mTORC1 hyperactivation in lymphangioleiomyomatosis leads to *ACE2* upregulation in type II pneumocytes: implications for COVID-19

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Increased pneumocyte expression of the SARS-CoV-2 entry receptor ACE2 in lymphangioleiomyomatosis (LAM) is associated with upregulation of interferon pathways in natural killer cells as well as increased IL6 expression in LAM-associated fibroblasts https://bit.ly/34ChSsg

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To the Editor:

Respiratory failure is among the gravest outcomes of coronavirus disease 2019 (COVID-19), and most of the severe cases are associated with significant lung comorbidities [1]. Lymphangioleiomyomatosis (LAM) is a progressive cystic lung disease of women caused by mutations in tuberous sclerosis genes, resulting in aberrant hyperactivation of the mTOR complex 1 (mTORC1) signalling network in LAM cells, which are of mesenchymal origin [2]. The risk of COVID-19 in LAM is unknown.