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On the Possible Beneficial Role for the Regular Use of Potent Mouthwash Solutions as a Preventive Measure for Covid19 Transmission; Invoking the Evolutionary Biology and Game Theory

Mukhtar K*

Department of Surgery, Hazm Mebaireek General Hospital, Qatar

***Corresponding author:** Khalid Mukhtar, Department of Surgery, Hazm Mebaireek General Hospital, Hamad Medical Corporation, Doha, Qatar, Tel: +974 77446394; Email: kmukhter@hamad.qa

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Abstract

In vitro studies demonstrated bactericidal and virucidal role for some of the over-the-counter mouthwash solutions. Meanwhile, Game theory and evolutionary biology suggests that inhibiting cooperation -reciprocal altruism- between two organisms can negatively affect their survival, based on a set of relevant publications, it is proposed here that "SARS-COV-2" may be relying on an "accomplice"; be it a certain organism (e.g. bacterial species), or a state of dysbiosis in general. On this premise, the regular use of potent disinfectant, through the repeated reduction in microbial load, may be able to induce a strain sufficient to inhibit reciprocal altruism, and hence halt the progression of the disease, as observed in the majority of those tested positive worldwide, yet with mild or no symptoms. We cite a group of observations related to COVID, that can be justified by the complicit hypothesis, predict a rather preventive than therapeutic advantage, suggestive for a possible role for the regular use of potent mouthwash as an additional control measure in the community level.

Introduction

Recently, there have been calls for studies to test the potential beneficial effect of potent disinfectants to be applied for mouthwash, and sometimes the nasal cavity, as an approach to COVID19 treatment. Caruso, et al. [1], argue that “nose/mouth/throat washing using Hydrogen peroxide (H2O2) can enhance local innate responses to viral infections and help protect against the current coronavirus pandemic. In fact, by the 4th week of June 2020, there were four registered similar trials at the NIH only, and likely more elsewhere, aided by the reported viricidal/bactericidal activity for H2O2, as well as other disinfectants.

However, there are many factors that could render such efforts futile; to begin with, the use of disinfectants will likely to be useless for intracellular microbes, and as the disease progresses, the virus seems to reside in anatomical sites other than the oral and nasal cavities, thus inaccessible to the direct effect of the disinfecting solution, and moreover, the continuous saliva flow, can replace the virus shortly after the mouthwash use, and that toothbrushes can be contaminated on their own accord [2], hence act as a secondary reservoir well isolated of the treatment regimen effect, and finally, there’s the arbitrary selection of solutions, concentration, frequency and duration of use. Eventually, one might argue that such solutions might help temporarily to reduce the viral load, as prior to dental procedure, yet not to cure the disease.

However, we think there’s still a merit for advocating such approach as an addition to current preventive measures, to aid in controlling the pandemic in a community, rather than curing the disease, by invoking “reciprocal altruism”,

a known principle in evolutionary biology, and argue that regular administration of a potent bactericidal / virucidal solution, only to the oral cavity, as it is repeatedly reduces the microbial and viral load, may aid in inhibiting both, the propagation of the disease in an infected person, specially earlier in the course, as well as reducing the transmission rate to others.

Reciprocal altruism implies that an organism may opt to accept "less gain" in order to benefit another organism, on the he expectation that the other organism will do the same in the future. This summative or "cross-sectional" view of the interaction, resembles "tit for tat" cooperation behavior of the "Game theory" [3] which is the iterative form of such interactions; effectively demonstrated by the mathematical psychologist Anatol Rapoport, in winning a famous computer tournament in the 1980s, organized by Robert Axelrod, titled "on the evolution of cooperation"; a computer code described as "tit for tat" implemented the iterative form of reciprocal altruism; as the two interacting agents -or organisms- will eventually have the maximal gain in relation to time, should both choose to cooperate, however, should one "defects", especially repeatedly, their cooperation ends as the other retaliates, and their interaction is then described as "death spiral".

Now where would that fit in COVID19 pandemic treatment? we hypothesize that the oral microbiota has a role in the development, and/or progression of the SARS-COV-2 infection, the premise is that the virus needs an accomplice, whether a certain organisms or, more likely, the state of dysbiosis, that at certain "thresholds" allows for encountering the infection in an exposed persons, and other ones to progress to different stages of the disease spectrum; either "asymptomatic", "mild symptoms", "moderate" and "severe pneumonia", then ARDS or SIRS and death at the dreaded end of the spectrum. Needless to mention, as the disease progresses, it is no longer dependent on the virus alone, as it then manifests as a composite clinical picture of the viral, secondary or super infections, mostly bacterial, host immune response, worsening comorbidities, and the capacity of healing and regeneration. Here the treatment approach must take all those factors into account, as even the best antiviral therapy, alone, will fail to reverse the disease.

