

## **30th "European Respiratory Society" Congress**

### **At the Symposium "Which target to protect and preserve lung health?" we talked about the management of patients with chronic lung diseases and new evidence in COVID-19 scientific research**

*Internationally renowned experts discussed the relationship between oxidative stress and lung health and the potential benefit of chronic therapy with antioxidant drugs in the prevention and treatment of acute and chronic lung diseases.*

**Milan, September 9<sup>th</sup>, 2020** – The Symposium, "**WHICH TARGET TO PROTECT AND PRESERVE LUNG HEALTH?**", sponsored by Zambon, was held this morning during the ERS International Congress 2020. World-renowned experts discussed high social-welfare impact lung diseases, such as COPD (Chronic Obstructive Pulmonary Disease) and IPF (Idiopathic Pulmonary Fibrosis), and some evidence that has emerged during the management of the global health emergency from SARS-COV-2 (COVID-19).

In particular, the scientific debate focused on the recent COVID-19 pandemic, and on the observation of the common denominator between COPD and IPF: oxidative stress, currently identified as a key element for understanding the onset and chronicity of various clinical conditions.

The COVID-19 epidemic has reconfirmed the importance of maintaining an effective protection system at the pulmonary level: the viral insult generates an imbalance between the antioxidant system and free radicals, which, if perpetuated, is one of the causes of post COVID fibrosis. An early restoration of this balance with antioxidant therapy is hoped to limit both the onset and the perpetuation of the damage.

The Symposium experts shared significant updates on oxidative stress and lung damage and discussed possible future developments in clinical research. A potential role of antioxidant therapy is the protection and preservation of lung function, for the frailest patients.

*"It is possible to prevent acute exacerbations in some lung diseases and in particular in COPD, thanks to the use of antioxidants and their anti-inflammatory action<sup>1,2</sup> - stated Prof. Alberto Papi, Pneumological Clinic of the S. Anna University Hospital, University of Ferrara, Chairman and speaker of the Symposium. "Viral infections are some of the main causes of COPD exacerbations<sup>3</sup>, that cause the development of oxidative stress and start a vicious cycle that leads to an increase in inflammation and the severity of the infection.<sup>4, 5</sup> Antioxidant molecules such as glutathione can block this cascade of*

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<sup>1</sup> Rushworth GF. *Pharmacol Ther* 2014; 141(2):150-9.

<sup>2</sup> Bavarsad Shahripour R. *Brain Behav* 2014; 4(2):108-22

<sup>3</sup> GOLD 2017

<sup>4</sup> Papi A, Johnston SL. *FASEB J* 2002

<sup>5</sup> Papi A, Contoli M. *J Biol Chem* 2008

inflammatory events and thus potentially prevent the risk of exacerbation as well.<sup>6, 7, 8, 9.</sup> Physicians should inform COPD patients of the role of NAC in reducing the number of exacerbations and its high safety profile".

*"The imbalance of the oxidant-antioxidant regulatory mechanism is one of the factors contributing to the progression of the disease in patients with pulmonary fibrosis. The most severe form of pulmonary fibrosis is the idiopathic one, which accounts for about 20% of cases<sup>10</sup>. For its treatment, two drugs are now available that can reduce the progression of the disease. NAC, currently being tested, could represent an additional therapeutic option for these patients – stated Prof. Luca Richeldi, A. Gemelli Polyclinic Foundation IRCCS, Catholic University of the Sacred Heart of Rome. "The search for new drugs in order to treat pulmonary fibrosis is still open and is receiving a further impulse from the new form of pulmonary fibrosis that can follow coronavirus infection SARS-Cov-2. In this direction, an Italian study is being evaluated to verify the efficacy and safety of NAC administered orally compared to placebo in patients with a history of SARS-Cov-2 infection and pulmonary fibrosis. One of the potential advantages of this therapeutic approach is represented by the fact that NAC is a drug with an excellent safety profile, already well known by the medical profession".*

The COVID-19 outbreak in Wuhan turned into a world-wide public health emergency. The virus causes acute respiratory syndrome in affected patients, which in severe cases is characterized by pneumonia and acute respiratory distress syndrome, sepsis, and multiple organ failure.

*"In order to manage patients who are very serious or already in intensive care affected by COVID-19, it was essential to try to reduce oxidative stress in the lungs - said Joan B Soriano, epidemiologist at the Pneumology Service of the Hospital de La Princesa in Madrid. "In pneumonia caused by COVID-19, we have seen how the bronchi and lungs are often covered with secretions that resemble 'mucus or snail slime' and which make bronchoscopies very difficult to perform. We also found that this phenomenon is accompanied by an inflammatory outbreak, called 'cytokine storm syndrome'. NAC has been shown to inhibit the reproduction of the influenza virus<sup>11</sup> and thus represents a safe and effective alternative to currently available antithrombotic agents to restore patency of vessels after arterial occlusion. By analogy, therefore, various groups have explored the use of NAC in severe COVID-19 pneumonia".*

Prof. Papi, Chairman of the Symposium concluded: *"this symposium highlighted how the maintenance of oxidant-antioxidant homeostasis is essential to preserve lung function and prevent the progression of chronic diseases and the role it could play in the treatment of IPF or for other forms of fibrosis following COVID-19 infection".*

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<sup>6</sup> Casoni et al CEA, 2003

<sup>7</sup> Sadowska AM, et al. Int J Chron Obstruct Pulmon Dis. 2006;1:425-34.

<sup>8</sup> Dekhuijzen PN, van Beurden WJ. Int J Chron Obstruct Pulmon Dis. 2006;1:99-106.

<sup>9</sup> Hutter D, Greene JJ. J Cell Physiol 2000;183:45-52.

<sup>10</sup> Lederer DJ and Martinez FJ, N Engl J Med 2018

<sup>11</sup> Mata M, et al. Biochemical Pharmacology 2012

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