

Curvature in infectious disease network: A metric for vaccination and control

Sara Najem

*Physics Department, American University of Beirut, Beirut, Lebanon
and Complexity and Network Science Cluster at the Center for Advanced Mathematical Sciences,
American University of Beirut, Beirut, Lebanon*

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We investigate the geometric structure of evolving infectious-disease networks using a notion of node curvature, which is based on the heat-kernel expansion that naturally extends to edges. Applying this measure to networks reconstructed from infection counts whose degree and strength distributions transition toward a steady power-law, we observe a corresponding transition in curvature. We relate the latter to an underlying hyperbolic space and reveal a phase transition in the network's effective temperature. We further compare our curvature measure with existing definitions in the literature, demonstrating that all capture the transition consistently. Our results suggest that curvature provides a metric for characterizing structural changes in dynamic networks arising from infectious processes and identifies distinct phases where targeted vaccination strategies may be most effective, when used within a multimetric public health decision-making framework.

DOI: [10.1103/c44l-89p7](https://doi.org/10.1103/c44l-89p7)**I. INTRODUCTION**

Physical networks live in finite metric spaces, where distances are defined as geodesics between nodes. Recently, other types of distance associated with network geometries have gained attention, particularly that of the geometry of a network's underlying latent space and that of the effective geometry dictated by the dynamical processes over the network [1]. Effective geometry, which is revealed by analyzing the dynamics over networks, provided a lens through which patterns can be revealed and predicted. For example, the evolution of an infectious disease, which uses probabilistic data on traffic to model the effective distance between locations rather than the geographic ones, allows to predict a wave-like pattern with a center of infection and an evolving front that reaches effective neighbors in deterministic arrival times [2–4]. The latent geometry was also put forth by physicists as a model that can explain a number of features of real complex networks. The model proposes that these networks emerge from a hyperbolic geometric space represented by the Poincaré circle. In this representation, a node's distance from the center is indicative of its centrality, and the angular displacement between nodes is representative of their topological closeness or similarity.

Hyperbolic geometry of latent metric spaces of networks has been shown to explain the emergence of a wide variety of topological measures including power-law degree distributions, community structure, high clustering, and low average path lengths, among others [5–10]. This introduced a wave of interest in discrete differential geometry, where curvature

remained a central topic, as is the case for the continuous differential geometry counterpart. Applications include the quantification of impact of surgery on the brain through hyperbolic embedding [11], host pathogen interactome embedding [12], stock market stability and volatility [13], and lately coupling to machine learning to infer coordinates in the hyperbolic circle [14]. Therefore, notions from the works of Gauss and Descartes made their way to the discrete context such as the discrete Laplace operator (Laplace-Beltrami) [15], Ricci curvature [16], and more recently, the Dirac operator and its relation to the Laplace operator and incidence matrices [17]. Networks, being discrete structures, were recently shown to be associated with curvatures such as the Olivier-Ricci and Forman-Ricci resistance curvatures [18,19]. These were introduced as measures of vulnerability [20], risk and fragility [21,22], robustness [23], structural connectivity [24], and communicability [25]. These approaches to network geometry are providing ways of understanding complex behavior over networks as well as multiple scale self-organizations and the associated information exchange. In this work, we use the notion of node curvature introduced in Ref. [26] that relies on the heat kernel, which generalizes to edge curvature. We compute it for a series of networks recovered from infection counts of an evolving infectious disease. These networks' degree- and strength distributions were shown to exhibit a transition to a steady power-law, which we relate to the curvature of an underlying hyperbolic space, and subsequently capture a phase transition in effective temperature of the network. We compare our measure of curvature with the ones used in the literature and show that they all capture the transition.

While the heat-kernel curvature was introduced in Ref. [26] and the network reconstruction methodology in Ref. [27], the present work provides three contributions: (1) the first application of heat-kernel curvature to identify dynamic structural transitions in evolving networks, (2) a comparative

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analysis demonstrating that different curvature measures capture distinct geometric features of the same network evolution, and (3) a direct connection between heat-kernel curvature and the hyperbolic embedding of the infection network, revealing a transition in effective temperature that is not accessible through local curvature measures alone.

II. METHODS

We start by describing the network recovery and the data used in the rest of the paper. The anonymized COVID-19 counts were provided by the Surveillance Unit of the Ministry of Public Health (MoPH) in Lebanon. Data entries included the locality, or the administrative area of the infected individual, and the time of reporting. There are 1544 localities in Lebanon and the data reporting spanned 860 days starting February 2020. This period spanned distinct phases of the pandemic in Lebanon, with a critical juncture occurring on August 4, 2020, coinciding with the Beirut port explosion. Prior to it, initial strict lockdown and airport closure were implemented, whereas in the aftermath, social distancing and mask wearing were compromised amid waves of protests. Thereafter, throughout the remainder of the period, sporadic targeted restrictions were imposed with brief full lockdowns lasting two to three weeks at a time. From this an autoregressive model of infection counts was built in a previous work [27], where the daily count of infections Y_{it} in locality i on day t is modeled as conditionally independent negative binomial random variables with mean function μ_{it} decomposed into three components,

$$\mu_{it} = e_{it} v_{it} + \lambda_{it} Y_{i,t-1} + \phi_{it} \sum_{j \neq i} \omega_{ji} Y_{j,t-1}, \quad (1)$$

