Boffin Access

Open Access Journal | Volume: 2.1

Journal of Emerging and Rare Diseases

ISSN 2517-7397

The Sixth Extinction: Global Warming Release of Disease from the Permafrost?

Bruce Rothschild[1,2*]

[1]Ball Memorial Hospital, Indiana University, Muncie, Indiana, USA
[2]Carnegie Museum, Pittsburg, Pennsylvania, USA

Summary

We are on the cusp of a sixth extinction. The most recent was the Pleistocene extinction, which established the environment permitting development of modern humans. Current permafrost warming is releasing into the environment organisms with epidemic histories, for which we are currently unprepared. The level of preservation in the permafrost is so pristine that cloning has been contemplated for animals preserved within. Viable viruses and bacteria have been found in 30,000 year old Siberian permafrost. Animal contamination with permafrost-derived diseases (e.g., anthrax) has already occurred. Organisms of concern include *Mycobacerium tuberculosis* and atypical mycobacteria, fungi, *Brucella* sp., *Yersinia pestis*, *Variola*, *Influenza* virus, *Hantavirus* and zoonoses and animal epidemics. We are at risk of catastrophic epidemics related to release of viable microorganisms from the melting permafrost. Unless the causative warming can be halted, it is only a matter of time.

Keywords

Contamination; Global warming; Smallpox; Ancient disease; Epidemics

Five major extinctions (Table 1) are responsible for creating the environments for a change in direction of the evolutionary process [1-3]. The Permian extinction permitted dinosaurs and their ancestors to become the major Mesozoic megafauna. The Cretaceous extinction paved the way for mammals to similarly achieve subsequent prominence. It is unclear what was wrought by the Pleistocene extinction, except perhaps the rise of modern humans and the opportunity for our own self-destruction.

The source of the most recent extinction has been subject to great debate. Four hypotheses have been offered: Environmental (climate) change, human "intervention" (e.g., hunting related overkill), extraterrestrial impact and hyperdisease [4,5]. Disease was clearly present throughout at least the late Pleistocene. Presence of the *Mycobacterium tuberculosis* complex was documented both by DNA and lipid analyses in bovids (e.g., bison, cattle, big horn sheep, musk oxen) and mastodon through at least the last 100,000 years of the Pleistocene [6,7]. The population prevalence of bone evidence of disease ranged from 25 to 50%. Since not all individuals with the disease have bone involvement, the implication is that virtually 100% of those species were afflicted with tuberculosis. That would suggest that tuberculosis was responsible for their extinction. However, that population prevalence remained unchanged for 100,000 years. While penetrance of the disease into the population was essentially complete, sustainability of the involved species was apparently not impaired-until the latest Pleistocene.

As only 5% of humans with tuberculosis have active, debilitating disease [8], it is clear that a very effective mechanism is present that reduces the impact/spread and metabolic effects of tuberculosis. It is only when a stressor is "applied" that the majority of tuberculosis-afflicted individuals succumb to the disease [9]. The debate has persisted for over a century as to the responsibility of climate change, human action (e.g., hunting), extraterrestrial impact or disease for the Pleistocene extinction [3,4,10]. Lyons et al. [4] went as far as to suggest that humans were the source of the dooming infections, so the

Extinction Event	Million ybp*	Families affected	Genera affected	Species affected	Life affected
Ordovician-Silurian	450-439	27%	57%	60-70%	86%
Devonian-Carboniferous	375-360	19%	50%	70%	75%
Permian-Triassic	252-251	57%	83%	90-96%	96%
Triassic-Jurassic	214-199	23%	48%	70-75%	80%
Cretaceous-Paleogene	66-65	17%	50%	75%	76%

*Years before present

Table 1: Timing and effect of past major extinctions

Article Information

DOI: 10.31021/jer.20192113
Article Type: Short Communication
Journal Type: Open Access
Volume: 2 **Issue:** 1
Manuscript ID: JER-2-113
Publisher: Boffin Access Limited

Received Date: 29 January 2019
Accepted Date: 08 February 2019
Published Date: 11 February 2019

*Corresponding author:

Bruce Rothschild
Ball Memorial Hospital
Indiana University, Muncie
IN 47303, USA
Tel: 785-615-1523
E-mail: spondylair@gmail.com

Citation: Rothschild B. The sixth extinction: global warming release of disease from the permafrost? J Emerg Rare Dis. 2019 Feb;2(1):113

hypothesized causes of extinction are not necessarily distinct.

