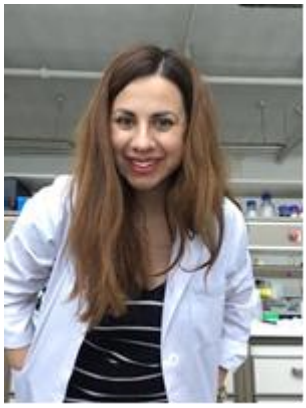
ImmunoTools *special* Award 2024



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"Unlocking the Potential: SGLT2 Inhibitors as a Promising Frontier in Alleviating Ferroptosis-Induced Airway Inflammation in COPD"

Background

Chronic Obstructive Pulmonary Disease (COPD) is a debilitating respiratory condition characterized by persistent airflow limitation and inflammation in the airways. Recent research has unveiled a fascinating connection between ferroptosis, a unique form of regulated cell death driven by iron-dependent lipid peroxidation, and the pathogenesis of airway inflammation in COPD.

Ferroptosis represents a novel mechanism that has been implicated in various diseases, including neurodegenerative disorders and cancer. In the context of COPD, it emerges as a key player in exacerbating inflammation within the airways. The dysregulation of iron homeostasis and subsequent lipid peroxidation contribute to cellular damage, creating a pro-inflammatory microenvironment.

Understanding the molecular intricacies of ferroptosis in COPD is crucial for developing targeted therapeutic interventions. One promising avenue of exploration is the potential role of Sodium-Glucose Co-Transporter 2 (SGLT2) inhibitors in mitigating ferroptosis-induced inflammation. SGLT2 inhibitors, originally designed for diabetes management, have demonstrated anti-inflammatory and antioxidative properties, suggesting a potential dual benefit in managing both metabolic and respiratory aspects of COPD.

The prospect of utilizing SGLT2 inhibitors to ameliorate ferroptosis in COPD opens up new possibilities for personalized and effective treatment strategies. Further research is needed to unravel the specific mechanisms involved and establish the clinical efficacy of these inhibitors in managing ferroptosis-associated airway inflammation in individuals with COPD. This intersection of respiratory medicine and cellular biology holds promise for advancing our understanding and treatment of COPD, offering hope for improved outcomes and enhanced quality of life for those affected by this chronic respiratory condition.

Experimental plan

In our laboratory, we cultivate primary cells derived from lung tissue biopsies obtained from patients with COPD and other respiratory conditions. Our research strategy involves subjecting these cells to erastin, a ferroptosis inducer, followed by attempts to reverse the ensuing inflammatory phenotype using varying concentrations of empagliflozin, an SGLT2 inhibitor. To achieve this objective, we intend to conduct ELISA on cell culture supernatants and perform quantitative PCR and Western-blot analysis on cell lysates and construct relevant heat-maps about the differential gene expression between untreated and treated cells, from COPD patients, patients with other respiratory diseases (IPF, asthma, chronic cough, and sarcoidosis) and healthy controls. These analyses aim to discern variations in key molecules at the intersection of inflammation and ferroptosis pathways.

We will need ELISA kits for IL-6 and IL-1β, as we already have promising results from PCR and a variety of antibodies and a variety of antibodies for human interleukins such as IL-2, IL-4, IL-6, IL-8/CXCL8, IL-10, IL-15, IL-17a and IL-18.

I am earnestly seeking this award to obtain the necessary funding for crucial reagents that are pivotal to the success of our research. Winning this award would significantly contribute to the advancement of our work and enable us to make meaningful contributions to the field.

ImmunoTools *special* AWARD for **Charikleia Ntenti** includes 10 reagents

ELISA: human IL-6 (four reagents)

recombinant human cytokines: IL-2, IL-4, IL-6, IL-8/CXCL8, IL-10, IL-15, and IL-17a.

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