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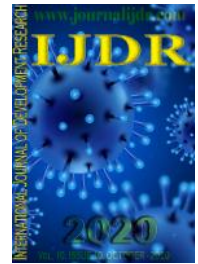
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RESEARCH ARTICLE

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DIFFERENT BACKGROUND OF IMMUNOLOGICAL RESPONSE ACCORDING TO PREVIOUS ZONOTIC EXPERIENCE WITH CORONAVIRUS*

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ABSTRACT

The new coronavirus, severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has caused more than one million deaths worldwide. However, little is known about the causes of death and the virus's pathologic features. In two leading Italian cities, almost 80 patients have been treated so far with severe but not very serious respiratory problems with plasma and none have died with a zero mortality rate. A plasma bank was created in Mantua, an idea that should be replicated throughout Italy and which could stem a possible second wave. The population of Africa is younger than in regions hardest-hit by COVID-19. Some researchers have shown that other human coronaviruses that cause common colds can elicit an immune response that could provide protection against COVID-19.

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INTRODUCTION

T cells were studied for their response to structural (nucleocapsid, NP) and non-structural proteins (accessory NSP-7 and NSP-13 of ORF1) of the viral particles of SARS-CoV-2 in 24 subjects convalescent by COVID-19. CD4 and CD8 T cells were recognized in all convalescents which recognized multiple regions of the nucleocapsid protein (NP). 23 patients recovered from the first SARS, 17 years after the outbreak, still have a long-lasting memory of T cells with respect to the structural proteins of the nucleocapsid (NP), which demonstrates a cross-reaction towards the NP of the current SARS. The subjects without history of the primitive SARS, in 50% of cases (9/18) have T cells that react with the accessory proteins (non-structural NPS-7 and NSP-13 of ORF1). Epitopic characterization of NSP-7 specific T cells showed recognition of protein fragments with respect to animal betacoronaviruses, but very reduced for human common cold coronaviruses. Therefore infection with betacoronavirus induces a strong and long-lasting immunity of T cells with respect to NP structural proteins (Nina Le Bert *et al.*, 2020).

METHODOLOGY

The most comprehensive documentation on plasma therapy in Covid patients can be found in the article of Kai Duan *et al.*, 2020, here just a few words. The plasma transfusion (i.e. the most 'liquid' part of our blood, where antibodies are present formed after the battle won against the virus) has been successfully used in the other two coronavirus epidemics, SARS of 2002-2003 and MERS of 2012-2015, entering the plasma at a precise stage of the disease (Tarro, 2020 a); that is, when there is already poor oxygenation and the patient is subjected to assisted ventilation with a C-pap helmet, but is not yet intubated (Tarro, 2020 b). It is a therapy that, like many, presents risks but, frankly, it is not clear exactly why the World Health Organization - which had confined its use "only in the case of serious diseases for which there is no effective drug treatment" - did not suggest, at least, the experimentation during this Covid19 emergency (Karpas, 2020 a). Despite this, after the positive results attested in the above article, timidly, challenging bureaucratic dispositions, not a few doctors, even in Italy, have started experimenting; in some cases - such as

the team of the San Matteo Hospital in Pavia and the one of Poma Hospital in Mantova - developing health protocols in a week that would have taken months (Karpas, 2020 b).

RESULTS

According to the experience of the first SARS and of the MERS, the children were not exposed to the civet cat and camels in a similar way (Tarro, 2020 b). It was thought that the same fact could take place with the SARS from COVID-19 (Fan et al, 2020). Indeed children are infected with the virus without suffering a serious disease and represent an important source of infection. The virus is found in their rectal swabs (Arantes de Araújo et al, 2020). Growing with age many specific cells of the immune system are no longer active and therefore the body loses its ability to respond effectively. In fact, it has been experimentally proven that young mice respond to lung tissue damage from viral infection through prostaglandins, while adult mice succumb (Tarro, 2020 c). The juvenile immune system and its efficient T Helper cells respond to SARS COVID-2.

The Helper cell's CD4 lymphocytes stimulate B cells to produce antibodies against the virus and control infection. In this case Th2 lymphocytes are able to control the inflammatory response caused by the viral infection, preventing an exuberant and delayed reaction as occurs in adults. The different hormonal structure and the same prostaglandins favor the female subject against the coronavirus responsible for the current pandemic (Tarro, 2020 d).

DISCUSSION

Immune system protection: According to South Korean scientists a number of reported cases of COVID-19 patients relapsing after overcoming the diseases were due to faulty testing; researchers at the South Korean CDC now say that it is impossible for COVID-19 to reactivate in human bodies. The South Korean CDC has found that the PCR test results for the suspected relapsed patients were false positive, warning that the tests were not able to distinguish between live traces of the virus and the harmless dead samples that remain after the patient has recovered. Another complicating factor for immunity passports is that antibody tests can't rule out that a person is no longer infectious. A study published in Nature found that viral RNA declines slowly after antibodies are detected in the blood. The presence of viral RNA could mean that the person is still shedding infectious virus (Mallapaty, 2020).

Conclusion

Finally I would like to explain the puzzle concerning the lack of coronavirus outbreaks in Africa. One hypothesis that can explain the disparity between Africa and other continents concerns the overall age of the population. In general, the population of Africa is younger than in regions hardest-hit by COVID-19. Some researchers have shown that other human coronaviruses that cause common colds can elicit an immune response that could provide protection against COVID-19. The same crowded neighborhoods that would lead to the quick spread of other coronaviruses may have protected the population from SARS-CoV-2. It's a hypothesis. Some level of pre-existing cross-protective immunity...might explain why the epidemic didn't unfold (the way it did in other parts of

the world). Professor Shabir Madhi said <the protection might be much more intense in highly populated areas, in African settings. It might explain why the majority (on the continent) have asymptomatic or mild infections> (Smith, 2020).

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