where Y_{it} is the infection count at locality i at time t , and $\mu_{it} = \mathbb{E}(Y_{it} | Y_{j,t-1}, e_i)$. The first term in Eq. (1) accounts for nonlocal cases introduced to the country through ports of entry, mainly the airport. The infected individual is counted as a new case in his or her corresponding destination locality i . It assumed proportionality to the population size e_i . The second term is the autoregressive effect, which accounts for branching of infections within the locality due to a local encounter. Its effect is to multiply the number of cases at the previous time within the locality. The last is the “spatiotemporal” component capturing transmission from other localities, with $\omega_{ij} \propto d_{ij}^{-2}$ modeling distance-dependent mobility as a power law [28,29]. We followed the framework of Ref. [30] and its implementation in the R package SURVEILLANCE [31], where the log-transforms of the λ_{it} , ϕ_{it} , and v_{it} coefficients are modeled as linear functions of time: $\log(\lambda_{it}) = \alpha_i^{(\lambda)} + \beta_i^{(\lambda)} t$, $\log(\phi_{it}) = \alpha_i^{(\phi)} + \beta_i^{(\phi)} t$, and $\log(v_{it}) = \alpha_i^{(v)} + \beta_i^{(v)} t$, where α_i and β_i are locality-specific intercepts and slopes. This formulation allows each locality to have its own baseline transmission intensity and its own trend over time, capturing heterogeneity in how the epidemic evolved across different regions.

For each time interval $[0, \text{day}]$ (where day increases in 20-day increments), the parameters α_i and β_i for all three coefficients are estimated by maximizing the log-likelihood of the observed counts conditional on past observations, using numerical methods implemented in the surveillance package.

Once the α_i and β_i are estimated, the time-varying coefficients at any *day* are recovered by exponentiating the linear predictors: $\hat{\lambda}_{it} = \exp(\hat{\alpha}_i^{(\lambda)} + \hat{\beta}_i^{(\lambda)} t)$, $\hat{\phi}_{it} = \exp(\hat{\alpha}_i^{(\phi)} + \hat{\beta}_i^{(\phi)} t)$, and $\hat{v}_{it} = \exp(\hat{\alpha}_i^{(v)} + \hat{\beta}_i^{(v)} t)$. Details on the model parameters can be found in Ref. [27]. The main observation made in Ref. [27], which we rely on in this work, is that the last term of Eq. (1) is a time-evolving network:

$$A_{i,j}(\text{day}) = \phi_i(\text{day}) \cdot w_{i,j}(\text{day}). \quad (2)$$

Specifically, the flux of mobile individuals into i from j is defined by ω_{ji} , which carries no temporal dependence. However, accounting for control measures and the subsequent attenuation of the flux between them is captured by the time-dependent ϕ_{it} , which is inferred from the data. Therefore, control measures are captured in their effect on infection counts, which themselves affect ϕ . Further, the multiplication of the ω_{ji} by ϕ_{it} is consequently a time-dependent vector, and when generalized over all localities, it is matrix which we defined to be A . It captures the infected fraction of mobile individuals between localities and thus controls the spread of the disease inside the country. $A_{i,j}(\text{day})$ are the elements of the interaction matrix between localities as a function of time. That is, the matrix A has a definitive role in the interlocalities dynamics, and thus any control measure has to account for the topology of A . The interlocality transmission network $A(\text{day})$ is then constructed directly from the estimated parameters by rewriting the second term in Eq. (1) as $\phi_{it} \sum_{j \neq i} \omega_{ij} Y_{j,t-1} = \sum_{j \neq i} A_{ij}(t) Y_{j,t-1}$. Thus, the weight of the directed edge from locality j to locality i at time t is the product of the mobility flow ω_{ij} (determined by geographic distance) and the time-varying ϕ_{it} of imported infections. Importantly, ϕ_{it} is locality specific and time dependent, meaning that even if two localities share the same proximity to a source, their edge weights may differ if their ϕ evolves differently over time. The estimation is performed sequentially over expanding time windows $[0, T]$ for $T = 20, 40, \dots, 820$ days, yielding a time-ordered sequence of networks $A(\text{day})$ that captures the evolving pattern of interlocality transmission and forms the basis for the topological analyses that follow.

The degree- and strength distributions of the A networks were shown to exhibit a transition to a power law of -2.5 at day 180 after the first reported case in Lebanon [27]. Our goal here is to characterize the transition to this power with curvature measures and subsequently relate it to an underlying hyperbolic geometry explaining the distribution.

For this purpose, we introduce the curvature recovered from the heat kernel given in Ref. [26].

Heat kernel

In the context of networks, the heat equation describes diffusion through the graph structure over time. Rather than spreading continuously through space, diffusion on a network occurs along the discrete connections between nodes. The solution to this equation is controlled by the kernel e^{-tL} , which is an $n \times n$ matrix that governs how an initial distribution diffuses through the network over time. L is the graph Laplacian defined below. The entry $(e^{-tL})_{ij}$ can be interpreted as the amount of heat transferred from node j to node i after time t , or equivalently, the probability that a random walk starting

at node j is found at node i at time t . On the other hand, in the continuum limit on manifolds, the heat kernel has been used for characterizing their geometries. In particular, the heat-kernel expansion, which is rooted in the spectral action principle, was introduced for curvature computation [32]. The coefficients of the expansion, in the vicinity of $t \rightarrow 0^+$, of the kernel K in powers of t , where t is time, reveal the scalar curvature of the manifold [33]. Here, we borrow this idea, introduced in Ref. [26], to calculate the coefficients of the expansion, which is now equivalent to solving the heat equation on the network, to reveal node and edge curvatures. It is essential to emphasize that this diffusion time t is independent of the time at which the network itself is inferred. t is the time of a hypothetical dynamical process unfolding on the network. It governs how far and how fast heat propagates through a structure that is treated as static over the diffusion interval. It probes the infinitesimal behavior of this process, extracting purely local geometric information from $A(\text{day})$, which refers to the real observational time during which the empirical data are collected and the transmission network is reconstructed. This network may itself evolve over real-world time due to changing conditions, interventions, or behavior. For that, we proceed with the definition of the operators needed for the computation. The graph Laplacian defined as $L = D - A$, where D is the degree and A is the adjacency, can alternatively be written as $L = BB^T$, where B is the incidence matrix; see below. The n th order Hodge Laplacian is represented by the matrix [17,34]

