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monia and humoral and cellular immune responses were observed**’’. Moreover, prolonged virus shedding peaked early in the course of infection in asymptomatic macaques”, and old monkeys showed severer intersti- tial pneumonia than young monkeys’*, which is similar to what is seen in patients with COVID-19. In human ACE2-transgenic mice infected with SARS-CoV-2, typ- ical interstitial pneumonia was present, and viral anti- gens were observed mainly in the bronchial epithelial cells, macrophages and alveolar epithelia. Some human ACE2-transgenic mice even died after infection”. 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’’. Golden hamsters also showed typical symptoms after being infected with SARS-CoV-2 (REF.”). In other animal models, including cats and ferrets, SARS-CoV-2 could efficiently replicate in the upper respiratory trac but did not induce severe clinical symptoms**”. As trans- mission by direct contact and air was observed in infected ferrets and hamsters, these animals could be used to model different transmission modes of COVID-19 (REFS’~-”®), 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”'***!, However, clinical manifesta- tions differ with age. In general, older men (>60 years old) with co-morbidities are more likely to develop severe respiratory disease that requires hospitalization