# Editorial – COVID-19 and the microbiota: new kids on the block

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Viruses are an insignificant part of the intestinal Microbiota, when evaluated in terms of weight, as we usually do[1]. If you ask experts about the weight of viruses in relation to all other microorganisms (now defined around 1 kg[2]), the answer will be "their weight is practically zero". Despite that, viroma is a very important component of our microbiota, able to share information and to exert biological properties together with all other microbial components, in a very smart and cooperative manner[2].

While many researchers have studied how infectious agents may interact with our immune system, sometimes leading to the occurrence of different conditions, such as metabolic, cardiovascular, autoimmune and neoplastic diseases, with apparently dissimilar etiologies[3], others have pointed out that microbiota may represent a common denominator.

Indeed, some evidence indicates that microbiota can modulate the immune response in the course of both bacterial and viral infections, becoming a potential target in the management of all these diseases[3]. Recently, a particular focus has been made on the role of intestinal viroma as well as any possible interactions among exogenous viruses, viroma and all the other components of the GUT microbiota[4,5].

The recent pandemic induced by COVID-19[6-9] reminded us how difficult is the management of a viral disease, since it is associated with:

- Great capacity of transmission through easy-to-travel contagion routes;
- Extreme ease of spread, making it potentially pandemic. This does not occur, in most of the cases, because of the antigenic characteristics of viruses in relation to environmental conditions. Certain diseases may also be subclinical, due to the low pathogenicity of the virus;
- Way of transmission and activation of immune defenses, which in the GI tract are strongly represented by lymphoid tissue, which is the largest one of the entire body[10]. Lymphoid tissue will determine both immune and inflammatory response within different organs, thus affecting the clinical course of the infectious disease;
- Rapidity of its clinical expression and strong effect on comorbidities;
- Needing of an appropriate symptomatic, antiviral and sometimes immune-suppressive therapy;
- Vaccination, which may accelerate reduction of the spreading of the virus among individuals and possibly attenuating its aggressivity, even though mutations in the viral structure or poor antibody production can be a reason of failure.

The recent infection sustained by COVID-19 came to our attention too late also because of an overlapping with seasonal flu; due to its aggressivity, at least in patients with peculiar characteristics, it caused a hospital and intensive care unit overcrowding with tragic consequences. Pathological damage caused by this virus seems to be mainly inflammatory/thrombotic and is mostly localized in the pulmonary circulation[11-13]; besides that, intestinal localization possibly associated with occurrence of GI symptoms has also been reported in about 5-10% of the patients[14]. What are the interactions among COVID-19, GUT and lung-microbiota? Nobody still knows that, but it is mandatory to open a special focus on this issue to better understand why some of the patients were totally asymptomatic, some of them developed mild symptoms, while others underwent to acute distress respiratory syndrome. May we hypothesize that differences in GUT and lung microbiota composition may discriminate severity of COVID-19-related infection?

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Concerning therapy, besides anti-viral and hydroxychloroquine, in the absence of a vaccine and in patients with extremely serious conditions, there is also a role of corticosteroids, whose anti-inflammatory efficacy is well known[7], alone or in association with the most innovative anti-interleukin 6 drugs, at least in our opinion. In addition, considering the hyper-coagulation status demonstrated in those patients, heparin therapy at an early stage is also strongly indicated, checking the clotting factors, since thrombosis, disseminated intravascular coagulation and pulmonary embolism is a feature of COVID-19 infection[15,16]. Moreover, the use of antibiotics, particularly azithromycin, and possibly of selected probiotics may also be requested in some patients[17,18].

Finally, we would like to highlight some concepts concerning the risk for all health workers who deal with COVID-19 every day, sometimes without appropriate personal protective equipment; just try to think about how many doctors and nurses became a vehicle for transmission of the virus among relatives or other people or they even lost their life. Is it acceptable for a health worker to die from the disease he is treating? Obviously the answer will always be "no", but the reason why we chose our profession is to help people to maintain their health or cure diseases, while the risk to acquire infections is just part of the game. This is why we do not think we can be heroes; we are just doing our duty in a more complicated environment.

## Conflict of Interest

The Authors declare that they have no conflict of interests.

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