$$L_{[n]} = B_{[n]}^T B_{[n]} + B_{[n+1]} B_{[n+1]}^T = L_{[n]}^{\text{down}} + L_{[n]}^{\text{up}}, \quad (3)$$

where $B_{[n]}$ denotes the incidence between n and $n+1$ simplices. Put simply, if $n=0$, $B_{[0]}$ represents the oriented relation between nodes and edges. Similarly, $B_{[1]}$ represents the oriented edge-triangle relations. We consider the simplicial complex associated with the graph, where 0-simplex is a node (vertex) and 1-simplex is an edge (pair of connected nodes). Generally, $[B_{[n]}]_{\alpha',\alpha} = (-1)^p$ for every pair of n -dimensional simplex α and $(n-1)$ -dimensional simplex α' such that $\alpha = [v_0, v_1, \dots, v_n]$; $\alpha' = [v_0, v_1, \dots, v_{p-1}, v_{p+1}, \dots, v_n]$, where any complete subgraph on $n+1$ nodes can be viewed as an n -simplex [17,34–36].

For our case, $n=0, 1$ represent our network and we consider only $L_{[0]}$ and $L_{[1]}$. The super Laplacian L_s is defined as a block diagonal matrix composed of the Hodge Laplacians of all orders,

$$L_s = \begin{pmatrix} L_{[0]} & 0 \\ 0 & L_{[1]} \end{pmatrix}. \quad (4)$$

Now, using the $A(\text{day})$, which is the inferred adjacency matrix, we use the data from the start of the pandemic up to a given day to recover the incidence matrix $B_{[0]}$, which is what we need to compute the evolving $L_s(\text{day})$, using Eq. (3) to get $L_{[0]} = B_{[0]} B_{[0]}^T$ and $L_{[1]} = B_{[0]}^T B_{[0]}$, since our graph is not a higher-order network and we do not have triangular hyperedges or faces. In order to compute the node and edge curvatures, following the prescription in Ref. [26], we first compute the cumulative density of the eigenvalues of $L_{[0]}$ in the asymptotic limit [34], given by

$$\rho(\lambda)_{[0]} \propto \lambda^{d_s^{[0]}/2}, \quad \text{for } n=0,$$

where $d_s^{[0]}$ is the spectral dimension of the network's Laplacian $L_{[0]}$. Similarly, for $n=1$ the spectrum of $L_{[1]}$ is

$$\rho(\lambda)_{[1]} \propto \lambda^{d_s^{[0]}/2} + \lambda^{d_s^{[1]}/2},$$

Then, we proceed to compute the heat kernel. We start by diagonalizing $L_s = V^T \Sigma V$, where Σ is the diagonal matrix of the eigenvalues, and thus we can write

$$K(i, j, t) = e^{-tL_s/\Lambda^2} = V^T e^{-t\Sigma/\Lambda^2} V. \quad (5)$$

Given the block structure of L_s given in Eq. (4), $K(i, i, t)$ is defined on nodes and edges. We thus split it into $K_v(i, i, t)$ and $K_e(i, i, t)$, which in the limit $t \rightarrow 0^+$ is given by

$$K_v(i, i, t) \approx u_{0,v}(i, i) \left(\frac{1}{4\pi t} \right)^{d_s^{[0]}/2} + u_{1,v}(i, i) \left(\frac{1}{4\pi t} \right)^{d_s^{[0]}/2} t + \mathcal{O}(t^{2-d_s^{[0]}/2}). \quad (6)$$

Likewise,

$$K_e(i, i, t) \approx u_{0,e}(i, i) \left(\frac{1}{4\pi t} \right)^{d_s^{[1]}/2} + u_{1,e}(i, i) \left(\frac{1}{4\pi t} \right)^{d_s^{[1]}/2} t + \mathcal{O}(t^{2-d_s^{[1]}/2}). \quad (7)$$

Now, as in Refs. [32,33], the scalar curvature is related to the second coefficient in the expansion via

$$\frac{R_{v_i}}{6} = u_{1,v}(i, i), \quad (8)$$

where R_{v_i} is scalar curvature or, equivalently in this case, the i th node curvature. Similarly, $u_{1,e}(i, i)$ defines edge curvatures R_{e_i} . In order to recover these curvatures, we compute the corresponding $K = e^{-Lt}$ for each L for $t \in [\frac{1}{\lambda_{\max}}, t_{\max}]$ in the vicinity of $t \rightarrow 0^+$ and fit it against the terms of Eqs. (6) and (7); we truncate the power series at order $\mathcal{O}(t^{k-d_s^{[0]}/2})$, where $\lambda_{\max}$ is the largest eigenvalue of L whose inverse together with $t_{\max}$ define the interval over which the logarithm of the kernel scales linearly with t , and k is the max network degree. The matrix exponentiation was computed using EXPMGPU of the GPUMATRIX package in R. We then compare our results for node curvature with those recovered from Forman-Ricci and Olivier-Ricci using the GRAPHRICCICURVATURE package in python and resistance curvatures which we computed following Ref. [18]. It is worth noting that the day associated with the recovered A and subsequently L is different from the time that we use to fit for the coefficients. More specifically, the role of the heat kernel is the following: given a network, the heat equation is solved on the structure up to t the vicinity of $t \rightarrow 0^+$ in order to recover the networks' geometric features. This t is independent of the time at which the structure is inferred; it is the time of a dynamical process over a network, rather than the evolution time of the network itself.

Furthermore, given that the degree- and strength distribution followed a power law with an exponent γ , we used the relation between curvature and the former to recover an effective temperature of the network. The relation is given by $R = -\zeta^2$ and $\gamma = 1 - \frac{1}{\zeta^2}$ [5–8], where R is defined as the curvature of the hyperbolic space.

