



## SP-A Induces Preterm Birth through Promoting Oxidative Stress via Upregulating STOX1

Xiafang Li<sup>1\*</sup>, Chunlian Zhang<sup>2</sup>

<sup>1</sup> Department of Cell Biology, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing, China

<sup>2</sup> Department of Gynecology and Obstetrics, Chinese Academy of Medical Sciences & Peking Union Medical College Hospital, Beijing, China

Received: 18. June, 2018; Accepted: 20. July, 2018; Published online: 21. July, 2018  
Copyright: © 2018 Li et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Open Access

SP-A is a secreted protein that plays a role in the regulation of cellular processes such as apoptosis, proliferation, and differentiation. It has been implicated in various diseases, including preterm birth. In this study, we found that SP-A induced preterm birth by promoting oxidative stress via upregulating STOX1. SP-A increased the expression of STOX1 in a dose-dependent manner. STOX1 is a transcription factor that regulates the expression of genes involved in oxidative stress, including CAT1, GSH-P, NOS3, ARG2, and HO-1. SP-A also increased the expression of these genes. Additionally, SP-A increased the production of ROS and RNS, leading to DNA damage and cell death. These findings suggest that SP-A may play a role in preterm birth by promoting oxidative stress via upregulating STOX1.

**Keywords:** SP-A; STOX1; oxidative stress; preterm birth

Inflammation is a key factor in the initiation of labor and delivery. SP-A is a secreted protein that has been implicated in various diseases, including preterm birth. In this study, we found that SP-A induced preterm birth by promoting oxidative stress via upregulating STOX1. SP-A increased the expression of STOX1 in a dose-dependent manner. STOX1 is a transcription factor that regulates the expression of genes involved in oxidative stress, including CAT1, GSH-P, NOS3, ARG2, and HO-1. SP-A also increased the expression of these genes. Additionally, SP-A increased the production of ROS and RNS, leading to DNA damage and cell death. These findings suggest that SP-A may play a role in preterm birth by promoting oxidative stress via upregulating STOX1.

SP-A is a secreted protein that plays a role in the regulation of cellular processes such as apoptosis, proliferation, and differentiation. It has been implicated in various diseases, including preterm birth. In this study, we found that SP-A induced preterm birth by promoting oxidative stress via upregulating STOX1. SP-A increased the expression of STOX1 in a dose-dependent manner. STOX1 is a transcription factor that regulates the expression of genes involved in oxidative stress, including CAT1, GSH-P, NOS3, ARG2, and HO-1. SP-A also increased the expression of these genes. Additionally, SP-A increased the production of ROS and RNS, leading to DNA damage and cell death. These findings suggest that SP-A may play a role in preterm birth by promoting oxidative stress via upregulating STOX1.



**SP-A  
STO 1**

STO 1 is a secreted protein that has been implicated in various physiological processes, including lung development and homeostasis. It is also involved in the regulation of cellular responses to oxidative stress. SP-A is a key component of the airway surface liquid and plays a role in the clearance of pathogens and debris from the respiratory tract. It has been shown to bind to various proteins, including SOD, GSH-P, and CAT, which may contribute to its protective effects against oxidative damage. The expression of STO 1 is upregulated by SP-A, suggesting a potential mechanism for SP-A's protective作用 against oxidative stress. Further research is needed to fully understand the complex interactions between SP-A and STO 1 in the context of preterm birth and other diseases.

