The Functional Communicativity of COVID-19 An Astro-Socio-Biological Multi-Level Informatic Analysis

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August 7, 2020

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Abstract

A multi-scale entropic analysis of COVID-19 is developed on the micro-biological, meso-social, & macro-astrological levels to model the accumulation of errors during processes of self-replication within immune response, communicative functionality within social mitigation strategies, and mutation/genesis within radiation conditioning from solar-cosmic cycles.

1 Multi-Scale Integration: The Informatic Thermodynamics of Functional Communicativity

To enable population-wide compliance with public-safety health measures, the cause of the high-risk behaviors associated with breaking quarantine must be identified and treated systemically. This approach thus conceives of infection as having a pathogenesis from the behaviors that induce it, which is itself caused by the cognitive-dysfunctional residuals to the malfunctioning of an underlying social-systemic process. City and State variation in infection rate-curves (i.e. time-based differential distributions) can be explained by the presence or absence (and degree) of different high risk behaviors and there may be learning

opportunities from successful cities or states. These differences between platial socialities may be classified according to the $functional - communicativity$ of the underlying communication system, a complex variable ($\lambda = \sigma + \omega i$), which may be measured inversely by a real measure of the economic dysfunctionality (i.e. 'shut-down') and an imaginary measure of the communal miscommunication (i.e 'social-distancing'). The present crisis derives from the divergence between the policy of the required λ and the actual sampled $\hat{\lambda}$.

The communicable SARS-COV-2 viral spread within different localities should be analyzed as a message within a communication system, mutating based upon the properties of the local communication system that propagates it. The two properties of a communication state-system can be expressed as a complex number (λ), with a real component as the total functionality (σ) of the communication system that propagates it and an imaginary component of the communicability (ω) of the lifeworld within which it is embedded, i.e. $\lambda = \sigma + \omega i$. This analytical format for any particular state or place of analysis encodes thus the percent of a shut-down (i.e. dysfunctionality) and the social-distancing policy in place (i.e. non-communicability), both of which can be calculated as a combination of the policy and the percent-compliance. While a communication system may be thus represented as a complex-variable system with the real system functionality and an imaginary lifeworld communicativity, any of its sub-systems or states, i.e. at lower levels of analysis, can be represented by a complex number. Our public health response system must thus seek to reduce λ^2 at all levels of analysis and components of interaction.

The genesis of SARS-CoV-2, with its internal code of a precise self-check mechanism on reducing errors in RNA replication and external attributes of ACE2 binding proteins, is an entropy-minimizing solution to the highly functionally communicative interconnected human societies embedded within high-entropic geophysical conditions of higher cosmic radiation atmospheric penetration with radioactive C-14 residues due to the present solar-cycle modulation. This background condition explains the mutation differences between SARS-1 & SARS-2, where the latter has a more persistent environment of C-14 to evolve steadily into stable forms. The counter-measures against the spread of the virus, either as therapeutics, vaccines, or social mitigation strategies, are thus disruptions (entropy-inducing) to these evolved entropy-reducing mechanisms within the intra-host replication and inter-host communicability processes.

The point of origin for understanding the spread of the virus in a society or subdivision is through its communicative functionality, which may be expressed as a complex variable of the real functionality of the social system and the imaginary communicativity of its lifeworld, the two attributes which are diminished by the shut-down and social distance measures. Conditions of high communicativity, such as New York City, will induce mutations with greater ACE2 binding proteins, i.e. communicability, as the virus adapts to its environment, while one of high functionality will induce error-minimization in replication. These two micro & meso scale processes of replication and communicability (i.e. intra- & inter- host propagation) can be viewed together from

the thermodynamic-informatic perspective of the viral RNA code *as a message* - refinement and transmission - itself initialized ('transcribed') by the macro conditions of the Earth's spatio-temporality (i.e. gravitational fluxuation). This *message* is induced, altered, amplified spatially, & temporallized by the entropic functional-communicative qualities of its environment that it essentially describes inversely.

2 Micro-Scale: Computational Biology of RNA Sequence

As with other viruses of its CoV virus family, the RNA of COVID-19 encodes a self-check on the duplication of its code by nuclei, thereby ensuring it is copied with little error. With little replication-error, the virus can be replicated many more rounds (exponential factor) without degeneration, which will ultimately stop the replication-process. Compare an example of $t = 3$ rounds for a normal virus with $t = 8$ for a Coronavirus under simple exponential replication viral count C by replication rounds t as $C(t) = e^{(t)}$: $C(3) = e^3 = 20.1$ vs. $C(7) = e^7 = 1096.6$.