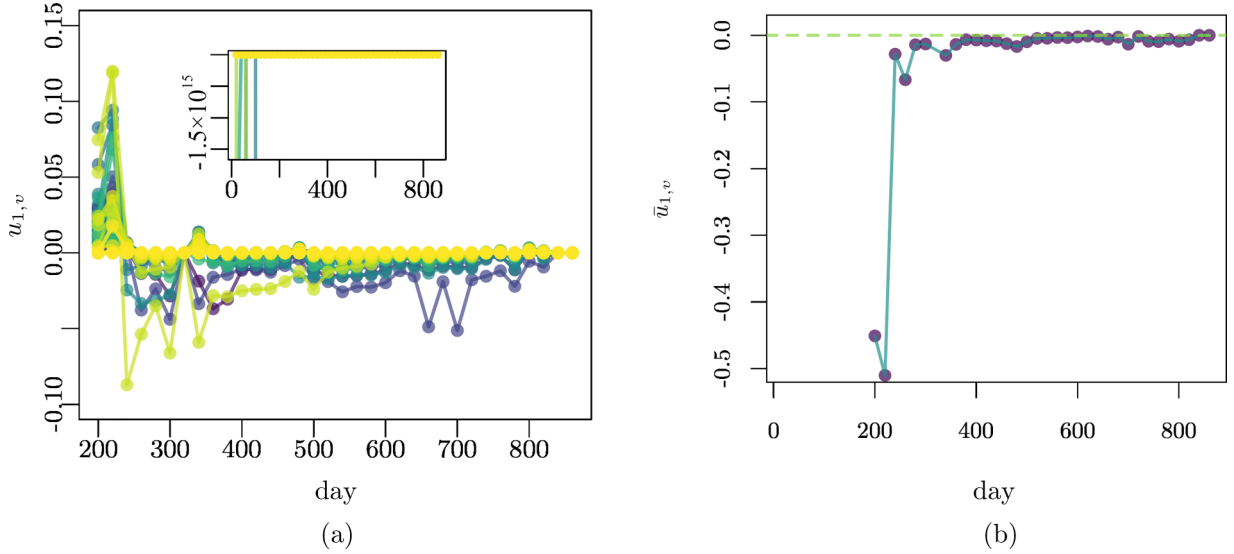


FIG. 1. (a) The evolution of the individual node curvature $u_{1,v}$ as a function of time. We note that before $t = 180$ the values of $u_{1,v}$ could not be displayed on the same plot due to their large negative values, which is shown in the inset. (b) The evolution the average node curvature $\bar{u}_{1,v}$ as a function of time. Again, below 180 the averages were negative large values of the order of -10^{15} .

III. RESULTS

Below, we show the results of the node curvatures $u_{1,v}$ recovered for the evolving $L_{[0]}$. The curvatures were highly negative before $t = 180$, which is the time associated with the transition to power law in Ref. [27]. The behavior is shown in Fig. 1(a), with the inset showing the behavior over the entire span of the disease. We followed the average node curvature and show its evolution in Fig. 1(b).

We denote by γ the value of the exponent recovered from the power law of the strength- and degree distribution in Ref. [27]. From the popularity similarity model, γ is given by

$$\gamma = 1 + \frac{1}{\zeta T}. \quad (9)$$

The relationship in Eq. (9), derived from the popularity-similarity model, establishes a direct link between the network's macroscopic structure (the exponent γ) and curvature. By substituting the expression for the average curvature, $R = -\zeta^2 = -6\bar{u}_{1,v}$, we can solve for the parameter ζ and, consequently, the “effective temperature” T associated with the network's evolution. This temperature is not a physical temperature but rather a parameter that controls the balance between popularity and similarity in the network's growth, influencing its geometry and the emergence of its scale-free structure. Using the empirically recovered values of γ from Ref. [27] and $\bar{u}_{1,v}$ from our heat-kernel analysis, we compute the evolution of T , which is shown in Fig. 2. The temperature exhibits a clear transition exactly at $t = 180$, the same time associated with the transition to a power law in the strength distribution, suggesting a fundamental shift in the organizing principles of the transmission network. The empirical behavior of T is well described by a smooth sigmoidal function, which captures the sharp but continuous transition from one

regime to another:

$$T \approx \frac{1}{2} \left[1 + \tanh \left(\frac{\text{day} - 242}{40} \right) \right]. \quad (10)$$

The hyperbolic tangent was chosen because it naturally models smooth, continuous transitions between two states and is commonly used to describe order parameters in phase transition phenomena. The parameters (shift of 242 days and width of 40 days) were estimated via nonlinear least-squares regression, yielding an excellent fit ($R^2 = 0.98$, RMSE = 0.05). We emphasize that Eq. (10) is an empirical description of the observed behavior for this specific dataset,

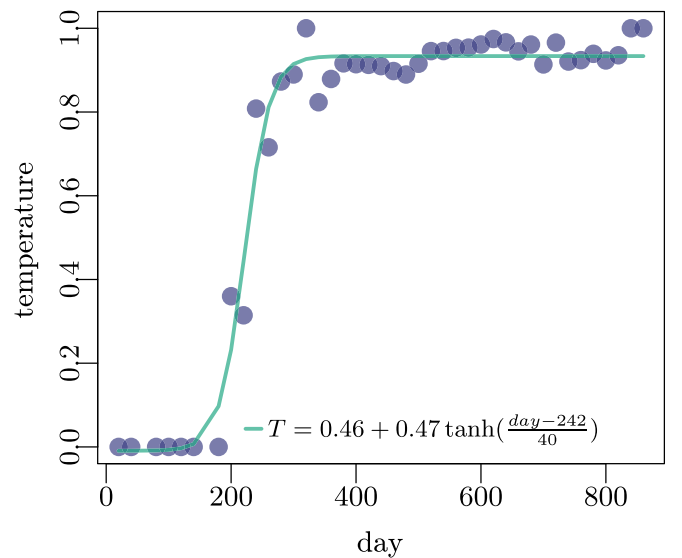


FIG. 2. The behavior of effective temperature. The fit given by Eq. (10) has a sum of squared errors (SSE) of 0.11, a total sum of squares (SST) of 5.31, a RMSE of 0.05, and R^2 of 0.98. The fitting was performed using nlsLM of minpack.lm in R.

