

Letter to Editor

Jose Artur Medina

Multiple Sclerosis, Chlamydia and Gut Disbiosis

Jose Artur Medina *

Fisiopatologia USP, Institute Butantan, SP, Brazil.

*Corresponding Author: Jose Artur Medina, Fisiopatologia USP, Institute Butantan, SP, Brazil..

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Abstract:

I believe that multiple sclerosis progression could be stopped through antibiotics, dewormers, beta glucans and garlic extract and, by far the most difficult, changing the quality of the water ingested, or used for washing salads, in addition to increasing the consumption of probiotics. I believe that MS sclerosis is due to the unfortunate encounter of 3 diseases: intestinal dysbiosis, chlamydia infections and orthopedic problems. However, how would these three diseases cause demyelination of nerve fibers?

Keywords: sclerosis; chlamydia; gut disbiosis; probiotics; enterobacterias; herpesvirus; chemotaxis; neurodegeneration; piriformis syndrome; bacterial vaginosis; ureoplasmas

Summary

Dear Editor, I believe that multiple sclerosis progression could be stopped through antibiotics, dewormers, beta glucans and garlic extract and, by far the most difficult, changing the quality of the water ingested, or used for washing salads, in addition to increasing the consumption of probiotics. I believe that MS sclerosis is due to the unfortunate encounter of 3 diseases: intestinal dysbiosis, chlamydia infections and orthopedic problems. However, how would these three diseases cause demyelination of nerve fibers?

1) By the consumption of contaminated water (enterobacterias), which would raise the levels of CD4-Th17 [1-3] that by itself reduce the blood-brain barrier [4-6].

2) A subclinical infection by chlamydia or any other pathogen of the genitourinary tract, via LPS, would stimulate TLR4 and the release of inflammatory mediators, at the expense of the stimulation of DAMPs (beta glucan / hyaluronic acid) that would elevate the levels of antiviral interferon instead of producing more inflammation [7-9].

3) Levels of the endemic herpesvirus would increase, which favors the exteriorization of type 2 MHC complexes as disruptions in signaling created by the own virus [10-12] and, subsequently, systemic Th17 chemotaxis and subsequent IL-17A release and secondary demyelinating neurodegeneration [13-15].

4) Orthopedic problems such as piriformis syndrome [16, 17] or herniated disc [18, 19] favor neurodegeneration and deserve treatment.

5) Waterborne diseases are epidemiologically important [20, 21], producing reactive arthritis (Reiter's Sd) [22, 23] and making rheumatoid tests positive [24, 25].

6) The relationship between lupus and MS could be explained by bacterial vaginosis, which makes the antinuclear factor test positive, when there is a significant presence of ureoplasms and mycoplasmas among the anaerobes that make up bacterial vaginosis, also called Gardnerella [26, 27], lupus would thus be caused by a pelvic inflammatory disease (PID), fibromyalgia would be secondary to the same infection, but without the presence of ureoplasmas and mycoplasmas. Clinical pain would be proportional to the LPS that would migrate through the lymphatic system reaching shoulders and knees as well as neurodegeneration.

7) Most PIDs are asymptomatic. [28-30]

8) In summary, the progression of MS would be blocked (the infiltration of Th17 and Th1) through the eradication of chlamydia, intestinal dysbiosis and, eventually, by decompression of the affected nerves, in addition to offering garlic extracts that increase interferon [31-33] has the potential to reduce the endemic herpes population in humans.

References:

- 1. Shao, Lihong, et al. (2020). "Bacterial dysbiosis incites Th17 cell revolt in irradiated gut." Biomedicine & Pharmacotherapy 131:110674.
- Bekiaris, Vasileios, Emma K. Persson, and William W. Agace. (2014). "Intestinal dendritic cells in the regulation of mucosal immunity." Immunological reviews 260(1):86-101.
- 3. Hirota, Keiji, et al. (2013). "Plasticity of TH 17 cells in Peyer's patches is responsible for the induction of T cell–dependent IgA responses." Nature immunology 14(4):372-379.
- 4. Balasa, Rodica, et al. (2020). "The action of TH17 cells on blood brain barrier in multiple sclerosis and experimental autoimmune encephalomyelitis." Human immunology 81(5):237-243.