Let us consider an example where a single RNA can create N-1 copies of itself before its code is degenerated beyond even replicative encoding, i.e. the binding segment code directing RNA replicase to replicate. The original RNA code is given by $\mathcal{N}_0$, with each subsequent code given by $\mathcal{N}_t$, where t is the number of times of replication. Thus, t counts the internal time of the "life" of the virus, as its number of times of self-replication. The relevant length of a sequence can be given as the number of base-pairs that will be replicated in the next round of replication. This will be expressed as the zero-order distance metric, $\mu^0(\mathcal{N}_t) = |\mathcal{N}_t|$.

The errors in the replicative process at time t will be given by $DISCR\_ERR(\mathcal{N}_t)$, for "discrete error", and will be a function of t, given as thus $\epsilon(t)$. Cleary, $|\mathcal{N}_t| = |\mathcal{N}_{t+1}| + \epsilon(t)$. In all likelihood, $\epsilon(t)$ is a decreasing function since with each round of replication the errors will decrease the number of copiable base-pairs, and yet with an exceptionally random alteration of a stable insertion, the error could technically be negative. There are two types of these zero-order errors, ϵ^-, as the number of pre-programmed deletions occurring due to the need for a "zero-length" sequence segment to which the RNA polymerase binds and is thereby directed to replicate "what is to its right in the given reading orientation", and ϵ^+ as the non-determined erroneous alterations, either as deletions, changes, or insertions. The total number of errors at any single time will be their sum, as thus $\epsilon(t) = \epsilon^-(t) + \epsilon^+(t)$. A more useful error-metric may be the proportional error, $PROP\_ERR(\mathcal{N}_t)$, since it is likely to be approximately constant across time, which will be given by the time-function $\epsilon'(t)$, and can similarly be broken into determined(-) and non-determined(+) errors as $\epsilon'(t) = \epsilon'^-(t) + \epsilon'^+(t)$. Expressed thus in proportion to the (zero-order) length of the RNA sequence, $\epsilon'(t) = 1 - \frac{|\mathcal{N}_{t+1}|}{|\mathcal{N}_t|} = \frac{\epsilon(t)}{|\mathcal{N}_t|}$.

The "length" (of internal time) of an RNA code, $\mathcal{N}_t$, in terms of the number of times it itself may be copied before it is degenerated beyond replication, is given as the first order "distance" metric $\mu^1(\mathcal{N}_t) = N(\mathcal{N}_t)$. For our generalized example, $\mu^1(\mathcal{N}_0) = N(\mathcal{N}_0) = N$. This may be expressed as the sum of all errors $N = \sum_{t=0}^{\infty} \epsilon(t) = \sum_{t=0}^{t_{max}} \epsilon(t) = \sum_{t=0}^{N} \epsilon(t)$.

We are interested in the "length" (of internal space) of the RNA code, second-order distance metric, $\mu^2(\mathcal{N}_0)$, as the number of copies it can make of itself, including the original copy in the counting and the children of all children viruses. This is the micro-factor of self-limitation of the virus, to be compared ultimately to the meso-factor of aerosolized half-life and the macro-factor of survival on surfaces.

These errors in replication, compounded by radiation exposure in the atmosphere, will add up to mutations of the virus, which by natural selection in the corporeal, social-communicative, and environmental (i.e. surfaces and aerosolized forms) levels has produced stable new forms of the virus.

Comparing SARS-1 & SARS-2, the former had a higher mortality rate and the latter has a higher transmission rate. There is certainly an inverse relationship between mortality and transmission on the meso-level as fatality prevents transmission, but there may also be inherent differences at the micro-level in methods of replication leading to these different outcomes. Mortality is due to intra-host replication exponentiation - whereby there are so many copies made that the containing cells burst - while communicability is due to the inter-host stability of the RNA code in the air and organic surfaces where it is subject to organic reactions and cosmic radiation.

