lower respiratory tracts. Acute viral interstitial pneumonia and humoral and cellular immune responses were observed[48,75]. Moreover, prolonged virus shedding peaked early in the course of infection in asymptomatic macaques[69], and old monkeys showed severe interstitial pneumonia than young monkeys[76], which is similar to what is seen in patients with COVID-19. In human ACE2-transgenic mice infected with SARS-CoV-2, typical interstitial pneumonia was present, with viral antigen observed mainly in the bronchial epithelial cells, macrophages and alveolar epithelium. 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 young 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]. Transmission by direct contact and air was observed in infected ferrets and hamsters, these animals could be used to study different transmission modes of COVID-19 (REF[77-79]). 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 [9,13,60,60,81] ,However, clinical manifestations differ with age. In generak, older men (>60 years old) with co-morbidities are more likely to develop server respiratory disease that requires hospitalization