- 5. Kebir, Hania, et al. (2007). "Human TH 17 lymphocytes promote blood-brain barrier disruption and central nervous system inflammation." Nature medicine 13(10):1173-1175.
- Platt, Maryann P., et al. (2020). "Th17 lymphocytes drive vascular and neuronal deficits in a mouse model of post infectious autoimmune encephalitis." Proceedings of the National Academy of Sciences 117(12):6708-6716.
- Gong, Tao, et al. (2020). "DAMP-sensing receptors in sterile inflammation and inflammatory diseases." Nature Reviews Immunology 20(2):95-112.
- Molteni, Monica, Sabrina Gemma, and Carlo Rossetti. (2016). "The role of toll-like receptor 4 in infectious and noninfectious inflammation." Mediators of inflammation 2016.
- Bhattacharyya, Swati, and John Varga. (2018). "Endogenous ligands of TLR4 promote unresolving tissue fibrosis: Implications for systemic sclerosis and its targeted therapy." Immunology letters 195:9-17.
- Wiertz, Emmanuel J., et al. (2007). "Herpesvirus interference with major histocompatibility complex class II-restricted T-cell activation." Journal of virology 81(9):4389-4396.
- Neumann, Jürgen, Anna Maria Eis-Hübinger, and Norbert Koch. (2003). "Herpes simplex virus type 1 targets the MHC class II processing pathway for immune evasion." The Journal of Immunology 171(6):3075-3083.
- Münz, Christian. (2016). "Autophagy beyond intracellular MHC class II antigen presentation." Trends in immunology 37(11):755-763.
- Milovanovic, Jelena, et al. (2020). "Interleukin-17 in chronic inflammatory neurological diseases." Frontiers in Immunology 11:947.
- Babaloo, Zohreh, et al. (2015). "The role of Th17 cells in patients with relapsing-remitting multiple sclerosis: interleukin-17A and interleukin-17F serum levels." Immunology letters 164(2):76-80.
- 15. Kolbinger, Frank, et al. (2016). "IL-17A and multiple sclerosis: signaling pathways, producing cells and target cells in the central nervous system." Current drug targets 17(16):1882-1893.
- 16. Does Anyone Have Piriformis Syndrome? | My MS Team
- 17. Sciatica and MS: Differences and Treatments (healthline.com)
- Drenska, Kalina V., and Ara G. Kaprelyan. (2013). "Simultaneous disc herniation in patients with multiple sclerosis." Journal of IMAB–Annual Proceeding Scientific Papers 19(1):399-401.
- Mullen, Ann E., Mary Ann Wilmarth, and Sue Lowe. (2012). "Cervical disk pathology in patients with multiple sclerosis: two case reports." (2012):1055-1064.

- 20. Causes and Symptoms of Waterborne Illness Minnesota Dept. of Health (state.mn.us).
- Cissé, Guéladio. (2019). "Food-borne and water-borne diseases under climate change in low-and middle-income countries: Further efforts needed for reducing environmental health exposure risks." Acta tropica. 194:181-188.
- 22. Yates, Johnnie A., and Lori C. Stetz. (2006). "Reiter's syndrome (reactive arthritis) and travelers' diarrhea." Journal of Travel Medicine 13(1):54-56.
- 23. Hayes, Kaitlyn M., et al. (2019). "Evolving patterns of reactive arthritis." Clinical rheumatology 38(8):2083-2088.
- 24. Reynolds, Kelly A. (2007). "Water-based Virus May Cause Rheumatoid Arthritis." Clinical Infectious Diseases 45(1):95-102.
- 25. Uotila, T., et al. (2011). "Reactive arthritis in a population exposed to an extensive waterborne gastroenteritis outbreak after sewage contamination in Pirkanmaa, Finland." Scandinavian journal of rheumatology 40(5):358-362.
- 26. Donmez, Hanife Guler, et al. (2020). "Is bacterial vaginosis associated with autoimmune antibody positivity?" Cytopathology 31(4):298-302.
- 27. Ginsburg, Katherine S., et al. (1992). "Ureaplasma urealyticum and Mycoplasma hominis in women with systemic lupus erythematosus." Arthritis & Rheumatism: Official Journal of the American College of Rheumatology 35(4):429-433.
- Judlin, P. G., and O. Thiebaugeorges. (2009). "Pelvic inflammatory diseases." Gynecology, obstetrics & fertility. 37(2):172-182.
- 29. Soper, David E. (1996). "Pelvic inflammatory disease (PID)." Infectious diseases in obstetrics and gynecology 4(2):62.
- Kreisel, Kristen, et al. (2017). "Prevalence of pelvic inflammatory disease in sexually experienced women of reproductive age— United States, 2013–2014." MMWR. Morbidity and mortality weekly report 66(3):80.
- Bhattacharyya, Mau, et al. (2007). "Systemic production of IFNα by garlic (Allium sativum) in humans." Journal of Interferon & Cytokine Research 27(5):377-382.
- Bakre, Adetolase A., Omolade A. Oladele, and Oluwaseun O. Esan. (2020). "Garlic Enhances Cellular Immune Response via Increased Secretion of IFN-γ-in Chickens." Alexandria Journal for Veterinary Sciences 66(2).
- 33. Percival, Susan S. (2016). "Aged garlic extract modifies human immunity." The Journal of nutrition 146(2): 433S-436S.



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