3 Meso-Scale: The Communicative Epidemiology of Viral Social Reproduction

We can apply the theory of communicativity to studying the natural pathology (disease) and the social pathology (violence) of human society through René Girard's Theory of Mimetics[1]. Viewing a virus and a dysfunctional social system under a single conceptual unity (Mimetics) of a communicative pathology, the former 'spreads' by communication while the latter is the system of communication. Yet, different types of communication systems can lead to higher outbreaks for a communicable disease. Thus, the system of communication is the condition for the health outcomes of communicable disease. Beyond merely 'viruses,' a dysfunctional communication system unable to coordinate actions to distribute resources effectively within a population can cause other pathologies such as violence and poverty. From this integrated perspective, these 'social problems' may themselves be viewed as communicable disease in the sense of being caused, rather than 'spread,' by faulty systems of communication. Since violence and poverty are themselves health concerns in themselves, such a re-categorization is certainly permittable. The difference in these communicable

[1] **VIOL-SAC**.

diseases of micro and macro levels is that a virus is a replication script read and enacted by human polymerase in a cell's biology while a dysfunctional social system is a replication script read and enacted by human officials in a society. We can also thereby view *health* in the more generalized political-economy lens as the quantity of life a person has, beyond merely the isolated corporeal body but also including the action-potentialities of the person as the security from harm and capacity to use resources (i.e. via money) for one's own survival. It is clear that 'money' *should* be the metric of this 'bio-quantification' in the sense that someone with more money can create healthier conditions for life and even seek better treatment, and similarly a sick person (i.e. deprived of life) should be given more social resources (i.e. money) to reduce the harm. Yet, the economic system fails to accurately price and distribute life-resources due to its nodal premise prescribed by *capitalism* whereby individuals, and by extension their property resources, are not *social* (as in distributively shared), but rather isolated & alienated for individual private consumption.

This critique of capitalism was first made by Karl Marx in advocation for socialism as an ontological critique of the lack of recognition of the social being to human existence in the emerging economic sciences of liberalism. In the 17th century, Locke conceived of the public good as based upon an individual rights to freedom, thereby endowing the alienated (i.e. private) nature with the economic right to life. This moral reasoning was based on the theological premise that the capacity for reason was not a public-communicative process, but rather a private faculty based only upon an individual's relationship with God. Today we may understand Marx's critique of Lockean liberalism from the deep ecology perspective that sociality is an ontological premise to biological analysis due to both the relationship of an organism grouping to its environment and the in-group self-coordinating mechanism with its own type. Both of these aspects of a biological group, in-group relationships ($H^+(G) : G \to G$) and out-group relationships ($H^-(G) = \{H^-_-(G) : G^c \to G, H^-_+(G) : G \to G^c\}$) may be viewed as communicative properties of the group, as in how the group communicates with itself and with not-itself. In the human-capital model of economic liberalism, the group is reduced to the individual economic agent that must act alone, i.e. an interconnected system of capabilities, creating thereby an enormous complexity in any biological modeling from micro-economic premises to macro-economic outcomes. If instead we permit different levels of group analysis, where it is assumed a group distributes resources within itself, with the particular rules of group-distribution (i.e. its social system) requiring an analysis of the group at a deeper level that decomposes the group into smaller individual parts, such a multi-level model has a manageable complexity. The purpose is therefore to study Communicativity as a property of Group Action.

A group is a system of action coordination functionally interconnecting sub-groups. Each group must *act as a whole* in that the inverse branching process of coordination adds up all actions towards the fulfillment of a single highest good, the supreme value-orientation. Therefore, the representation of a group is by a tree, whose nodes are the coordination actions (intermediate groupings), edges the value produced, and leaves the elemental sub-groups *at the level of analysis.*

The total society can be represented as a class system hierarchy of group orderings, with primary groups of individuals. The distribution of resources between a group follows the branching orientation (σ^-) from root to leaves as resources are divided up, while the coordination follows the inverse orientation (σ^+) from leaves to root as elemental resources are coordinated in production to produce an aggregate good.

In the parasite-stress theory of sociality[2], in-group assortative sociality arose due to the stress of parasites in order to prevent contagion. There is thus a causal equivalence between the viral scripts of replication and the social structures selected for by the virus as the optimal strategy of survival. Violence too has the same selection-capacity since existentially conflicting groups are forced to isolate to avoid the war of revenge cycles. This process is the same as the spread of communicable diseases between groups - even after supposed containment of a virus, movement of people between groups can cause additional cycles of resurgence.

