The verifiable origin of manifestations that are attributed to infectious agent transmission on the basis of analogy

Paul Ola

Institute for Exact Biomedical Sciences, Lagos, Nigeria

paulolatheorist@gmail.com

Abstract

For as long as it has been concluded on the basis of the similarity between the microscopic entities to which attention is called by the clearly different manifestations in patients with a particular diagnosis, that the origin of such manifestations lies in the transmission of a specific "infectious agent," it has remained impossible to prevent their worsening towards deaths and disabilities with precision. Following the principle in Sun Tzu's 'Art of War,' that the source of the foreknowledge by which the most successful armies in history decisively defeated the enemies of their people is not analogy but the findings of people who know the enemy situation, we chose to investigate the origin of such manifestations with the method of General Relativity, which requires medical foreknowledge to begin in the findings of those clinicians who fight the actual enemies of patients at the bedside. The results enable us to verifiably show here that the focus of research towards furnishing the foreknowledge that the "medical army" requires to defeat these enemies ought to be the investigation of a "deconditioning factor" that causes the failure of the mechanisms that render the illnesses that persist due to the absence of recovery unapparent or asymptomatic by means of myoglobin, even when the precipitation of severity by silently

harbored microscopic entities is so intense that sudden death occurs before the detection of these entities when such apparent health mechanisms fail.

.

Key words: Public health, viruses, monkeypox, COVID-19, immunity, apparent health, myoglobin

While developing the method by which Albert Einstein unravelled the phenomena behind astronomical observations by describing the physical reality in which such phenomena occur in the mathematical language of geometry [1], we furnished results with implications for the origin of the multi-country monkeypox outbreak outside of the African regions [2-5] where they were first reported [6,7], as well as the origin of the COVID-19 pandemic [8,9]. Given that we were first clinicians before we realized that the solution to the problem constituted by the inability of geometry to permit the conception of things that exist in other physical realities [1] lies in the development of a non-mathematical language which may permit even the conception of those in the clinical reality beneath our patients' skins, the goal of the papers in which we presented such results was that which required us to leave our colleagues at the bedside to solve this problem.

That goal, the understanding researchers require to develop interventions that will permit clinicians to finally defeat the deaths that occur in patients in whom even their best critical care efforts fail as well as the progressive disabilities that often make death preferable to life for survivors, required us, in our quest for the requisite scientific method, to be guided by the fundamental principle that the most successful armies in history obeyed on their

path to decisively defeating the enemies of their people. According to Sun Tzu's description of this principle in the famous 'Art of War,' the foreknowledge required to decisively defeat the enemy cannot be elicited by analogy with past events, nor from calculations and must be obtained from people who know the situation of the enemy [10].

The microscopic researches of Louis Pasteur yielded findings that enabled him to substantiate the germ theory towards the proof furnished by Joseph Lister by leading him to the verifiable proposition of microscopic organisms as the germs that antiseptic procedures prevent from being brought by clinicians into the bodies of patients towards such marked reduction in mortality [11] that followed the introduction of such procedures by Ignaz Semmelweis [12]. But his findings did not furnish understanding of the origin of the manifestations that clinicians fight at the bedside, which he proposed as residing essentially and uniquely in the presence of such clearly transmissible organisms [11] on the basis of the conception of such manifestations as transmissible, contagious and infectious diseases. After all, the theoretical consequence of his discovery that "sugar never undergoes alcoholic fermentation without the presence of living globules of yeast" is that the effects of microscopic organisms [11], which are conceived as diseases within the bodies of humans and the animals that are the concern of our colleagues in veterinary healthcare, are only necessary for the appearance of such manifestations just like the appearance of alcohol through such fermentation.

It was as clear to us as daylight upon considering these facts that those who know the situation of the actual enemies of patients are not the theorists who have conceived their manifestations as transmissible since ancient times but the clinicians who have fought

them at the bedside where patients succumb despite best efforts and made the findings in which our search for the pathogenesis of such manifestations must begin. Therefore, we abandoned conclusions such as that which, on the basis of the similarity between the viruses to which skin manifestations called attention in African villages in the 1970s and those which were earlier detected in research monkeys, proposed transmission from animals to humans as the origin of such manifestations that monkeypox has since labelled as well as that which proposed sexual transmission to account for their origin outside of Africa in 2022, when most cases were men who have had sex with men [13].

