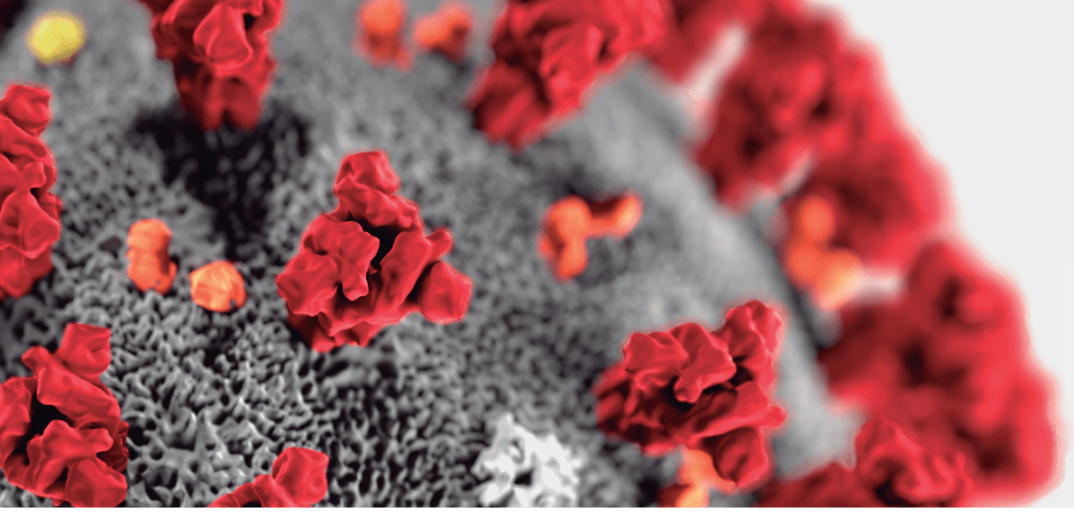

INNOVATIONS IN COVID-19

Bridging opportunities
at Oswaldo Cruz Institute

INVESTIGATION OF MOLECULAR MECHANISMS OF PULMONARY FIBROSIS IN COVID-19
(CÓD. 2020.031)

COORDINATOR

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RESEARCH AREA

New Drugs

DEVELOPMENT STAGE

Level 3 - TRL - Analytical and experimental critical function and/or characteristic proof of concept. MRL - Manufacturing proof of concept developed.

PROPOSITION / APPLICATION

The main organ affected in COVID-19 is the lung and lung failure is the most common cause of death. However, knowledge about its pathophysiology is still low and there is no effective therapy. When investigated "*in vitro*", SARS-CoV-2 infection causes apoptosis of bronchial epithelial cells and pneumocytes. This process stimulates the migration of inflammatory cells to the infection site. The exacerbated inflammatory response, associated to the intense production of cytokines causes oedema in the lung tissue. All of these factors together favour the appearance of fibrosis in the lungs. We believe that the investigation of the molecular mechanisms of fibrosis allows the development of therapeutic strategies for the prevention and / or reduction / reversal of pulmonary fibrosis. This is, therefore, the problem investigated in the present proposal.

INNOVATION

The molecular mechanisms of tissue fibrosis vary according to the affected organs. The cellular and molecular mechanisms of fibrosis in the liver, heart or kidneys are best known for being more common in diseases prevalent in the human population. However, the incidence of pulmonary fibrosis, which occurs in a percentage of cases of severe acute respiratory failure syndrome, is very recent. In this sense, the innovation of the project is precisely to put this issue in evidence thus generating a more accurate knowledge on the pathophysiology of pulmonary fibrosis in COVID-19, which is, in fact, possible therapeutic strategies.

OPPORTUNITY

Investigation of the modulation of fibrotic effects in patients positive for SARS-CoV-2

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