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Persistence of SARS-CoV-2: a new paradigm of COVID-19 management

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Key words: SARS-CoV-2, cytokine storm, re-infection, reservoir, survivors
Parole chiave: SARS-CoV-2, tempesta di citochine, reinfezione, serbatoio, superstiti

Abstract

Full attention must be given to the follow-up of patients recovered from Coronavirus disease 2019, which developed in Wuhan, China in December 2019. Among the most serious issues since the emergence of the Severe Acute Respiratory Syndrome Coronavirus 2 has been whether those who had it can experience a second episode of infection and what that implies for immunity. The earlier studies on COVID-19 disease focused primarily on the epidemiological, clinical, and radiological characteristics of patients with COVID-19. However, conclusions of these studies still require to be warranted by more careful design, larger sample size and statistically well structured studies. COVID-19 is an under-studied infection, and several aspects of viral transmission and clinical progress remain at present unclear. There is a concern about the persistence of SARS-CoV-2 on various surfaces and in the respiratory system of patients who have survived. One of the most concerning issues since the emergence of the SARS-CoV-2 is persistence in patients and whether patients can be re-infected. After hospital discharge, recovered patients were reported to have positive SARS-CoV-2 test in China, Japan, and South Korea. In addition to the persistence of the virus, SARS-CoV-2 re-infection may occur in survivors. In this paper, we focused on the evidence of persistence and re-infection of SARS-CoV-2.

Introduction

COVID-19 is a still under-studied contagious disease caused by a newly discovered coronavirus "SARS-CoV-2". This new disease was unknown before the outbreak officially started in Wuhan, China, in December 2019 with uncertainty about many aspects of transmission and disease (1). Signs and symptoms of COVID-19 have ranged from asymptomatic/mild symptoms to severe illness and death (2). The odds of more severe symptoms are higher with older people or with a health condition like diabetes or heart disease (3). Close contact with patients and respiratory droplets seems

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to be a prevalent mode of transmission, but there is increasing evidence that other modes, such as aerosol transmission, are possible (4).

Regarding the persistence of novel 2019 Coronavirus (2019-nCoV) in COVID-19 recovered patients, some of them have shown to be positive for 2019-nCoV, based on the throat and sputum swabs, for weeks. All of these patients worked as health professionals. Fever, cough, or both occurred at the onset of infection. Only one of these patients was asymptomatic, all patients tested positive for real time-PCR (RT-PCR) (5). Recurrence of infection refers to symptoms reappearing in recovered patients due to the persistence of the viruses in target sites, while re-infection refers to recovered patients, becoming negative when tested and that are susceptible to new infections (6, 7). Both of these phenomena represent a challenge to efforts to control the disease (8, 9). Indeed, there is something we don't understand yet about 2019-nCoV, so the consequences of recurrence or re-infection need to be timely reconsidered for disease control.

Host immune response proposed during infection with COVID-19

Based on the published literature and clinical interpretation of COVID-19 patients, the proposed sensible hypotheses about the pathogenesis of 2019-nCoV infection in humans is that the virus might pass throughout the mucous membrane, particularly larynx and nasal mucosa, and thereafter enter the lungs through the respiratory tract. The early most frequent manifestations of infection are cough and fever (10). The virus may enter the bloodstream from the lungs, and then cause viremia. After that, the virus would assault the targeting organs, for example heart, lung, kidney, and gastrointestinal tract (11-13). The Spike proteins (S) of SARS-CoV-2 bind to the targets cells via ACE2, fusing to the membrane, then release the viral RNA (14). The viral RNAs are detected by the pattern recognition receptors (PRRs). Often, Toll-like receptor (TLR3, TLR7, TLR8 and TLR9) sense viral RNA/DNA in the endosome (14, 15). The viral RNA receptor retinoic-acid inducible gene I (RIG-I) (14, 16), cytosolic receptor melanoma differentiation-associated gene 5 (MDA5), and nucleotidyltransferase cyclic GMP-AMP synthase (cGAS) are liable for the detection of viral RNA and DNA in the cytoplasm. These complex signaling have been recruit adaptors, including TIR-domain-containing adaptor protein including IFN-β (TRIF), mitochondrial antiviral-signaling protein (MAVS) (14, 16-18), and stimulator of interferon genes protein (STING) (19, 20) to trigger downstream cascades molecules, involving adaptor molecule MyD88, and produce the activation of the transcription factor nuclear factor-κB (NF-κB) and interferon regulatory factor 3 (IRF3) and the release of type I Interferons (IFN-α/β) and a series of pro-inflammatory cytokines (20, 21). So, virus-cell interactions release various sets of immune mediators against the invading virus (22, 23). The innate immunity is required in an accurate regulation to eradicate the virus, on the other hand, will result in immunopathology. A few plasma cytokines and chemokines were observed to ascend in COVID-19 patients, including IL-1, IL-2, IL-4, IL-7, IL-10, IL-12, IL-13, IL-17, GCSF, macrophage colony-stimulating factor (MCSF), IP-10, MCP-1, MIP-1α, hepatocyte growth factor (HGF), IFN-γ and TNF-α (24). Notable, an anatomy report of COVID-19 pneumonia corpse (25) indicated that COVID-19 caused an inflammatory response in the lower airway and led to lung injury. Supportively, the virus particles first attack the respiratory mucosa, then infect other cells, triggering a sequence of immune responses and the release of cytokine storm in the body, which

may be related to the serious condition of COVID-19 patients (26, 27).

