COVID-19: Epidemic Disaster or Evolution Pressure?

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Abstract

COVID-19 has undergone rapid spread worldwide, with the number of infected patients and death events being increased at exponential rates, and thus considered an epidemic disaster. However, SARS-CoV-2 has reduced case-fatality rate and enhanced transmission capacity that favor its propagation and make it evolutionarily more advanced than the other coronaviruses. Further, SARS-CoV-2 preserves virulence to certain patients while keeping some patients symptom-free. It is hypothesized here that SARS-CoV-2 functions as a natural selection pressure for human evolution. That is, COVID-19 is an epidemic disaster at the individual level but may be evolutionarily beneficial for the human at the population level, and survived individuals from SARS-CoV-2 infection may be more tolerable to redox pressure and not suffer anymore from diseases such as hypertension if without external intervention.

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Keywords: COVID-19, SARS-CoV-2, evolution, symbiosis, herd immunity

Word count: 964

Coronavirus disease 2019 (COVID-19), a novel pneumonia that is caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has undergone rapid spread worldwide, taken away the life of many civilians, and thus considered an epidemic disaster.[1]

Recent findings indicate that the outbreak of COVID-19 in different countries may not share the same pathogen origin,[2] some of which were suspected to have already appeared in other places in the world prior to the outbreak of COVID-19 in China. Therefore, COVID-19 may be a global health problem 'simultaneously' appeared at different locations on the earth in this era. While some patients are symptom-free, elder individuals with comorbidities especially hypertension have, in general, a higher case-fatality rate that may be a result of either weaker immune protection or higher redox level in these patients.[3, 4] In addition, evidence was reported on the involvement of human deaminases APOBECs/ADARs in SARS-CoV-2 transcriptome editing,[5] suggesting the attempt of SARS-CoV-2 in human genome integration and host natural response; and SARS-CoV-2 was demonstrated as an outcome of natural selection and evolution.[6] These make us

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wonder, is COVID-19 a pure epidemic disaster? Does it impose any global selection pressure beneficial for humankind evolution?

Co-evolving with viruses has long been part of the human evolution history, with up to 8% human genome sequences being originated from viruses.[7] The fate of viruses is either extinction or symbiosis which is determined by its virulence and transmission rate. High virulence would cause extinction of either its own or the host, and unless viruses could find another species for co-existence, the death of the host is equivalent to the end of its own fate. Low virulence and low transmission may easily lead virus to extinction. Low virulence and high transmission are beneficial for virus propagation, and viruses having these features are evolutionarily more advanced and are more likely to evolve into amensalism or commensalism with the hosts. External interventions may not eradicate these viruses but rather accelerate their evolutionary process. Hepatitis B virus (HBV) is an example of amensalism that may cause cirrhosis and liver cancer despite its long co-existence with the host; and human endogenous retrovirus W (HERV-W) is a good example of commensalism, as after integrating into human genome, the new gene it created, *ERVWE1* , encodes syncytin that plays critical roles in human embryo placentation.

SARS-CoV-2 is less virulent and more transmissive than SARS-CoV and MERS-CoV that outbroke in Asia and Middle East, respectively, in 2003 and 2012,[3] fulfilling the criteria of evolving into symbiosis with human. On one hand, reduced case-fatality rate and enhanced transmission capacity of SARS-CoV-2 favor its propagation and make it evolutionarily more advanced than the other coronaviruses. On the other hand, SARS-CoV-2 is lethal to certain patients but keeps some symptom-free that makes it function as a natural selection pressure for human evolution, the same as what has enabled human evolution from, e.g.,*Homo habilis* to *Homo sapiens denisova* . Importantly, COVID-19 is under vigorous medication control that may considerably accelerate this process.

Whether SARS-CoV-2 will evolve into amensalism or commensalism depends on whether human could co-evolve traits capable of tolerating its virulence and whether the genome of SARS-CoV-2 would convey beneficial effects to human once integrated into human genome. The existence of symptom-free COVID-19 patients suggests the natural tolerance of SARS-CoV-2 in some people. In addition, SARS-CoV-2 binds ACE2 that is protective of the cardiovascular system and capable of maintaining redox homeostasis. On redox stress that is typically imposed by viral infection, ACE2 may be translocated into the nucleus to regulate the expression of genes responsible for quenching elevated reactive species, and thus convey protective effects to human vascular system. Thereby, by integrating into human genome, SARS-CoV-2 may advance humankind towards enhanced tolerance to redox stress and less likelihood of developing hypertension that could be conceived as a disease of imbalanced redox metabolism.[8] As a trend, it is reasonable to perceive the establishment of commensalism between SARS-CoV-2 and human. Consistent with this, a recent publication showed evidence on the evolvement of SARS-CoV-2 towards reduced virulence.[9] Thereby, COVID-19 may not be a disaster but rather represent a selection pressure towards human evolution against diseases caused by redox imbalance such as cardiovascular disorders.

Hypertension is the global leading cause of modality and represents the most important factor predisposing the risk of developing cardiovascular diseases.[10] Naturally low level of reactive species, high tolerance to redox imbalance, or evolvement of mechanism that could quench excess redox pressure are advantageous in surviving from COVID-19. Individuals having such abilities primed in their genome or gained such abilities via co-evolving with viruses will survive from SARS-CoV-2 infection, and pass on their genetic information to the next generations. These selected or evolved traits would make survivors less likely to develop hypertension and cardiovascular diseases and thus enjoy prolonged longevity. Though the outcome of this epidemic may be favorable at the population level, the process suffers as the battle between viruses and hosts during co-evolution is indispensable and always at the sacrifice of the lives of many human beings at the individual level.

The British government announced the concept of 'herd immunity'[11] which though raised debates, to some extent, is in agreement with the concept of 'population evolution' here. Considering humanity and ethics, we are well suggested to rescue the lives of all COVID-10 patients including those severely infected. However,

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while we pray for those who died from COVID-19, we should be optimistic on the future as SARS-CoV-2 helped enriching mankind with survivors primed with high tolerance to redox pressure to some extent that may evolve additional traits favorable for human health, and thus should encourage lightly infected patients co-evolve with virus towards this potential genetic advance. Investigations on the genetic traits and health condition of COVID-19 survivors and symptom-free carriers are encouraged to advance our understandings on this epidemic.

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Competing interests

The author declares no competing interests.

Authors' Contribution

Xiaofeng Dai : Conceptualization; Writing; Funding Acquisition

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