The Effects of Oxidative Stress on Sacrcospan- Deficient Mice in the Cardiovascular and Respiratory System

Chinyere Oko, Bolade Olateju, Luaye Samarah, Isela C. Valera, Aida Rahimi Kahmini, Jocelyn Millan, Nazanin Mohammadipoor, and Michelle S. Parvatiyar
Department of Health,Nutrition,and Food Sciences
Florida State University, Tallahassee, FL, USA

Abstract

The use of tobacco is the leading cause of preventable death and disease in the United States. Smoking has increased at an alarming rate, especially with e-cigarettes being easily accessible these days. Smoking has a direct effect on homeostasis functions within the body, but in our studies we looked at the impact it had on the cardiovascular and respiratory system in Sarcospan- deficient mice. In this research we studied the Sarcospan gene (SSPN) which is a part of the dystrophin-associated glycoprotein complex (DGC). The DGC is an important complex that is seen to stabilize the muscle membrane and protect muscle fibers. If the SSPN gene is altered genetically, causing dysfunction to its expression, the individual can be at risk for cardiovascular disease. Using White-Type Mice (WT) , and Sarcospan deficient mice (SSPN), we would subject them to cigarette smoke (CS) for extended amounts of time. This is so we could assess what oxidative stress does to the expression of the SSPN gene, and see how it affects the heart and lungs.

Introduction

The heart is a vital organ that has multiple functions such as pumping oxygenated blood to the body, delivering nutrients and hormones, regulating blood pressure etc...
Susceptibility to cardiac injury can be due to hereditary causes such as mutations in proteins that regulate metabolism, diabetes, high body mass index, or impact by environmental factors such as inhaling too much smoke.
The SSPN Gene has many roles, but specifically in this study, we are looking at what its deletion has on the effect on Cardiovascular and Respiratory function.
The inhalation of smoke can cause the individual to develop cardiomyopathy, which in turn can cause arrhythmias. This can make the heart not pump blood as efficiently as it should.
Lung fibroblasts are important for the structure of the lung, as well as wound healing. Smoking can cause injury to these cells, and could result in scarring of the lung, which is irreversible.

References

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Methods

Using the Smoking Machine- SCIREQ, we conducted the experiment;

- Use 16 mice; 4 Male WT, 4 Female WT, 4 Male (KO), 4 Female(KO)
- Place the mice in the (SCIREQ), making sure it is closed.
- Load 7 cigarettes into the smoking machine and connect the respective tubes, so mice are able to inhale the smoke.
- Lock the machine and turn it on using the computer connected to the machine
- Smoke the mice for 1 hour and 30 minutes, checking them periodically.
- Repeat 5 times a week for 24 weeks to ensure the mice are subjected to oxidative stress

Using the ECHO (Echocardiographic machine) at the Florida State University's College of Medicine we were able to analyze the mice's heart rhythms and cardiac functions. Afterwards we sacrifice the mice in order to analyze their lung fibroblasts.

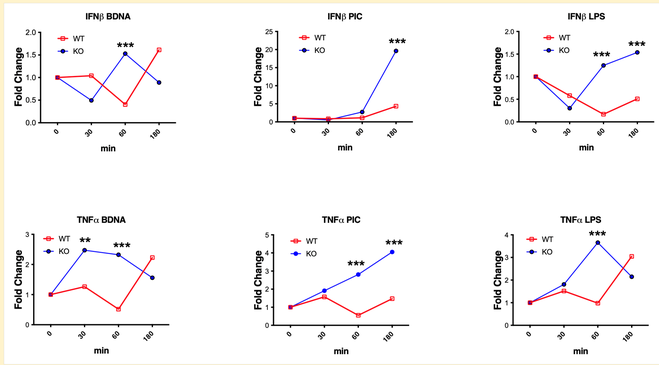
SCIREQ Smoking Machine

Conclusion

In inflammatory and oxidative stress pathways, the SSPN gene is vital for carrying out effective cellular communication relating to protecting and stabilizing muscle proteins. The risk of cardiac injury is greater especially when subjected to environmental conditions such as cigarette smoke (nicotine). This is likely due to ineffective communication between the SSPN and the respective pathways.

Results

Figure 1.1: The impact of SSPN in the lungs fibrotic response.



Results

Figure 1.2 (E-F): Highlights the changes in mouse cardiac function, while being smoked in the ECHO Machine. Left Ventricular fractional shortening increased with mice not subjected to (CS).