The virulence mechanisms of Coronaviruses (CoVs), including SARS-CoV-2, are linked to the functions of non-structural proteins (NSP) in the viral genome. For instance, research underlined that NSP can block the host innate immune response (28). Among functions of structural proteins, the envelope has a crucial role in virus pathogenicity as it promotes viral assembly and release.

Among the structural elements of CoVs, there are the spike glycoproteins, which are composed of two subunits S1 and S2. Homotrimers of S proteins compose the spikes on the viral surface, guiding binding to the host receptors. Of note, in SARS-CoV-2, the S2 subunit contains a fusion peptide, a transmembrane domain, and a cytoplasmic domain, which are highly conserved. Thus, it could be a target for antiviral (anti-S2) development. Contrary, and the spike receptor-binding domain present only a 40% amino acid identity with other SARS-CoVs. Other structural elements, on which research must necessarily focus, are the ORF3b, that has no homology with that of SARS-CoV and a secreted protein (encoded by ORF8), which is structurally different from those of SARS-CoV (14, 29, 30).

Persistence of SARS-CoV-2

Human coronaviruses can stay communicable nearly two hours to nine days at a temperature of around 4°C (39.2o F), veterinary versions of the coronavirus could stay live for up to 28 days (7, 31-33). Coronaviruses tend to persist for a shorter time at temperatures of 30–40°C (86–104°F) (7, 31-33). Such as, the HCoV-229E virus is liable for the common cold at room temperature and may persist longer in 50% humidity than in 30% humidity (34, 35). When the scientists delved into the literature on the persistence of coronaviruses on diverse surfaces, the results appear variable. For example, the MERS virus persisted for two days at 20°C on a steel surface (26, 27, 36). However, on a similar surface and at the same temperature, transmissible gastroenteritis virus (TGEV) survived for up to 28 days (26, 27, 36). Furthermore, two studies investigated the survival of two strains of SARS Coronavirus on a paper surface: one survived five days, the other for just 3 hours (26, 27, 36-38).

Novel 2019 coronavirus can survive at least 14 days remaining at humidity and temperature conditions found in an air-conditioned environment. The virus is stable for 21 days in a moist environment at room temperature, but it can be easily killed by heat at 56°C for 15 min (Figure 1) (39). That indicates that SARS CoV-2 is a stable virus that is potentially transmitted by indirect contact or fomites. These outcomes may indicate that contaminated surfaces may play a central role in the transmission of infection in the hospital and the community.

After 28 days of inoculation, SARS-CoV-2 was still detectable for all non-porous surfaces tested (glass, polymer notes, stainless steel, vinyl and paper notes) (40). On plastic surfaces, SARS-CoV-2 remains viable for at least 21 days (33, 39-41). On cardboard, no viable SARS-CoV-2 was measured after 24 hours. Still, the researchers indicated that there is a lot of variability in previously mentioned results, so caution is advisable in interpreting those numbers. The shortest survival time was on copper, where half the virus became inactivated within 4 hours (33, 39-42). It's not sure how long the virus that causes COVID-19 survives on various surfaces, but it seems to behave like other coronaviruses; the alredy quoted WHO studies propose that coronaviruses, including preliminary information on the SARS-Cov-2, may persist on surfaces "for a few hours up to several days" (33, 39-42).