Our focus in the quest to justifiably address the origin of those deaths that occur even in apparently healthy individuals with none of the underlying conditions [14] which must be invoked to account for them following the monkeypox diagnosis for as long as the manifestations that are expected to be transmitted to the diagnosed are such skin manifestations [15], similar to the cardiovascular cause which was invoked for the sudden cardiac death in an apparently healthy 32-year-old with the COVID-19 diagnosis because the respiratory tract manifestations in similarly diagnosed patients in Wuhan were expected to be transmitted by the detected coronavirus [16]. The following is what we find upon abandoning what is known from analogy about manifestations that Pasteur's version of the germ theory attributed to the transmission of microscopic entities and sufficiently considering the findings of clinicians who are at the bedside of the dying, from the different forms of manifestations of plague that Guy de Chauliac reported [17] to such form of COVID-19 manifestations that features sudden death even on arrival in healthy individuals without the expected respiratory tract manifestations [18]. *The nature of the manifestations that are conceived as transmissible, infectious and contagious is not*

determined by the microscopic entities that are assumed to be similar in the affected because they are descendants of a specific microscopic entity.

This conclusion implies that *the similarities between microscopic entities on the basis of which patients are diagnosed the same despite the differences between the forms of their manifestations are not the consequence of common ancestry but of common evolution following exposure to similar conditions*. Such evolutionary changes that make microscopic entities of different ancestry so similar upon exposure to similar conditions that they would appear to the mind that relies on analogy to have descended from a common ancestor explain, without assumptions that Charles Darwin's theory of universal common ancestry would have to make, why Mimivirus is so similar to bacteria in cell structure that it was believed to be a bacterium until the absence of a ribosome resulted in its recognition as virus [19].

But our concern here is not the implied theory of *evolution by means of exposure to conditions* which, by avoiding the following guidance of analogy, will permit biologists to account for the phenomena behind the similarities between clearly different biological entities without such debates as that between those who would rather preserve Charles Darwin's Tree of Life and colleagues who argued following the discovery of Mimivirus that viruses should not be dismissed as non-living material for the preservation of this inaccurate model of the relationship between all living things [20-28]. "Analogy would lead me one step further, namely, to the belief that all animals and plants have descended from some one prototype. But analogy may be a deceitful guide. Nevertheless, all living things have much in common…" [29].

What we shall concern ourselves with in the current work are the following questions that clinical findings require us to justifiably answer on the path where the actual factor that determines the nature of the manifestations that are attributed to infectious agent transmission is to be verifiably described. 1) Why are some forms of manifestations, such as that with the “the spitting of blood” which was conceived by Guy de Chauliac as being so contagious that it could be caught from another person “not just when living nearby but simply by looking” [16], suddenly become so widespread in a population that spread over a distance must be invoked to account for the speed of transmission that is deduced with the guidance of analogy because close contact cannot? 2) Why do such forms of manifestations which have been conceived as rapidly spreading in the past suddenly appear in cases that are so far removed from others, as smallpox manifestations were towards the declaration of their eradication when even indistinguishable manifestations which were labelled monkeypox manifestations also emerged in similarly isolated cases [30,31], that the transmission deduced with the guidance of analogy is conceived as having become slower?

In the clinical reality that we have grasped, the manifestations which monkeypox has since labelled cannot not be distinguished from those that smallpox has labelled since ancient times when their appearance is the consequences of similar exposure to the actual determinant of the nature of the manifestations that are attributed to infectious agent transmission on the basis of analogy. To the mind that is guided by the clinical findings that permitted us to grasp this clinical reality, the following is suggested by those that reveal those skin manifestations that are attributed to the transmission of a virus from

monkeys [6,7] to be so similar to skin manifestations in patients with incomparable microscopic entities such as the syphilis bacterium that mimicry had to be invoked to account for the observed phenomena [32] through the lens furnished by analogy. *The nature of the manifestations that are attributed to the transmission of a specific infectious agent does not depend on conditions that permit different microscopic entities in the affected to become so similar that they appear to the analogy-guided mind to have a common ancestor but rather on conditions which may permit similar manifestation of illnesses in patients with incomparable microscopic entities when exposure to them is similar.*

Therefore, rather than focusing on the nature of microscopic entities that are conceived as infectious agents in our quest for the knowledge researchers require to develop interventions by which progressive worsening of the outcome in patients with such manifestations may be prevented by clinicians, we deduced an empirical law of the critical illness that permits mechanistic understanding of the phenomena behind such manifestations that may be the same regardless of differences between the microscopic entities on the basis of which the affected are diagnosed [33].