Some, including the author (BMR), have suggested that a single cause was unlikely, but that a multiple "hit" phenomenon was in play. Perhaps it was stresses induced by climate change or human activity that compromised the accommodation to the disease?

Tuberculosis, however, was not a new disease when extinction occurred [7]. Hosts had a long opportunity to develop an accommodation. Perhaps the result would have been very different if there had been a sudden exposure to a new disease. The history of American Indian response to long-standing European afflictions illustrates the draconian effect of such contamination [11].

Whatever the role of humans in producing or abetting global warming, there is a clear and present danger to continuation of that process. As temperatures rise, natural barriers to spread of infection have been compromised. More equatorial and temperate diseases extend their range to newly hospitable environs [12]. Thus, phoma stem canker disease of oil seed rape plants is now found in the northern United Kingdom, Lyme as far north as Canada, and expansion of bovine tuberculosis in Great Britain and chytridiomycosis to amphibians [13-15]. Diseases which had been thermally "quarantined" have now expanded their ranges. Previous territorial limitations to infectious disease recommendations for vaccination are no longer sufficient, resulting in an expanded susceptible pool with an epidemiologically-expansile potential. Some diseases have apparently been eliminated or have been so reduced in prevalence and distribution as to have little epidemiological significance. Habitat alteration (e.g., spraying for the mosquitoes carrying the virus or utilizing protective modalities) has been a major factor in minimizing their environmental impact [16], but the environment is now creating new opportunities for disease spread.

Global warming alters plant zones and growing seasons [17], which has the potential to impact food supplies. Such could potentially be an extinction-"directing" event, but there are other more direct dangers lurking.

The permafrost has been a buffet for study of ancient DNA and protein [18,19]. The level of preservation is so pristine that cloning has been contemplated for animals preserved within [20]. Animals, however, are not the only preserved organisms. Viability of contained bacteria and viruses has been suggested. These organisms have had the opportunity for "containment" in the permafrost refrigerator since the Pleistocene creation of that ecological zone. Burial (natural or human activity-related) have added more modern organisms, including those of *Babesia* sp., *Bacillus anthracis*, *Vibrio cholera*, *Bordetella pertussis*, *Francisella tularensis*, *Yersinia pestis*, *Influenza*, *Variola* and West Nile Virus [4,21-27].Giant viruses, *Pithovirus* and *Mollivirus*, have been found in 30,000 year old Siberian permafrost, retaining sufficient viability to infect amoebae [20,28,29]. The last two viruses were revived from 30,000 year-old Siberian permafrost. Viable *Carnobacterium pleistocenium* have been isolated from 32,000 year old permafrost and much older bacteria have been isolated in viable form from the Antarctic [30]. Exposure of permafrost burials have transmitted anthrax and smallpox [25,30-33]and those who met their demise from the 1917 influenza epidemic may well harbor organisms with potential for infecting new victims. One concern has been for contamination of those who excavate such sites, leading to hazard suit usage and other preventative and decontamination approaches. Such precautions are quite reasonable and actually feasible to pursue when potential exposure sites are identifiable. Global warming, however, provides a general opportunity for new environmental exposures, ones not under scrutiny and therefore with great opportunity for undetected spread (at least initially).

Extinction and extinction-level effects by infectious agents has been noted in the land snail *Partula turgida* (microsporidium *Steinhausia*-related), black-footed ferrets *Mustela nigripes* (canine distemper virus-related), Tasmanian devils *Sarcophilus harrissi* (transmissible cancer), koala *Phascolarctos cinereus* (*Chlamydia pecorum*-related), little brown bat *Myotis lucifugus* (*Geomyces destructans*-related), frogs (*Batrachochytrium dendobatidis*-related) and Christmas Island rat *Rattus macleari* (*Trypanosoma lewisi*-related) [26,34-38]. Animal contamination with permafrost-derived diseases (e.g., anthrax) has already occurred [30,31-33], Organisms of concern include *Mycobacerium tuberculosis* and atypical mycobacteria, fungi, *Brucella* sp., *Yersinia pestis*, *Variola*, *Influenza* virus, *Hantavirus* and zoonoses and animal epidemics.

