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# Coronavirus survival on beach sand: Sun vs COVID-19

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## ARTICLE INFO

ABSTRACT

The disinfecting properties of sun (heat and UV radiation) are adequate in warm sunny regions to rid beach sand of coronavirus particles, if present. Here we detail the mechanism of natural disinfection offered by the sun on coronaviral particles that may find their way onto beach sand. We conclude that heat and UV radiation generated by the sun destroy the virus infection ability.

### **1.** Introduction

Contracting severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus that causes COVID-19, is done mainly by person-to-person interaction but a possible vehicle remains the environment, i.e. the use of fomites (doorknobs, tables, phones etc) that may carry viral particles. Transfer of the virus from the environment remains a concern, as population movement restrictions have slowed down the spread, but may not be as effective as hand washing, self-isolation and early detection ([Chinazzi et al., 2020](#page-2-0); [Kraemer](#page-2-0)  [et al., 2020](#page-2-0)). Viruses do not replicate outside living cells, but they can survive, be transported and upon entering a susceptible host, cause disease. In the case of SARS-CoV-19 the pathogenic viruses pass into the environment from ill humans or carriers mostly via the respiratory track. The role of the environment in disease transmission is indisputable, the survival potential for viruses include environmental factors such as temperature, ultraviolet radiation, salinity, pH as well as type of surface of potential fomites.

Beach sand is a transmission medium attracting much focus, because contracting COVID-19 through contact with polluted sand has been a matter of concern to regulators and the general public. Beaches worldwide are visited for recreation purposes, and contact with the beach sand is integral part of the recreation potential. Growing evidence suggests that beach sand can be a vehicle for transmission of human pathogens ([Sabino et al., 2014](#page-2-0); [Solo-](#page-2-0)[Gabriele et al., 2016](#page-2-0)). To understand the possibility of SARS-CoV-2 on sand posing risk to public health, it is necessary to examine the possibility of coronavirus particles surviving on this medium, which could allow them to transfer into the respiratory track of susceptible humans. In this short communication we shall discuss the behavior of the virus on the sand of bathing beaches and shall argue that SARS-CoV-19 virus contacting the surface of beach sand particles will suffer lethal thermal and radiation injury.

### **2.** Discussion

Coronaviruses, RNA enveloped viruses, have an outer coat of lipid membrane that has been demonstrated to be not just building blocks but play crucial part in the function of the virus  $(Ono, 2010)$  $(Ono, 2010)$  $(Ono, 2010)$ . The coronaviral genome encodes additionally four major structural proteins, embedded into the lipid membrane: the spike protein, nucleocapsid protein, membrane protein, and the envelope protein, which are involved in producing structurally complete viral particles. A subgroup also contains an additional membrane protein, the HE protein ([Schoeman and Fielding, 2019](#page-2-0); [de Haan and Rottier, 2005](#page-2-0)). The lipid composition of the envelope contains sphingomyelin, phosphatidylsrine, phosphatidylinositol and phosphatidylethanolamine ([van Genderen et al.,](#page-2-0)  [1995](#page-2-0)). Viruses do not multiply outside living cells. Little is known about SARS-CoV-2 in the environment ([Nunez-Delgado, 2020](#page-2-0)). It is currently thought that coronaviruses can survive for up to a few days outside living cells. Containment of the virus appears to this day particularly challenging (Li et al., [2020](#page-2-0)).

Concerning the presence of this virus on the sand of bathing beaches, it is expected that the viral particles, transferred via respiratory droplets, will land on the surface of the sand grains and will be exposed to environmental hazards. This is not the case with enteric viruses, who have their origin in misplaced sewage or animal waste, hence can be met in the deeper layers of sand, well protected from environmental fiuctuations ([Sabino et al., 2014](#page-2-0); Sinclair [et al., 2009](#page-2-0)).

The methods currently available to destroy coronaviral particles in the environment include inactivation with the use of biocidal agents, which has been extensively discussed ([Kampf et al., 2020](#page-2-0); [Wolf et al., 2005](#page-3-0)). Tons of disinfectants have been and continue to be spread on surfaces, in an attempt to minimize opportunities for the spread of the virus. Chlorination as a method of disinfection has been heavily criticized ([Zhang et al., 2020](#page-3-0)). Spreading

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Focus

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chlorine disinfectants on beaches destroys the normal microbiota and runoff will contaminate the water body at hand (river, lake, the sea) putting aquatic ecosystems in danger.