In the grasped clinical reality, individuals may be asymptomatic and apparently healthy even when severity is already precipitated by the microscopic entities they harbour undetected due to the failure of the immunity mechanisms that abate such precipitation and sudden death that follows rapid deterioration may become their fate upon the failure of apparent health mechanisms [33], even without the manifestations that are expected to call attention to the presence of such entities on the basis of analogy, as observed even in

those patients who succumb before arrival at the hospital where such entities are detected [18]. Furthermore, in the same reality where the findings of Bussani et al [34] are accommodated, those who are exempted from such fate by immunity mechanisms may deteriorate nevertheless and eventually succumb as these mechanisms that permit survival despite persistent failure of apparent health mechanisms progressively fail to abate severity precipitation by microscopic entities.

In the light of these, we realize that what the field of public health is to focus on more is not the prevention of the transmission of microscopic entities, which may already be harboured undetected even after they have already fatally precipitated the severity of illnesses that persist in the absence of recovery and the abating effects of immunity mechanisms, but rather, of exposure to a factor that eliminates the conditions that permit the asymptomatic status that apparent health mechanisms bring about even in the face of unabated precipitation by such entities. Given that our results suggest that the increase in serum myoglobin relative to prior levels that follows the failure of apparent health mechanisms upon exposure to this factor depends on the degree of reliance on the conditioning that suppresses exertion intolerance mechanisms towards the reduced exertional myoglobinemia [35] that Ritter et al found to be its effect [36], we may refer to it as the "deconditioning factor" for simplicity.

The following are the questions that this focus requires researchers to urgently answer in the light of the consequences of the law of the critical illness that recently permitted, through the reduction in serum myoglobin relative to prior levels (relative serum myoglobin decline), quantification of the processes behind the restoration of the effects

of apparent health mechanisms that results in the disappearance of the fatiguing symptoms whose worsening with time [35] may be followed by sudden death in patients on arrival at the hospital even without the symptoms that are expected on the basis of analogy [18].

1) What predicts an association between worsening of outcomes and the elevation of serum levels of myoglobin relative to prior values (relative serum myoglobin elevation) that is to be observed in the non-recovering patient when exposure to the deconditioning factor persists? 2) What predicts a change in course towards relative serum myoglobin decline that is sufficient for restoration of the asymptomatic status after severe outcomes (a favourable prognosis) regardless of how worsened outcomes may be during relative serum myoglobin elevation, which is to be observed in the non-recovering patient when exposure to the deconditioning factor fails to persist?

In the reality to be grasped towards the answers to these questions, the proportion of the population that is exposed to the deconditioning factor at a particular time may be so large that no speed of transmission of microscopic entities by contact will ever be able to account for the widespread appearance of manifestations that will follow widespread failure of immunity mechanisms to abate severity precipitation by already harboured and transmitted microscopic entities. Yet, in the same reality, exposure to this factor may be so rare at other times that manifestations will appear in no more than a member of a household or even a community, as the case was on the path to the conception of the viruses which were assumed on the basis of analogy to transmit those in monkeypox

patients as spreading with difficulty and the wrong conclusion that they will not become the public health problem [6,7] they clearly are now [37].

We owe it to our people to furnish these answers towards interventions that will permit the prevention of exposure to this deconditioning factor or its persistence, especially in the subset in whom the precipitation of severity by silently harboured microscopic entities has become so intense and yet unabated that sudden death may follow the failure of apparent health mechanisms even before arrival at the hospital and without the manifestations that are expected to call attention to such entities on the basis of analogy.

References

1. Einstein A. On the Method of Theoretical Physics. Philosophy of Science. 1934;1(2):163-169. doi: 10.1086/286316

2. Ola P. The origin of the mysterious multi-country monkeypox outbreak in non-endemic countries. OSF Preprints 2022, Jun 2. doi: 10.31219/osf.io/c4q8d.

3. Ola P. Monkeypox Is the Manifestation of Spectral Diseases and Not of Monkeypox Virus Transmission. OSF Preprints. 2022, Aug 31. doi:10.31219/osf.io/k3ayb.