There is an additional consideration: Multiple approaches allow recognition of known pathogens, but an extinct pathogen, brought back to life by permafrost melting, escapes such targeted analyses. When a priori knowledge is lacking as to which organisms may be present, such targeted analyses often fail, even for contemporary organisms. Metagenomics overcomes those limitations and provides an opportunity for detection of organisms independent of current knowledge of their pathogenicity or even of their very existence [39]. Also referred to as "shotgun" sequencing [40], it allows identification of all organisms present, not just those for which one has the insight to search [41-43].

Arens and West [44] suggested the effect combination of "long term pressure on the eco-system" and a sudden catastrophe. The same two-hit opportunity for extinction exists today, although the pressure and upcoming catastrophe are integrally connected. Global warming exposes permafrost-contained pathogens to an unprotected population. This certainly is a time of stress and new introduction of an old disease could be catastrophic. Many have been skeptical about the infectious disease extinction hypothesis, but there is now reasonable fear we are about to test that very hypothesis.

Conflicts of Interest

None.

References

1. Alvarez LW, Alvarez W, Asaro F, Michel HV. Extraterrestrial cause for the cretaceous-tertiary extinction. Science. 1980 Jun;208(4448):1095-1108.
2. Erwin DH. The Permo-Triassic extinction. Nature 1994;367(1):231-236.
3. Martin PS, Klein RG. Quaternary Extinctions: A Prehistoric Revolution. Arizona Press, Tucson. 1984.
4. Lyons SK, Smith FA, Wagner PJ, White EP, Brown JH. Was a 'hyperdisease' responsible for the Late Pleistocene megafaunal extinction? Ecol Lett 2004;7(1):859-868.
5. McCallum H. Disease and the dynamics of extinction. Phil Trans Roy Soc B. 2012;367(1):2828-2839.
6. Rothschild BM, Laub R. Hyperdisease in the late Pleistocene: validation of an early 20th century hypothesis. Naturwissenschaften. 2006 Nov;93(11):557-564.
7. Rothschild BM, Martin D. Did Ice Age bovids spread tuberculosis? Naturwissenschraften. 2006;93(1):565-569.
8. Dutta NK, Karakousis PC. Latent tuberculosis infection: Myths, models and molecular mechanisms. Microbiol Mol Biol Rev 2014;78(3):343-371.
9. Scanga CA, Mohan VP, Joseph J, Yu K, Chan J, et al. Reactivation of latent tuberculosis: Variations on the Cornell murine model. Infect Immun. 1999;67(9):4531-4538.
10. Koch PL, Barnosky AD. Late Quaterrnary extinctions: State of the debate. Ann Rev Ecol Evol Syst. 2006;37(1):215-250.
11. Rossi A. Two Cultures Meet: Native American and European. Washington, DC: Natl Geograph Soc. 2002:31-32.
12. Ogden NH, Maarouf A, Barker IK, Bigras-Poulin M, Lindsay LR, et al. Climate change and the potential for range expansion of the Lyme disease vector Ixodes scapularis in Canada. Int J Parasitol. 2006 Jan;36(1):63-70.
13. Bosch J, Carrascal LM, Duran L, Walker S, Fisher MC. Climate change and outbreaks of amphibian chytridiomycosis in a montane area of central Spain; is there a link? Proc Roy Soc B.

2007;274(1607):253-260.

