tissues is more pronounced and prevalent than the other SODs. A significant association of low EcSOD expression with reduced cancer patient survival further suggests that loss of extracellular redox regulation promotes a conducive [microenvironment](https://www.sciencedirect.com/topics/medicine-and-dentistry/microenvironment) that favors cancer progression. The vast array of mechanisms reported in mediating deregulation of EcSOD expression, function, and [cellular distribution](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/subcellular-localization) also supports that loss of this extracellular antioxidant provides a selective advantage to cancer cells. Moreover, overexpression of EcSOD inhibits [tumor growth and metastasis](https://www.sciencedirect.com/topics/medicine-and-dentistry/metastatic-carcinoma), indicating a role as a tumor suppressor. This review focuses on the current understanding of the mechanisms of deregulation and tumor suppressive function of EcSOD in cancer.

## Graphical abstract



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# Topical application of superoxide dismutase mediated by HIV-TAT peptide attenuates UVB-induced damages in human skin

[XiaochaoChen](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[ab](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[ShutaoLiu](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[a](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[PingfanRao](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[a](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[JeremyBradshaw](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[c](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[RichardWeller](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)[b](https://www.sciencedirect.com/science/article/abs/pii/S0939641116303423" \l "!)

## Highlights

Topical application of superoxide dismutase can be enhanced by binding to HIV-TAT peptide.

TAT-SOD protein efficiently penetrated through the stratum corneum.

TAT-SOD protein significantly attenuated UVB-induced skin damage in man.

## Abstract

The purpose of this study was to evaluate whether topical application of superoxide dismutase with cell penetrating peptide (HIV-TAT) could protect against skin damage induced by UVB irradiation in humans. The permeability through stratum corneum of large proteins linked to TAT peptide was firstly confirmed by confocal microscopy and tape stripping. Ten healthy volunteers with either Fitzpatrick skin type II or III were recruited in this clinical study. TAT-SOD (300 units/cm2) and vehicle cream were applied on two symmetric areas of both inner upper arms 1 h prior to UVB irradiation. After one hour of pretreatment, subjects received 10 incremental doses of UVB on pretreated areas. 24 h later, erythema, blood flow and apoptotic cells were measured. Pretreatment with TAT-SOD 1 h prior to UVB radiation promoted a mean minimal erythema dose (MED) increase of 36.6 ± 18.4% (p = 0.013 < 0.05. n = 10) compared to vehicle control. The median blood flow values of all subjects following 2 and 3-MED of UVB were 107.8 ± 51.0 units and 239.5 ± 88.0 units respectively, which account for 26% and 25% decrease with respect to vehicle groups. These data suggest that TAT-SOD significantly suppresses UVB induced erythema formation and blood flow rise. Furthermore, pretreatment with TAT-SOD 1 h prior to 2-MED of UVB irradiation reduced the apoptotic sunburn cell formation by 47.6 ± 8.6% (p < 0.0001) in all subjects. Evaluating results generated from all measurements, we conclude that topical application of TAT-SOD significantly attenuates UVB-induced skin damage in man. These biological effects of TAT-SOD are probably mediated via its free radical scavenging properties, clearly differentiating it from other physical sunscreen agents.

## Graphical abstract

A close up of text on a white background

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<https://link.springer.com/article/10.1007/s00109-019-01845-2>

# Anti-oxidative effects of superoxide dismutase 3 on inflammatory diseases

* [Nguyen Hoai Nguyen](https://link.springer.com/article/10.1007/s00109-019-01845-2#auth-1),
* [Gia-Buu Tran](https://link.springer.com/article/10.1007/s00109-019-01845-2#auth-2) &
* [Cuong Thach Nguyen](https://link.springer.com/article/10.1007/s00109-019-01845-2#auth-3)

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## Abstract

Free radicals and other oxidants are critical determinants of the cellular signaling pathways involved in the pathogenesis of several human diseases including inflammatory diseases. Numerous studies have demonstrated the protective effects of antioxidant enzymes during inflammation by elimination of free radicals. The superoxide dismutase (SOD), an antioxidant enzyme, plays an essential pathogenic role in the inflammatory diseases by not only catalyzing the conversion of the superoxide to hydrogen peroxide and oxygen but also affecting immune responses. There are three distinct isoforms of SOD, which distribute in different cellular compartments such as cytosolic SOD1, mitochondrial SOD2, and extracellular SOD3. Many studies have investigated the anti-oxidative effects of SOD3 in the inflammatory diseases. Herein, in this review, we focus on the current understanding of SOD3 as a therapeutic protein in inflammatory diseases such as skin, autoimmune, lung, and cardiovascular inflammatory diseases. Moreover, the mechanism(s) by which SOD3 modulates immune responses and signal initiation in the pathogenesis of the diseases will be further discussed.