Lower respiratory tracts. Acte viral interstitial pneumonia and humoral and cellular immune responses were observed 48,75. Moreover, prolonged virus shedding peaked early in the courseof infection in asymptomatic macaques 69, and old monkeys showed severer interstitial pneumonia than young monkeys76, which is similar to what is seen in patients with COVID-19. In human ACE2-transgenic mice infected with SARS-CoV-2, typeical interstitial pneumonia was present, and viral antigens were observed mainly in the bronchial epithelial cells, macrophages and alveolar epithelia. Some human ACE2-transgenic mice even died after infection 70,71. In wide –type mice, a SARS-CoV-2 mouse-adapted strain with the N501Y alteration in the RBD of the S protein was generated at passage 6. Interstitial pneumonia and inflammatory responses were found in both yound and aged mice after infection with the mouse-adapted strain 74. Golden hamsters also showed typical symptoms after being infected with SARS-CoV-2 [REF.77]. In other animal models, including cats and ferrets, SARS-CoV-2 could efficiently replicate in the upper respiratory tract but did not induce severe clinical symptoms 43,78. As transmission by direct contact and air was obwerved in infected ferrets and hamnsters, these animals could be used ot model different transmission modes of COVID-19[REFS77-79]. Animal models offer important information for understanding the pathogenesis of SARS-CoV-2 infection and the transmission dynamics of SARS-CoV-2, and are important to evaluate the efficacy of antiviral therapeutics and vaccines.  
Clinical and epidemiological features  
It appears that all ages of the population are susceptible to SARS-CoV-2 infection, and the median age of infection is around 50 years 9,13,60,80,81. However, clinical manifestations differ with age. In general, older men (>60 years old) with co-morbidities are more likely to develop severe respiratory disease that requires hospitalization