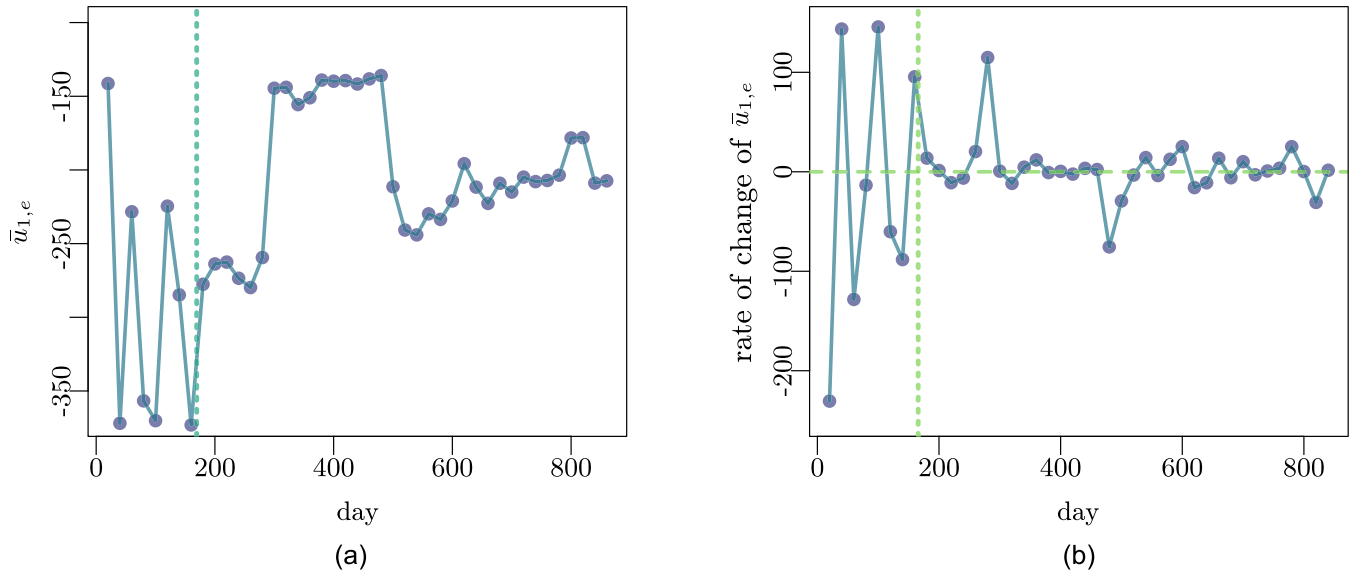


FIG. 3. (a) The evolution of the average edge curvature $u_{1,e}$. (b) The rate of change of the average edge curvature $u_{1,e}$ is followed as a function of time. We note that it fluctuates around zero after the 180th day of the disease.

not a claim of universality. It compactly captures the sharp but continuous nature of the transition around day 180 and provides a convenient parametric summary of the temperature evolution within the scope of this study. Whether similar sigmoidal transitions appear in other epidemiological or network contexts remains an interesting question for future investigation. We also followed the edge curvatures using Eq. (7); their average evolution $\bar{u}_{1,e}$ throughout the disease span is shown in Fig. 3(a). We opted to show the average edge curvature since the number of edges is very big and the visual clutter of the visualization makes it noninformative. We further look at the rate of change of $\bar{u}_{1,e}$ and observe that it fluctuates around zero at the onset of the transition at day 180.

Given the transition in curvature recovered using the heat kernel, we expected to detect a change in behavior of

curvature computed using resistance, Forman, and Olivier-Ricci curvatures. First, we start with the average node resistance curvature shown in Fig. 4(a). We observe a drop in its value with high fluctuations before day 180. Similar to what we did for the $\bar{u}_{1,e}$ we followed its rate of change and observed that it stabilized to almost zero also after the transition, which is shown in Fig. 4(b).

Further, we computed the average Forman and Olivier-Ricci edge curvatures shown respectively in Figs. 5(a) and 5(b). It is worth noting that the reference to simplicial complexes in our methodological discussion was meant to situate the approach within the broader mathematical framework of higher-order networks, but the actual computations presented in this work are confined to the 1-skeleton (graph) derived from the transmission networks. Therefore, the curvatures