4. Ola P. Monkeypox Deaths Demand Understanding of Spectral Theory of Infectious Disease Pathogenesis. OSF Preprints. 2022, Sept 15. doi:10.31219/osf.io/mwtns.

5. Ola P. Monkeypox Deaths Are Not Consequences of Underlying Conditions but of Severe Outcomes That Occur Even in Healthy Individuals. OSF Preprints. 2022. Nov 21. doi:10.31219/osf.io/2k3dn.

6. Breman JG, Kalisa-Ruti null, Steniowski MV, Zanotto E, Gromyko AI, Arita I. Human monkeypox, 1970-79. Bulletin of the World Health Organization. 1980;58(2):165-182.

https://pubmed.ncbi.nlm.nih.gov/6249508/

7. Arita I, Henderson DA. Monkeypox and whitepox viruses in West and Central Africa. Bull World Health Organ. 1976;53(4):347-53. https://pubmed.ncbi.nlm.nih.gov/186209/

8. Ola P. Zero COVID Is Possible but Preventing Coronavirus Transmission Is Not the Path to This Noble Reality. OSF Preprints. 2022, Nov 29. doi:10.31219/osf.io/s5aq4.

9. Ola P. Severe Outcomes in the Infected Are Not Consequences of Pathogenicity and Fatalities Do Not Decline Because the Pathogen Is Weakened. OSF Preprints. 2022. Dec 6. doi:10.31219/osf.io/5j96y.

10. McNeilly M. 'Deception and Foreknowledge: Winning the information War', Sun Tzu and the Art of Modern Warfare (New York, 2015; online edn, Oxford Academic, 10 Feb. 2015), https://doi.org/ 10.1093/acprof:osobl/9780199957859.003.0004 accessed 28 Sept. 2024.

11. Smith KA. Louis Pasteur, the father of immunology? Front Immunol. 2012 Apr 10;3:68. doi: 10.3389/fimmu.2012.00068.

12. Stang A, Standl F, Poole C. A twenty-first century perspective on concepts of modern epidemiology in Ignaz Philipp Semmelweis' work on puerperal sepsis. Eur J Epidemiol. 2022 May;37(5):437-445. doi: 10.1007/s10654-022-00871-8.

13. Antinori A, Mazzotta V, Vita S, Carletti F, Tacconi D, Lapini LE, D'Abramo A, Cicalini S, Lapa D, Pittalis S, Puro V, Rivano Capparuccia M, Giombini E, Gruber CEM, Garbuglia AR, Marani A, Vairo F, Girardi E, Vaia F, Nicastri E; INMI Monkeypox Group. Epidemiological, clinical and virological characteristics of four cases of monkeypox support transmission through sexual contact, Italy, May 2022. Euro Surveill. 2022 Jun;27(22):2200421. doi: 10.2807/1560-7917.ES.2022.27.22.2200421.

14. Browne E. Men Who Died From Monkeypox Brain Swelling Were Healthy Before Virus Hit. Newsweek 2022 Jul 3 https://www.newsweek.com/monkeypox-deaths-spain-healthy-risk-factors-1730385

15. Tin, A Illinois, Maryland, New York and Nevada announce monkeypox deaths as official CDC tally rises to 6. CBS News. 2022 Oct 22. https://www.cbsnews.com/news/monkeypox-deaths-illinois-maryland-new-york-nevada/

16. Abkouh DT, Tusha J, Khanam V, Giri P, Kumar S. Sudden cardiac death and the peculiar COVID-19 case. Chest. 2020;158(4):A271. doi: 10.1016/j.chest.2020.08.272

17. Barr J, Schalick WO, Shortell, CK. Surgeons in the time of the plague: Guy de Chauliac in fourteenth-century France. J Vasc Surg Cases Innov Tech 2020;6:657-658.

18. Isozaki S, Kakimoto Y, Ikeda H, Matsushima Y, Tsuboi A, Osawa M. Unexpected sudden death on arrival in a healthy middle-aged man associated with COVID-19-related diffuse cardiac injury: A case report. Heliyon. 2023 Dec 10;10(1):e23460. doi: 10.1016/j.heliyon.2023.e23460.

19. La Scola B, Audic S, Robert C, Jungang L, de Lamballerie X, Drancourt M, Birtles R, Claverie JM, Raoult D. A giant virus in amoebae. Science. 2003 Mar 28;299(5615):2033. doi: 10.1126/science.1081867.

