Dear Rep Woods,

I would like to submit the following for the record of the study group:

ARE ANTIBIOTICS USEFUL FOR TREATING CHRONIC LYME DISEASE PATIENTS? MYLYMEDATA STUDY PROVIDES SOME ANSWERS.

https://www.lymedisease.org/antibiotics-for-lyme-disease/

Excerpt:

"...longer treatment durations were associated with better treatment response—with most high responders and well patients reporting treatment durations of four or more months and many reported durations exceeding a year. As the chart below reveals, those treated for less than a month were unlikely to report improvement."

In contrast, the "Klempner Trials" were stopped after only three months:

Two Controlled Trials of Antibiotic Treatment in Patients with Persistent Symptoms and a History of Lyme Disease

http://www.nejm.org/doi/ref/10.1056/NEJM200107123450202#t=references

Mark S. Klempner, M.D., Linden T. Hu, M.D., Janine Evans, M.D., Christopher H. Schmid, Ph.D., Gary M. Johnson, Richard P. Trevino, B.S., DeLona Norton, M.P.H., Lois Levy, M.S.W., Diane Wall, R.N., John McCall, Mark Kosinski, M.A., and Arthur Weinstein, M.D.

N Engl J Med July 12, 2001

Conclusion:

"In these two trials, treatment with intravenous and oral antibiotics for 90 days did not improve symptoms more than placebo."

Per the 1992 publication below, Klempner reported <u>antibiotic resistance</u> as fibroblasts protected B. burgdorferi for at least 14 days of exposure to ceftriaxone. We have known for decades that we're dealing with an antibiotic resistant/tolerant superbug.

<u>J Infect Dis.</u> 1992 Aug;166(2):440-4.

Fibroblasts protect the Lyme disease spirochete, Borrelia burgdorferi, from ceftriaxone in vitro.

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Abstract

The Lyme disease spirochete, Borrelia burgdorferi, can be recovered long after initial infection, even from **antibiotic-treated patients**, indicating that it resists eradication by host defense mechanisms and antibiotics. Since B. burgdorferi first infects skin, the possible protective effect of skin fibroblasts from an antibiotic commonly used to treat Lyme disease, ceftriaxone, was examined. Human foreskin fibroblasts protected B. burgdorferi from the lethal action of a 2-day exposure to ceftriaxone at 1 microgram/mL, 10-20 x MBC. In the absence of fibroblasts, organisms did not survive. Spirochetes were not protected from ceftriaxone by glutaraldehyde-fixed fibroblasts or fibroblast lysate, suggesting that a living cell was required. The ability of the organism to survive in the presence of fibroblasts was not related to its infectivity. Fibroblasts protected B. burgdorferi for at least 14 days of exposure to ceftriaxone. Mouse keratinocytes, HEp-2 cells, and Vero cells but not Caco-2 cells showed the same protective effect. Thus, several eukaryotic cell types provide the Lyme disease spirochete with a protective environment contributing to its long-term survival.

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