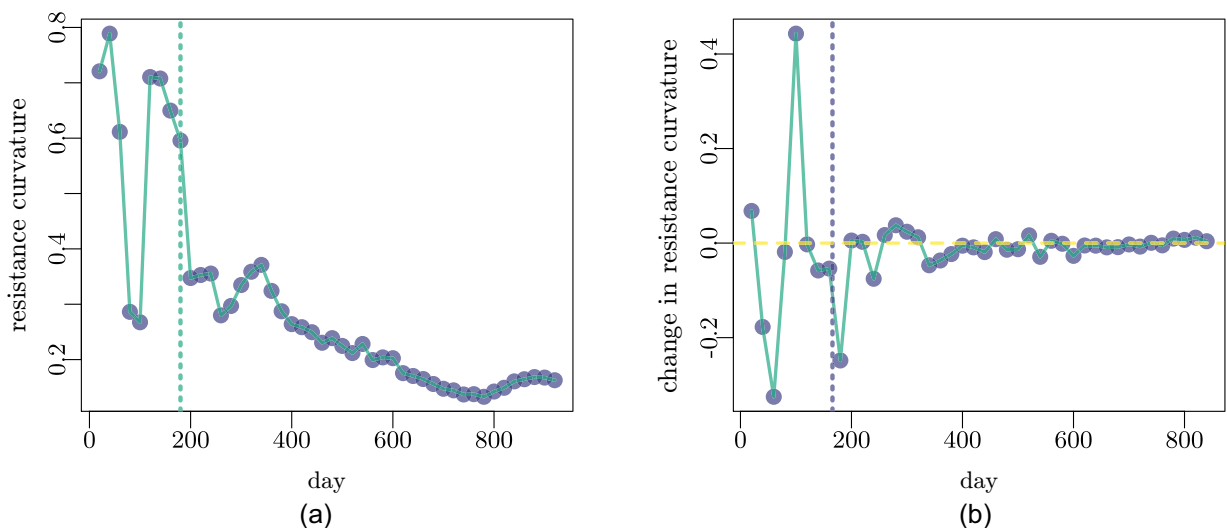


FIG. 4. (a) We show the evolution of the average node resistance curvature, which steadily drops after 180. (b) We follow the evolution of the rate of change of the average node resistance curvature, which stabilizes around zero after day 180 of the disease.

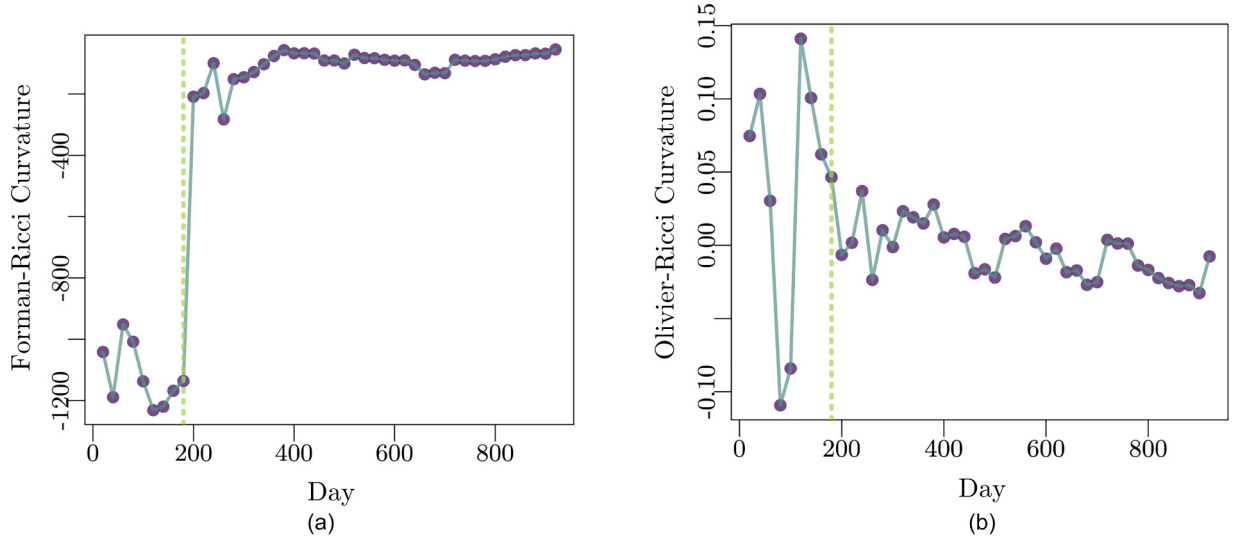


FIG. 5. (a) We show the evolution of the average Forman-Ricci curvature of the edges, which also stabilizes after 180. (b) We show the evolution of the average Olivier-Ricci curvature of the edges, which also stabilizes after 180.

computed are standard edge curvatures on graphs, as directly implemented in GRAPHRICCICURVATURE in python [37]. We did not compute curvatures for higher-dimensional faces (e.g., triangles or 2-simplices). Further, in our analysis we used unweighted graphs, so all node and edge weights default to 1. Subsequently, we computed their associated average node curvatures shown in Figs. 6(a) and 6(b), all of which exhibit a transition at day 180, which is in line with the behavior of the topological transition.

The above results, especially the transition in network temperature, suggest there is a corresponding change in the probability of edge formation [8], given explicitly by

$$p(x) = \frac{1}{1 + e^{\zeta(x - \mu_{cp})/2T}}, \quad (11)$$

where $\mu_{cp} = \bar{k}N/2$ is the chemical potential defined as the ratio of the product of the average degree $\bar{k}$ and the number of

nodes N to 2, and x is the hyperbolic distance between the two nodes in the Poincare disk. The connection probability follows the grand canonical Fermi-Dirac distribution. We show its evolution for the span of the disease in Fig. 7.

IV. DISCUSSION AND CONCLUSION

The computed negative nodes' curvatures and their average $\bar{u}_{1,v}$, observed before day 180 in both Figs. 3(a) and 3(b), are associated with the networks in a transient, far-from-equilibrium state, consistent with the early phase of the pandemic when the transmission network was still forming and connectivity patterns were rapidly evolving.