The development of the infection among the repeatedly exposed individuals, say the medical staff, suggests that, among others, the cumulative exposure effect and encountering viral load from multiple infected cases can overcome the effect of the "protective commensals", also in line with the dynamically changing microbiota, that seems to allow SARS-COV-2 entry., as the status is "shifting into dysbiosis", which can serve as an accomplice, and allows for the presence or absence of a specific organism, that leads to acquiring to the infection, whether in a protective role, "i.e. the absence of certain organisms that could've limited the acquisition of the infection", or the other side of the coin, "the harmful commensal", that facilitates the onset of the COVID19.

As the infection is spread through droplets, oral inhalation is likely to pose more risk than nasal breathing; given the lack of natural filtering capacity, and the protection provided by the high levels of nitrous oxide produced in the nasal passages [4] which proved to inhibit viral replication, including SARS CoV-2 [5].

There're a group of observations that can be justified by the assumption of an accomplice agent residing in the oral cavity:

a) The oral cavity serves as a portal for both, the respiratory tract and digestive system, as both being the commonly affected systems on presentation to the hospitals. As it harbors diverse hundreds of microbial species, some are a continuum to the respiratory tract microbiota, thus it is justified to target the oral cavity with treatment protocol such as mouthwash.
b) The role of ACE2 receptors: previously known and linked to gut dysbiosis, and also identified in the oral cavity [6] ACE2 can hence serve as a mode of interaction for reciprocal altruism, as it both serves the SARS-COV-2 virus for its eventual replication and disease manifestation, as well in propagating a state of dysbiosis for the benefit of the accomplice.
c) The Cytokines storm: frequently reported in cases of COVID19, Cytokine response has been crucial in Periodontitis pathophysiology [7] and might suggest a shared microbial factor.
d) Less infected/exposed ratio: despite of the fast spread SARS-COV-2 in the absence of herd immunity, the reported "incidence" figures, are rather "lower" than expected by SER predictive models published earlier in the pandemic by academic and health institutions. Even when considering that such forecasts might have been positively biased, and that observed figures are function of geographical variation in the frequencies of reported cases or complications, we propose that such models suggest the presence of another agent, a "mode of interaction" between the virus and the URT microbiota; thus such model need to include a factor that can represent the probability of encountering the infection for an exposed individual, based on a parameter that can describe the microbiota of the URT. Being difficult to measure such "parameter", yet for purpose of research, we can theoretically modify it, for instance, by inflicting the modification or reduction of oral microbiota. This assumption requires demonstration of different (less) infection / complication rates among those being treated

for gingivitis, as an example, as regular antibiotics or potent mouthwash are targeting the oral microbiota.

e) Higher transmission among cohabitants: greatly accounted to by the higher exposure rate to the virus, also among others, is the higher probability of sharing similar microbiomes. The existing literature suggests that periodontal pathogens can be shared between family members and cohabitants [8,9] and hence, once the virus has become acquainted with interaction to certain microbial communities, practically had exercised the input and output of interaction with such environment, this would increase its chances in improving its future cooperation interaction techniques with these environments, whether in the same person (reinfection), or others with similar microbial communities in other persons, and hence spread faster. This assumption requires that: the subsequently infected cohabitants or family members, tend to have a more sever disease or worse outcome, in comparison to the primary source of infection -person- in the house; in short: those who are infected later on, tend to do worse.

f) Variation of the disease severity, within and among different geographical regions: Geographical variation is a well-recognized factor in microbial diversity, as evident by studies on dental caries. [10].

g) The milder form of the disease among children: The difference of composition of the oral microbiota in younger age groups is reported in the first two years of life, [11] where certain organisms are more prevalent, while age-related microbiota transformation is being observed well into adulthood. This assumption implies that certain microbial communities, isolated by culture independent techniques, may associate with higher or lower incidence rates of COVID19; either for the disease or complications.

Manipulation of oral microbiota therapeutically to change lung disease progression is not a new idea, that might be affordable through the regular use of a potent mouthwash, and may have a beneficial effect as an additional method in the community to contain the pandemic, along with wearing mask, hand hygiene and social distancing, by altering dysbiosis in the microbiota of the oral cavity, which share similar composition with the lower respiratory tract. The key effect, we hypothesize, is in the repeated use, as the inflicted strain in both sides is likely to inhibit any kind of cooperation between the newly arriving SARS-COV-2 and the native microbiota. Such approach may be utilized in the large community scale; and even more applicable for "closed communities", where other measures might be impractical, such as in hospitals, nursing home and similar long-term facilities, schools, sports training facilities, as well as prisons and army dormitories. Yet in order to get a momentum, such use of mouthwash can be advocated openly, if it demonstrated clinical or lab results; the former will require RCTs for either preventive or therapeutic role, while the later demands, after the repeated use of such potent mouthwash solutions, demonstrating a disproportional reduction in the viral cytopathological activity, to the sequentially decreasing viral load. However, as most of mouthwash solutions are over-the-counter preparations, we do advice using such solutions regularly, even in the absence of clinical trials.

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