Reply☆

Réplica

Dear Editor,

I am grateful for the interest aroused by the recently published leading article in REUMATOLOGÍA CLÍNICA.[1] I would like to comment on the interesting issues expressed in the recently received elegant letter to the editor.[2]

Several months ago the coordinators of the 2019, 8th edition of Reumatopics proposed that I participate in the roundtable entitled "Fibromyalgia in debate". The format of this roundtable was standard: one speaker in favour and one against. It was suggested I should be the one against fibromyalgia. I consulted several colleagues about this: some told me to accept, others said not to "entangle myself" in it. I accepted and the roundtable was a success. You may check it out on: portaldelsocio.ser.es/reumatopics19. Afterwards I received appraisal from the roundtable participants: it had been highly praised and what was more important: it extended links between those for and those against fibromyalgia. These links are missing from your letter.

I usually always write my papers. This was what I proposed to the Editorial Committee of REUMATOLOGÍA CLÍNICA. I myself chose the format of the leading article. It is worth recalling that a leading article is a short article which expresses an opinion or interprets the facts of others. The leading article should be brilliant, well argued or course, imperative or conciliatory. It emotionally and intellectually stimulates its readers. It may be the origin of substantial reactions towards controversy and debate. The leading article expresses a personal opinion on the theme. What's more, I envisaged ideological stances which would transcend mere critical argumentation.[3] In my humble opinion the finished leading article met with expectations and I am satisfied that controversy was aroused. The Editorial Committee of REUMATOLOGÍA CLÍNICA is exempt from all responsibility.

You use the word "recovery". I did not intend to recover anything in the leader. I insist, it is my personal opinion, it is a story of the history of fibromyalgia, exactly as I, and probably many other rheumatologists of my generation, have experienced it. It may be that the concept of psychogenic rheumatism – created by the pioneers of rheumatology – was a mistake, but there can be not the least bit of doubt that without the concept of psychogenic rheumatism, fibromyalgia would not exist today. Numerous examples may be found in the history of medicine.

I regret that you perceive the writer to have scarce knowledge, regarding terminology used. I am not claiming that fibromyalgia is one of my specific areas. I accept your comments regarding nomenclature but not de-authorisation, which I believe to be an inappropriate term.

Finally, I am very pleased that we agree about the financial cost of fibromyalgia. These data are not open to debate. Healthcare policies carried out have not been successful. Last but not least, it is a duty of the specialists dedicated to fibromyalgia and of the scientific societies to recruit young rheumatologists. In other words, to make rheumatology scientifically appealing. The leader reflects this, as does your learned opinion.

My final point: "*All wisdom is not new wisdom*".[4]

References

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Why choose cyclosporin A as first-line therapy in COVID-19 pneumonia☆

¿Por qué elegir ciclosporina A como primera línea terapéutica para la neumonía causada por COVID-19?

Dear Editor:

The use of immunosuppressive therapies in COVID-19 infection is a recently raised topic which comes to fill an unmet need in the management of the patients.[1] Intriguingly, not only COVID-19 but also SARS and MERS CoVs – all members of the Betacoronavirus genus – associate to an increased risk of respiratory distress syndrome. Already in patients with SARS-CoV, the development of respiratory failure was thought to be the consequence of a vigorous innate immune response, while effectiveness of tocilizumab in COVID-19 infected patients also supports the participation of a cytokine storm in severe phenotypes.[1,2] A factor underlying this explosive response could be the capacity of betacoronaviruses to invade immune-competent cells, particularly macrophages, thereby hijacking the major drivers of innate immune responses. Nonetheless, targeting pro-inflammatory cytokines is neither the sole nor the first-line immunomodulatory approach in combating the infection. As represented in the figure, complex virus-host cell interactions providing opportunities for therapeutics should be regarded (Fig. 1).

Betacoronaviruses replicate and carry out transcriptional activities at the cell cytosol, where the viral genome is detected by RIG-1 like receptor (RLR) helicases. Upon binding of vRNA, RLR activate mitochondrial antiviral proteins (MAVS). These in turn trigger phosphorylation of transcription factors and gene

☆ All authors have contributed to the conception of the manuscript, have revised it critically, have approved the final version and agree to be accountable for all aspects of the work.