The transition to much smaller curvature values after day 180 signals a stabilization of the network geometry, which is in line with the emergence of scale-free structure reported in Ref. [27]. Also, the effective temperature T , shown

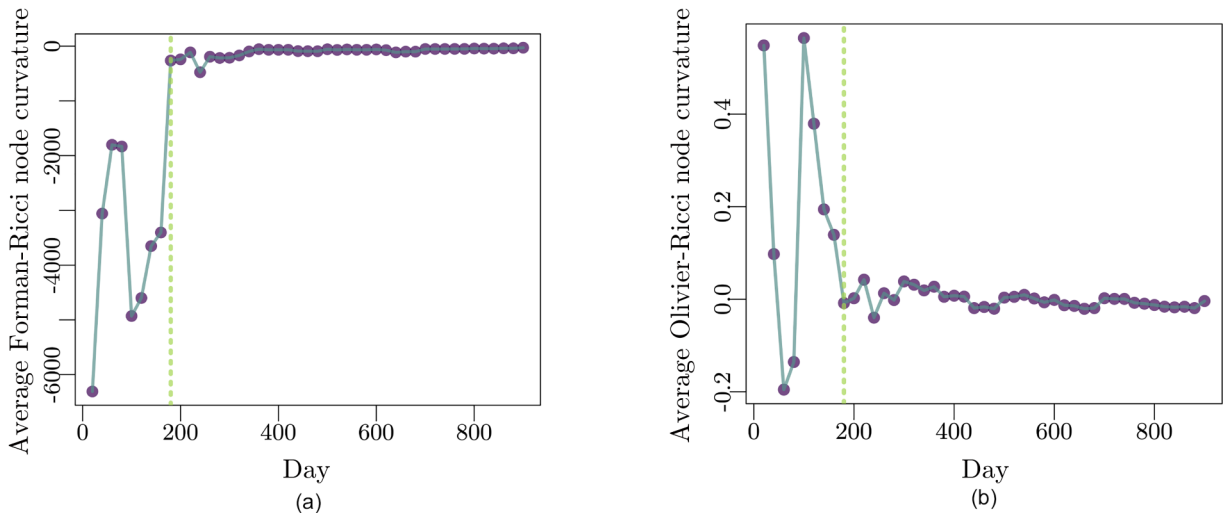


FIG. 6. (a) We follow the evolution of the average Forman-Ricci curvature of the nodes, which exhibits a clear transition at 180. (b) We follow the evolution of the average Olivier-Ricci curvature of the nodes, which stabilizes at zero after the transition at 180.

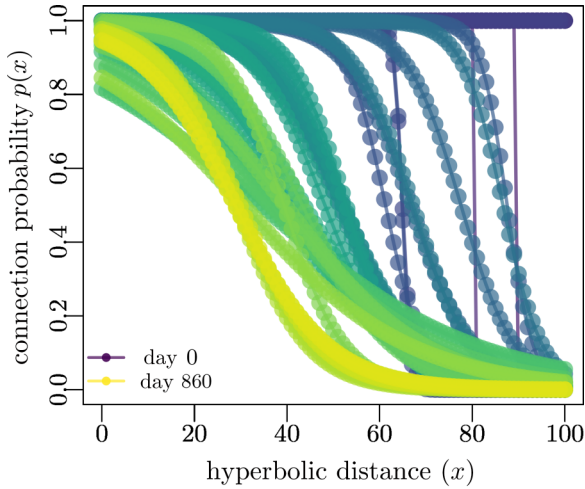


FIG. 7. The connection probability is followed as a function of the hyperbolic distance color-coded by day, with the upper and lower bound shown in the legend.

in Fig. 2, derived from the relationship $R = -\zeta^2 = -6\bar{u}_{1,v}$ and the power law exponent γ , provides a complementary perspective. The fit captures a continuous tanh transition which is characteristic of phase transitions in complex systems. It suggests that the network underwent a fundamental reorganization. Particularly, in the popularity-similarity model, temperature controls the balance between random and geometric orderly attachment. Thus, the observed increase in T indicates that the network evolved from a state where connections were strongly deterministic (low T , high ζ) to a state where randomness played a larger role in edge formation (higher T , lower ζ). This interpretation aligns with the observed stabilization of the network into a scale-free configuration: the network adopted more random attachment rules while simultaneously reorganizing its geometric embedding into a more clustered arrangement, as reflected in the drop in $p(x)$ for large distances in Fig. 7. The latter regime corresponds to a phase where the disease was widely circulating, with transmission patterns shaped by a combination of randomness and localized clustering, in contrast to the early phase where deterministic connections dominated.

Then, we further comment on the observed plateau in edge curvature between days 250 and 450 in Fig. 3(a), which could also reflect a period of network stabilization. This followed the disruptive early phase of the pandemic, during which no major external shocks, comparable to the initial lockdown, airport reopening, or Beirut port explosion, disrupted the network topology [27]. The consistent application of targeted lockdowns, the introduction of the Alpha variant, and the gradual rollout of vaccination contributed to a quasi-steady state in interlocality transmission patterns. The subsequent drop after day 450, followed by a shallow increase through day 800, coincides with the introduction of the Delta and Omicron variants. These variants, characterized by higher transmissibility, may have altered the spatial dynamics of the spread. This could have initially disrupted the established network structure, and hence the observed curvature drop. The latter is then

followed by an increased transmissibility characterized by the shallow increase in curvature. This pattern suggests that while new variants perturbed the network, the underlying geometry remained more resilient than during the initial transition, which reflects an accumulated level immunity and sustained public health measures. Forman-Ricci, Ollivier-Ricci, and resistance curvatures, shown in Figs. 5(a), 5(b), 6(a), and 6(b), all detected the transition at day 180 but did not exhibit a subsequent plateau behavior. The shallow increase after day 450, coinciding with the Delta and Omicron waves, suggests that subsequent variant-driven perturbations were sufficient to alter diffusion geometry but insufficient to fully revert the network to its pretransition configuration.

