

Zydus files IND application for NLRP3 inhibitor candidate to fight COVID-19

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Zydus has announced that it has filed the IND application of ZYL1, a novel oral small molecule NLRP3 inhibitor candidate. Following up on its initiatives to fight COVID 19 with diagnostics, vaccines and therapeutics, the company is now focussing on cutting edge research to bring targeted therapies that can selectively modulate the inflammatory responses caused by the Cytokine Storm.

NLRP3 inflammasomes are involved in the inflammation process by production and release of proinflammatory cytokines IL-1 β and IL-18. This harmful inflammation within the body leads to the onset and development of various kinds of diseases, including auto-immune diseases, inflammatory diseases, cardiovascular diseases, metabolic disorders, Gastro-intestinal diseases (inflammatory bowel disease), renal diseases, CNS diseases as well as Acute Respiratory Distress Syndrome (ARDS).

SARS-CoV-2 has been reported to activate the innate immune signalling sensor NLRP3 inflammasome thereby leading to 'Cytokine Storm' in COVID-19 patients and causing Acute Respiratory Distress Syndrome (ARDS) complications like organ failures, and death in severe cases.

As an NLRP3 inflammasome inhibitor, ZYL1 will bridge a critical unmet healthcare need in several inflammatory diseases including the current pandemic of COVID-19 and address complications caused by chronic, uncontrolled inflammation.

Speaking on the development, Pankaj R Patel, Chairman, Cadila Healthcare, said, "We are at the forefront of targeting the innate immune system through novel NLRP3 inflammasome inhibitors candidates with deep understanding of the inflammasome biology. We are committed to developing these pioneering novel treatments to the clinic for the patients in need."

ZYL1, has demonstrated promising efficacy in a number of validated pre-clinical models of Inflammatory Bowel Disease (IBD), Multiple Sclerosis (MS), Sepsis and acute lung injury models of Acute Respiratory Distress Syndrome (ARDS). The studies have demonstrated that ZYL1 can selectively suppress inflammation caused by the NLRP3 inflammasome.