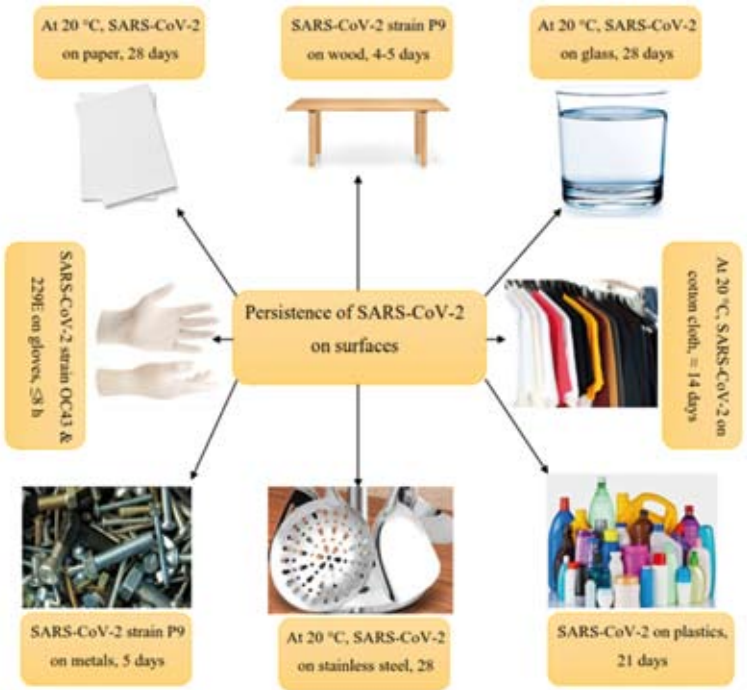

Figure 1 - Persistence of SARS-CoV-2 on surfaces (33, 39)

Re-infection with SARS-CoV-2

Several questions raised about the possibility of re-infection with SARS-CoV-2, although the confirmed case of re-infection remains unclear, are lingering. In this case, it was thought that lowered immunity in a recovered patient might have contributed to re-infection following exposure to SARS-CoV-2 (43).

The WHO has announced it is investigating reports of patients with SARS-CoV-2 who tested positive as a second time. Catching an infection with a SARS-CoV-2 typically means that a person is immune, at least for some time, from repeating the infection (44). According to Osaka's prefectural government, serious concerns arose about COVID-19 on February 27, 2020, when a woman, in her 40s, who had been discharged from Osaka Hospital, Japan, was tested positive for the second time. In another report, a virus was detected through real-time reverse transcriptase-polymerase chain reaction (RT-PCR) tests in four clinically recovered patients at Zhongnan Hospital of Wuhan University, Wuhan, China, and had positive PCR results after hospital discharge. Disease severity was mild to moderate (5). According to the Korea Centers for Disease Control and Prevention (KCDC), 91 people who had previously been cleared of SARS-CoV-2 have been tested positive (45). The KCDC reported an increase in the number of cases from 51, reported earlier.

However, confirming these cases as re-infection is not yet possible. Different alternative explanations (like defective, over-sensitive, or over-diligent testing) could be discussed, including that the virus has been dormant for a time and then re-emerged.

Conclusions

This current pandemic of COVID-19 has challenged the world's economic, medical, and public health infrastructure. Time alone will tell us how SARS-CoV-2 impacts our lives. The reason this novel 2019 coronavirus spreads so quickly and has such a significant effect on humans is that our immune systems have never seen it before.

A potential outbreak of pathogens and viruses of zoonotic origin are continuing, and attempts should be taken to develop effective social and medical steps to avoid zoonotic outbreaks and again COVID-19 as a second wave. SARS-CoV-2 can remain viable and contagious on the contaminated surfaces for significant periods of time. This has prompted reports about the possibility of people being "re-infected" with COVID-19 even though they could have fought off the virus for the first time around. These results confirm the need for focused prevention efforts among survivors and continued ability to quickly diagnose and respond to new cases of coronavirus disease to prevent a widespread epidemic from recurring.

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Riassunto

Persistenza del SARS-CoV-2: un nuovo paradigma della gestione del COVID-19

Occorre porre molta attenzione al follow-up dei pazienti guariti dalla malattia da Coronavirus 2019, che si è sviluppata in Cina, a Wuhan, nel Dicembre 2019. Tra i maggiori problemi causati dall'irrompere del SARS Coronavirus-2 è l'eventuale possibilità che chi ne è guarito possa andare incontro ad una nuova infezione e cosa questo evento implichi per l'immunità. I primi studi sulla malattia furono ovviamente dedicati agli aspetti epidemiologici della malattia ed a quelli clinici e radiologici dei pazienti. Le conclusioni di questi studi attendono però ancora una riconferma da nuovi studi di più accurate disegno, con dimensioni campionarie più generose e supportati da un'analisi statistica meglio strutturata. Quella SARS-Cov-2 è una patologia ancora poco capita, e diversi aspetti della trasmissione del virus e dell'andamento clinico non sono ad oggi sufficientemente chiariti. Esistono ancora problemi circa la sopravvivenza del virus sui diversi tipi di superfici e nell'albero respiratorio dei sopravvissuti. Uno dei principali è la durata della presenza nei pazienti e se i pazienti possono re-infettarsi. Dopo la dimissione ospedaliera, molti pazienti "guariti" hanno dimostrato di essere ancora positivi al virus in Cina, Giappone e Corea del Sud. Ed oltre a spiegare la persistenza del virus nell'organismo umano, occorre chiarire come possa avvenire nei guariti una reinfezione. Questo lavoro è dedicato a chiarire queste circostanze.

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