# **Special Issue**

# Oxidative Stress in Diabetes and Complications

## Message from the Guest Editor

Elevated blood glucose is present in all diabetes, and elevated lipids are a feature of type 2 diabetes, which accounts for 75% of all diabetes. Many of the most serious complications of diabetes are a direct consequence of hyperglycemia and dyslipidemia associated with diabetes, which can result in the production of advanced glycation end products (AGES), dysregulation of the polyol pathway, and increased production of lipids and protein derived reactive oxygen species (ROS). These products have particularly been associated with vascular complications of diabetes, such as cardiovascular diseases. This Special Issue seeks to further explore the causes, consequences, and challenges of treating oxidative processes associated with type 1, type 2, and gestational diabetes mellitus to prevent the exacerbation of diabetes and development of diabetic complications. Interventions that may prevent or reverse the damage caused by ROS generated by diabetes are of special interest. However, both original research and critical reviews providing novel insights into the causes and treatments of pathologies related to diabetes-induced oxidative stress are welcome.

## **Guest Editor**

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#### Deadline for manuscript submissions

closed (20 April 2023)



## **Antioxidants**

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## **About the Journal**

## Message from the Editor-in-Chief

It has been recognized in medical sciences that in order to prevent adverse effects of "oxidative stress" a balance exists between prooxidants and antioxidants in living systems. Imbalances are found in a variety of diseases and chronic health situations. Our journal *Antioxidants* serves as an authoritative source of information on current topics of research in the area of oxidative stress and antioxidant defense systems. The future is bright for antioxidant research and since 2012, *Antioxidants* has become a key forum for researchers to bring their findings to the forefront.

## Editor-in-Chief

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