COVID-19 and the Angiotensin-Converting Enzyme 1 D/I Genotype: Protection

However, a closer analysis of the data reveals that this not to be the case. For example, the five major countries in Central Europe, namely, Italy, Spain, France, the United Kingdom and Germany, have over 100 times more deaths (number of deaths/million) than the East Asian countries of China, South Korea, Japan and Taiwan [2,3,5]; however, the average levels of high-risk people in both regions are almost the same, that is, about 30% of their respective populations. Looking at specific countries, the proportion of high-risk people in Japan, whose life expectancy is the oldest in the world, is 33.4%, well above the world average, but the country's death toll is extremely low at 9 out of 1 million [2,3]. In Taiwan, where the high-risk population is even higher than in Japan, the death toll is only 0.3 out of 1 million [2,3]. It is more likely that the difference in fatalities between Central Europe and East Asia is more related to differences in the ACE1 genotype than in living standards. If so, then the ACE 1D allele may play an active role in coronavirus infections.

Since ACE2 is a viral receptor, it has been the focus of most current research. However, our study suggests for the first time that the ACE1 genotype may be strongly involved in the pathogenesis of COVID-19. As described above, the equilibrium of RAAS is maintained by the positive and negative actions of the ACE1-AngII-AT1 and the ACE2-Angiotensin1-7-Mas receptor axis, respectively. Therefore, ACE2 acts as a suppressor in RAAS. However, it is also important to view the role of ACE1 and AngII as accelerators when considering the reason for the imbalance of the system. The ACE 1 D allele is known to be associated with many comorbid conditions, such as hypertension [24], type 2 venous thromboembolism and myocardial infarction [28]. This suggests that people suffering from these comorbidities may have a high proportion of the ACE 1 D allele. This is a hypothesis that should be carefully studied as soon as possible. For this, please also refer interesting reviews written by Morris [29] and Gard [30]. More importantly, a marked difference in serum ACE levels has been observed between subjects in each of the three ACE genotype classes. Rigat et al. reported that serum immunoreactive ACE concentrations

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