14. Brownstein JS, Holford TR, Fish D. Effect of climate change on Lyme disease risk in North America. Ecohealth. 2005;2(1):38-46.
15. Wint GR, Robinson TP, Bourn DM, Durr PA, Hay SI, et al. Mapping bovine tuberculosis in Great Britain using environmental data. Trends Microbiol. 2002 Oct;10(10):441-444.
16. Severo OP. Eradication of the Aedes aegypti mosquito from the Americas. In: Yellow Fever, a Symposium in Commemoration of Carlos Juan Finlay, 1955. Jefferson Digital Commons. 1955;6:39-59.
17. McKenney DW, Pedlar JH, Lawrence K, Papadopol P, Campbell K, et al. Change and evolution in the plant hardiness zones of Canada. Bio Science. 2014;64(4):341-350.
18. Lewis K, Epstein S, Godoy VG, Hong S-H. Intact DNA in ancient permafrost. Trends Microbiol. 2008;16(3):92-94.
19. Lydolph MC, Jacobsen J, Arctander P, Gilbert MT, Gilichinsky DA, et al. Beringian paleoecology inferred from permafrost-preserved fungal DNA. Appl Environ Microbiol. 2005 Feb;71(2):1012-1017.
20. Loi P, Saragusty J, Ptak G. Cloning the mammoth: A complicated task or just a dream? Adv Exp Med Biol. 2014;753(1):489-502.
21. Hornick RB. Tularemia. In: Hoeprich PD, Jordan MC, Ronald AR. Infectious Diseases: A Treatise of Infectious Processes. New York: Lippincott;1994:1296-1297.
22. Kenefic LJ, Pearson T, Okinaka RT, Schupp JM, Wagner DM, et al. Pre-Columbian origins for North American anthrax. PLoS One. 2009;4(3):e4813.
23. Nickell ZD, Moran MD. Disease introduction by Aboriginal humans in North America and the Pleistocene extinction. J Ecol Anthropol. 2017.
24. Revich BA, Podolnaya MA. Thawing of permafrost may disturb historic cattle burial grounds in East Siberia. Global Health Action. 2011;4(1):8482.
25. Shadomy S, El Idrissi A, Raizman E. Anthrax outbreaks: A warning for improved prevention, control and heightened awareness. Food Agricult Org UN. 2016;37(1):1-7.
26. Taubenberger JK, Baltimore D, Doherty PC, Markel H, Morens DM, et al. Reconstruction of the 1918 influenza virus: unexpected rewards from the past. MBio. 2012 Sep;3(5): e00201-e00212.
27. Vannier E, Gewurz BE, Krause PJ. Human babesiosis. Infect Dis Clin North Am. 2008;22(3):469-488.
28. Legendre M, Lartigue A, Bertaux L, Jeudy S, Bartoli J, et al. In-depth study of Mollivirus sibericum, a new 30,000-y-old giant virus infecting Acanthamoeba. Proc Natl Acad Sci U S A. 2015 Sep;112(38):E5327-E5335.
29. Yong E. Giant virus resurrected from 30,000-year-old ice-Largest virus yet discovered hints at viral diversity trapped in permafrost. Nature. 2014.
30. Luu C. The dystopian future is only just beginning, thanks to climate change. Refinery. 2017.
31. Doucleff M. Anthrax outbreak in Russia thought to be result of thawing permafrost. Npr. 2016.
32. Goudarzi S. What Lies Beneath. Sci Am. 2016 Oct;315(5):11-12.
33. Popov A. Anthrax strikes in rapidly thawing Arctic. Cnn. 2016.
34. Cunningham AA, Daszak P. Extinction of a species of land snail due to infection with a microsporidian parasite. Conserv Biol. 1998;12(5):1139-1141.
35. Jackson M, White N, Giffard P, Timms P. Epizootiology of Chlamydia infections in two free-range koala populations. Veterin Microbiol. 1999;65(4):255-264.
36. Pearse AM, Swift K. Allograft theory: Transmission of devil facial-tumor disease. Nature. 2006;439(7076):549.
37. Skerratt LF, Berger L, Speare R, Cashins S, McDonald KR, et al. Spread of chyridiomycosis has caused the rapid global decline and extinction of frogs. Eco Health. 2007;4(1):125-134.
38. Warnecke L, Turner JM, Bollinger TK, Lorch JM, Misra V, et al. Inoculation of bats with European Geomyces destructans supports the novel pathogen hypothesis for the origin of white-nose syndrome. Proc Natl Acad Sci U S A. 2012 May;109(18):6999-7003.
39. Miller RR, Montoya V, Gardy JL, Patrick DM, Tang P. Metagenomics for pathogen detection in public health. Genome Med. 2013;5(9):81.
40. Relman DA. Metagenomics, infectious disease diagnostics, and outbreak investigations: Sequence first, ask questions later? JAMA. 2013;309:1531-1532.
41. Wu J, Cal K, Feng Q. The application of metagenomics approaches in the management of infectious diseases. Trop Med Surg. 2015;3:4.
42. Blomström A-L. Viral metagenomics as an emerging and powerful tool in veterinary medicine. Veterin Quart. 2011;31:107-114.
43. Mokili JL, Rohwer F, Dutilh BE. Metagenomics and future perspectives in virus discovery. Curr Opin Virol. 2012;2:63-77.
44. Arens NC, West ID. Press-pulse: A general theory of mass extinction? Paleobiol. 2008;34(4):456-471.