Racism is an example of non-effective extrapolation of in-grouping based on non-essential categories. As a highly contagious and deadly disease, on the macro-social level COVID-19 selects for non-racist societies via natural selection since racist societies spend too many resources to organize in-group social structure along non-essential characteristics, as race, and thus have few reserves left to reorganize along the essential criteria selected for by the disease (i.e. segregating those at-risk). Additionally, racism prevents resource sharing between the dominant group and the racially marginalized or oppressed group, and thus limits the transfer of scientific knowledge in addition to other social-cultural resources since what the marginalized group knows to be true is ignored.

With a complex systems approach to studying the communicability of the virus between groups (i.e. different levels of analysis) we can analyze the transmission between both persons and segregated groups (i.e. cities or states) to evaluate both social distancing and shut-down policies. A single mitigation strategy can be represented as the complex number $\lambda = \sigma + \omega i$, where σ is the dysfunctionality of the social system (percent shut-down) and ω is the periodicity of the shut-down. We can include s_d for social distance as a proportion of the natural radii given by the social density. The critical issue now is mistimed reopening policies, whereby physical communication (i.e. travel) between peaking and recovering groups may cause resurgences of the virus, which can be complicated by reactivation post immunity and the threat of mutations producing strands resistant to future vaccines. This model thus considers the long-term perspective of social equilibrium solutions as mixed strategies between socialism and capitalism (i.e. social distancing and systemic shut-downs) to coronaviruses as a semi-permanent condition to the ecology of our time.

[2] **fincher_thornhill_2012**.

4 Macro-Scale: Astro-biological Genesis of COR-VIR by Solar Cycles

The genesis of COR-VIR are by mutations (and likely reassortment) induced by a burst of solar flare radiation and a conditioning by cosmic radiation, each with different effects on the viral composition. Comparison with SARS-1 (outbreak immediately after a solar maximum) reveals that solar radiation (i.e. UVC) from flares & CMEs, more frequent and with higher intensity during solar maximums yet also present during minima, is responsible for the intensity (mortality rate) of the virus, while cosmic radiation, enabled by the lower count of sun spots that decreases the Ozone in the atmosphere normally shielding the Earth's surface from radiation, gives the virus a longer duration within and on organic matter (SARS-2), likely through mutation by radioactive C-14 created by cosmic radiation interaction with atmospheric Nitrogen, The increased organic surface radioactivity is compounded by the ozone-reduction due to N^2 emissions concurrent with "Global Warming". The recent appearance of all coronaviruses in the last 5 solar cycles is likely due to a global minimum within a hypothetical longer cosmic-solar cycle ($\sim$ 25 solar cycles) that modulates the relative sun cycle sunspot count, and has been linked to historical pandemics. A meta-analysis has detected such a frequency the last milenia with global pandemics[3]. The present sun cycle, 25, beginning with a minimum coincident with the first SARS-2 case of COVID-19, has the lowest sunspot count in recorded history (i.e. double or triple minimum). Likely, this explains the genesis of difference in duration and intensity between SARS-1 & SARS-2.

This longer solar-cosmic cycle that modulates the relative sunspot count of a solar cycle, the midpoint of which is associated with global pandemics, has recently been measured to 208 years by C-14 time-cycle-analysis, which is itself modulated by a 2,300 year cycle. These time-cycles accord to the (perhaps time-varying) Mayan Round calendar: 1 K'atun=2 solar cycles ($\sim$20 years); 1 May = 13 K'atun ($\sim$ 256 years); 1 B'ak'tun = 20 K'atun ($\sim$394 years) ; 1 Great Cycle = 13 B'ak'tun ($\sim$ 5,125 year). Thus, the 208-year cycle is between 1/2 B'ak'tun ($\sim$197 years) and 1 May ($\sim$ 256 years, 13 K'atuns). It is likely the length of 25 sun cycles, the same as the May cycle, yet has decreased in length the last few thousand years (perhaps as well with sun-spot counts). The 2,300 year cycle is $\sim$ 6 B'ak'tuns (2,365 years), constituting almost half of a Great Cycle (13 B'ak'tuns). We are likely at a triple minimum in sunspot count from all 3 solar-cosmic cycles, at the start of the first K'atun (2020) of the beginning of a new Great Cycle (2012), falling in the middle of the May (associated with crises).

The entropic characterization of the pathogenesis as prolonged radioactivity - low entropic conditioning of high entropy - leads to the property of high durability on organic matter and stable mutations.

[3] **2017JAsBO...5..159W**.