

Genetic Science Spotlight

French National Institute of Health and Medical Research: Polymorphisms in the SOD2 MTS modulates MnSOD mitochondrial import and its mRNA levels

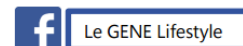
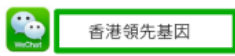


Mitochondrial oxidative stress is proven as an aging factor for our skin. Mitochondria continuously generate potentially damaging reactive oxygen species (ROS) during oxidative phosphorylation, which can be neutralized by Superoxide dismutase 2 (SOD2), also known as manganese superoxide (MnSOD). SOD2 is a mitochondrial protein encoded in the nucleus and subsequently imported into the mitochondria. SOD2 is imported into mitochondria during or after RNA-protein translation, driven by the mitochondrial targeting sequence (MTS). The SOD2 MTS exhibits a genetic dimorphism in humans, with the presence of either Ala (codon GCN) or Val (codon GUN). Ala-SOD2 leads to four-fold higher levels of the exogenous, mature (imported) SOD2 protein ($p < 0.05$) and of the exogenous SOD2 in-gel activity than transfection of a vector encoding for tagged Val-SOD2 ($p < 0.05$).

Sutton A., et al. 2005. The manganese superoxide dismutase Ala16Val dimorphism modulates both mitochondrial import and mRNA stability. *Pharmacogenetics and Genomics*, 15(5): 311-319.

<https://www.ncbi.nlm.nih.gov/pubmed/15864132>

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