

## عنوان مقاله:

Oxidative stress and mitochondrial dysfunctions in personalized cancerous signalling pathway

## محل انتشار:

دومین کنگره بین المللی پزشکی شخصی (سال: 1396)

تعداد صفحات اصل مقاله: 1

## نویسندگان:

Nafise Taromi - *Department of Medical Biotechnology, Faculty of Allied Medical Sciences, Iran University of Medical Sciences, Tehran, Iran*

Neda Saray-Gord Afshari - *Department of Medical Biotechnology, Faculty of Allied Medical Sciences, Iran University of Medical Sciences, Tehran, Iran*

## خلاصه مقاله:

Extracellular/intracellular oxidative stress and mitochondrial dysfunctions are known to play important roles in onset and the progression of the long-term complications in late-stage of numerous cancers. In cancer patients, according to it genetic differences, oxidative stress can be caused by many mechanisms including increased protein glycation and glucose autooxidation, activation of protein kinase C isoforms, and overproduction of superoxide. Down regulation and disruption in function of components of mitochondrial respiratory chain have been implicated as a key factor in the development of most of all cancer complications. Oxidative damages, which caused by increase in mitochondrial reactive oxygen species (ROS) production due to overproduction of free radical and reduction in antioxidant defences, are increased under cancerous conditions. Moreover, alterations in structure and function of mitochondrial DNA (mtDNA), which may be result from increase in rate of mutations, reduction in content and number of mitochondrial DNA and also decreased mitochondrial fusion have been linked to the pathogenesis of almost all cancers. In addition, abnormal mitochondrial reactive oxygen species production may be the main cause of mitochondrial DNA alterations and hyperglycaemic damages, which themselves, can induce the generation of reactive oxygen species. Hence cancer complications and increase in reactive oxygen species levels have direct association with each other and also patient genome structure, inhibition of ROS production or modulation of mitochondrial biogenesis will be one of the .next personalized therapeutic target for cancerous patients

## کلمات کلیدی:

cancer, personalized therapy, oxidative stress, mitochondria, reactive oxygen species, mitochondrial DNA

## لینک ثابت مقاله در پایگاه سیویلیکا:

<https://civilica.com/doc/714162>