Heat is a natural non-invasive way of destroying microorganisms and viral particles. Coronavirus decays more rapidly when temperature increases (Biryukov et al., 2020; Riddell et al., 2020). SARS-CoV-2 cell membranes are often found to undergo gross structural changes associated with characteristic disturbances of function, if the environmental conditions are altered beyond the normal limits within which the virus can perform its metabolic function ([Wang](#page-3-0)  [et al., 2004](#page-3-0)). High temperature poses fundamental constraints on metabolic processes of cells, through the reduction of membrane fiuidity, decrease of viscosity of biological fiuids and, when temperatures rise, through the denaturation of proteins. Temperatures above 41 ◦C (105.8 ◦F) break the weak hydrogen bonds that dictate the secondary and tetriary structure of cell wall proteins, and denature them. In vitro analysis demonstrated that after 5 min exposure to 56 ◦C (Kariwa et al., 2006) or 70 ◦C (Chin et al., 2020) SARS-CoV in aliquots, the virus infectivity was inactivated. Lipid molecules are similarly affected. The lipidic nature of membranes makes enveloped viruses more susceptible to environmental stress. Viral lipid envelops are fragile and sensitive to heat. There is strong evidence that the composition of the lipid component of the envelope changes in response to environmental conditions such as temperature. If the environmental conditions change beyond the normal limits within which the organism survives, the whole structure undergoes major structural changes. The protein/lipid capsule of coronaviruses is readily destroyed if exposed to temperatures above 50 ◦C.

Sand can be over 38  $°C$  (100 °F) when the outside temperature is only 24  $°C$  (75 °F); indeed, when the ambient temperature is 32 °C (90 °F), sand temperature can be over 49  $°C$  (120 °F). As the temperature increases, the duration of exposure to the heat source required to result in thermal injury decreases (Cohen, 2019). A temperature of approximately 50 ◦C of sand particles of the surface layer on a sandy beach is sufficient to cause second degree burn to the delicate skin of a human foot, if in contact for several minutes. Any particle of SARS-CoV-2, if transferred onto the sand by a coughing ill human, would be destroyed.

Exposure of microorganisms to Ultra Violet (UV) radiation is considered the principal natural virocide in the environment and has been exploited in solar disinfection applications. Absorption of UV light by the protein molecule leads to dissociation of hydrogen bonds, and thus denaturation. UV additionally induces damage to the genomes of viruses and microorganisms in general by modifying their genetic material, breaking bonds (Lytle and Sagripanti, 2005), denaturating protein, because of oxidative damage to amino acids, producing lipid oxidation and altering lipid composition (Santos et al., 2013). SARS-CoV-2 is highly susceptible to ultraviolet light (Hellingloh et al., 2020; Hessling et al., 2020). In vitro exposure in cell cultures of the virus for 15 min reduced severely the infectivity potential. After 30 min exposure no infectivity could be observed (Duan et al., 2003). The geographic location and the elevation influence are important factors, when it comes to solar light inactivation of viruses. The overall research agreement is that in culture media a 90% inactivation is achieved from 20 min to 1 h (Lytle and Sagripanti, 2005). In simulated saliva SARS-CoV-2 loses 90% of its infectivity every 6.8 min on a steel surface (Ratnesar-Shumate et al., 2020) or every 8 min in aerosols (Schuit et al., 2020), if exposed to simulated sunlight representative of the summer solstice at 40  $^0{\rm N}$  latitude at sea level. In the experiments discussed the virus particles were exposed to either heat or UV protected by the culture medium, into which they were tested. The culture medium is declared by the authors to be 'poorly penetrative' to UV, plus it absorbs itself part of the available heat, thus decreasing the virocidal ability of elevated temperatures. Heat exposure of SARS-CoV-2 inside minute droplets 0.1–30 μm diameter brings about the desired outcome faster. From a virus safety perspective, the combination of complementary methods (elevated temperatures and UV exposure) increases the total quantity of virus that is eliminated.

We appeal to local authority regulators to refrain from disinfecting beach sand by spreading it with chlorine based disinfectants. Instead we propose that - in environments where the air temperature reaches or exceeds 30 °C during the warmer hours of the day - they let nature (heat and UV radiation) perform

its healing effects.

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### **CRediT authorship contribution statement**

**Maria Adamantia Efstratiou:** Conceptualization, Methodology, Writing – original draft, Writing – review & editing. **Ourania Tzoraki:**  Writing – original draft, Writing – review  $\&$  editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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