20. Moreira D, López-García P. Ten reasons to exclude viruses from the tree of life. Nat Rev Microbiol. 2009;7(4):306-11. doi: 10.1038/nrmicro2108.

21. Raoult D. There is no such thing as a tree of life (and of course viruses are out!). Nat Rev Microbiol 2009;7:615. https://doi.org/10.1038/nrmicro2108-c6

22. Navas-Castillo J. Six comments on the ten reasons for the demotion of viruses. Nat Rev Microbiol. 2009;7(8):615; author reply 615. doi: 10.1038/nrmicro2108-c2.

23. Claverie, JM., Ogata, H. Ten good reasons not to exclude viruses from the evolutionary picture. Nat Rev Microbiol 2009;7:615. https://doi.org/10.1038/nrmicro2108-c3

24. Brüssow H. The not so universal tree of life or the place of viruses in the living world. Philos Trans R Soc Lond B Biol Sci. 2009 Aug 12;364(1527):2263-74. doi: 10.1098/rstb.2009.0036.

25. Koonin, E., Senkevich, T. & Dolja, V. Compelling reasons why viruses are relevant for the origin of cells. Nat Rev Microbiol 2009;7:615. https://doi.org/10.1038/nrmicro2108-c5

26. Colson, P., La Scola, B., Levasseur, A. et al. Mimivirus: leading the way in the discovery of giant viruses of amoebae. Nat Rev Microbiol 2017;15:243–254. https://doi.org/10.1038/nrmicro.2016.197

27. Harris HMB, Hill C. A Place for Viruses on the Tree of Life. Front Microbiol. 2021;11:604048. doi: 10.3389/fmicb.2020.604048.

28. Volland JM, Gonzalez-Rizzo S, Gros O, Tyml T, Ivanova N, Schulz F, Goudeau D, Elisabeth NH, Nath N, Udwary D, Malmstrom RR, Guidi-Rontani C, Bolte-Kluge S, Davies KM, Jean MR, Mansot JL, Mouncey NJ, Angert ER, Woyke T, Date SV. A centimeter-long bacterium with DNA contained in metabolically active, membrane-bound organelles. Science. 2022 Jun 24;376(6600):1453-1458. doi: 10.1126/science.abb3634.

29. Darwin, C.R. 1859. On the origin of species by means of natural selection, 1st ed. London: John Murray.

30. Foege WH, Millar JD, Henderson DA. Smallpox eradication in West and Central Africa. Bull World Health Organ. 1975;52:209-22.

31. Breman JG, Kalisa-Ruti, Steniowski MV, Zanotto E, Gromyko AI, Arita I. Human monkeypox, 1970-79. Bull World Health Organ. 1980;58:165-82.

32. Long B, Liang SY, Carius BM, Chavez S, Gottlieb M, Koyfman A, Brady WJ. Mimics of Monkeypox: Considerations for the emergency medicine clinician. Am J Emerg Med. 2023 Mar;65:172-178. doi: 10.1016/j.ajem.2023.01.007.

33. Ola P. The Law of the Critical Illness and Its Far-reaching Implications. OSF Preprints. 2023. Nov 16. doi:10.31219/osf.io/69v3m.

34. Bussani R, Zentilin L, Correa R, Colliva A, Silvestri F, Zacchigna S, Collesi C, Giacca M. Persistent SARS-CoV-2 infection in patients seemingly recovered from COVID-19. J Pathol. 2023 Mar;259(3):254-263. doi: 10.1002/path.6035.

35. Ola P. The quantifiable prediction of the disappearance of symptoms that worsen persistent illnesses towards disability on the path to biology-based diagnoses. OSF Preprints. 2024. July 30. doi:10.31219/osf.io/8d7t2.

36. Ritter WS, Stone MJ, Willerson JT. Reduction in exertional myoglobinemia after physical conditioning. Arch Intern Med. 1979 Jun;139(6):644-7. ; 139(6):644-647. doi: 10.1001/archinte.1979.03630430026010

37. Du M, Liu M, Niu B, Liu J. The global alarm bell is ringing due to the threat of potential severe cases and deaths caused by clade I of monkeypox virus. Lancet Infect Dis. 2024 Oct;24(10):e616-e617. doi: 10.1016/S1473-3099(24)00530-9. .