The plateau observed in the heat-kernel edge curvature during days 250–450, which is not captured by Forman-Ricci, Ollivier-Ricci, or resistance curvatures, warrants a mechanistic explanation that highlights the distinct geometric information each measure encodes. This differential sensitivity arises from fundamental differences in what each curvature probes. Forman-Ricci and Ollivier-Ricci curvatures are inherently local measures: the former depends only on edge multiplicity and the degrees of adjacent nodes, while the latter relies on optimal transport distances between the local neighborhoods of edge endpoints. Consequently, these measures saturate when the local connectivity structure stabilizes, which occurred during the period of sustained public health measures and relatively stable transmission patterns. Resistance curvature, on the other hand, is derived from global effective resistance distances, making it sensitive to large-scale connectivity but dominated by the overall network topology rather than its fine-grained geometric texture. In contrast, the heat-kernel curvature coefficient $u_{1,e}$ is obtained from the short-time asymptotic expansion of the heat kernel, which encodes the full Laplacian spectrum. More formally, for a graph Laplacian L with eigenvalues λ and corresponding eigenfunctions ψ_λ , the heat kernel is given by $K(t) = \sum_\lambda e^{-\lambda t} \psi_\lambda \psi_\lambda^\top$, and the coefficient $u_{1,e}$ in the expansion $K(t) \approx (4\pi t)^{-d_s/2} (u_0 + u_1 t + \mathcal{O}(t^2))$ is sensitive to the entire spectral distribution, including the spectral dimension d_s 's asymptotic behavior, and the local curvature via the second coefficient of the expansion. This means that even when local connectivity patterns are stable, reflected in constant degrees and neighbor overlap, the heat-kernel curvature can detect subtle changes in the diffusion geometry of the network, such as shifts in the spectral gap that alter heat propagation or changes in the effective dimension as the eigenvalue density evolves. The plateau in $u_{1,e}$ thus reflects a period of stability in the spectral geometry, the distribution of eigenvalues, and their corresponding eigenfunctions, which may persist even as local connectivity patterns remain unchanged. This multiscale sensitivity is not a peculiarity of this dataset; the spectral decomposition of the graph Laplacian is a universal feature of any undirected graph, and the short-time heat-kernel expansion provides a well-defined geometric characterization applicable to any evolving network. Such situations, where spectral geometry evolves independently of local connectivity, could arise in social networks where individual links stabilize but community structures continue to rearrange, in financial networks where local exposures remain constant but systemic risk metrics shift,

or in ecological networks where local species interactions stabilize while population-level dynamics undergo gradual change. The general principle is that the heat-kernel curvature probes the geometry of the graph's diffusion space, which can evolve independently from the local connectivity structure, offering a complementary perspective that is not accessible through purely local or global-in-a-different-way measures.

We also note that Fig. 3(b) shows the rate of change of the average edge curvature as a function of time. The observed reduction in fluctuation amplitude after day 180 could indicate that the network's geometric evolution slows considerably following the transition to scale-free networks, but it does not necessarily imply a complete cessation of structural change. The sustained small oscillations thereafter may reflect ongoing epidemiological dynamics, such as successive waves of infection or the introduction of new variants, that continue to perturb the network geometry, nonetheless with less dramatic effect than the initial transition.

Turning to the connection probability $p(x)$ whose behavior is captured in Fig. 7, we note that at the onset of the disease, the underlying transmission network is largely disconnected: few localities have reported cases, and interlocality spread has not yet established itself. In this regime, the high connection probability $p(x)$ near unity across a broad range of hyperbolic distances reflects the potential for transmission: given the geographic proximity structure ω_{ij} and the absence of significant immunity or serious behavioral changes, any introduction of an infected case into a new locality has a high probability of seeding onward transmission to neighboring localities. The network is thus highly responsive to the spread, with each new infection capable of creating links between previously nonconnected localities.

As the disease progresses, several factors contribute to the observed drop in $p(x)$ after day 180. First, the buildup of population immunity, whether through infection or later on through vaccination, reduces the susceptibility of localities. This makes it less likely that an introduction of infected cases leads to sustained transmission. Second, the network itself becomes more connected; once many localities have experienced outbreaks, the marginal gain in connectivity from each new transmission event decreases. Third, behavioral changes further suppress long-range transmission. Thus, the drop in $p(x)$ reflects the system's transition from a susceptible, sparsely connected state, where each new case can easily connect localities, to a more saturated state where existing immunity and network density limit the formation of new infection links.

This interpretation aligns with the observed timeline: the early high $p(x)$ corresponds to the phase of rapid geographic expansion of the virus, while the decline after day 180 coincides with the stabilization of the network topology and the emergence of scale-free structure reported in Ref. [27]. In this view, $p(x)$ captures the effective ease with which new interlocality connections emerge, which naturally decreases as immunity accumulates due to sufficient mixing.

Returning to the earlier point about temperature, we can now reconcile it with the behavior of $p(x)$. Before day 180, the temperature T was very low (near zero), meaning the connection probability function was extremely steep: connections

formed almost deterministically within a threshold hyperbolic distance. Simultaneously, the magnitude of ζ was extremely large (since $\zeta^2 = -6\bar{u}_{1,v}$ and $\bar{u}_{1,v}$ was highly negative), which compresses the effective hyperbolic distance scale. The product $\zeta(x - \mu_{cp})$ thus becomes large for even small separations, pushing the sigmoid into its step-like regime. The combination of low T and large ζ produced the observed high connection probability across a broad range of distances: the steepness ensured that any pair within the threshold connected, and the compressed distance scale meant that many pairs fell within that threshold.

After day 180, both T and ζ increased substantially, which expanded the effective distance scale. This means that for the same geometric separation, the hyperbolic distance became smaller. The net effect on $p(x)$ depends on how these changes interact with μ_{cp} . The observed drop in $p(x)$ for large x indicates that the shift in μ_{cp} (the connection threshold) overcompensated for the changes in T and ζ , moving leftward so that fewer pairs fell within the high-probability region. Thus, after day 180, the network is characterized by a flatter attachment rule together with a lower connection threshold, resulting in a more clustered geometric arrangement that supports a stable scale-free topology.

Effectively, this drop in connection probability, which is concomitant with the changes observed in temperature and curvature, is an indicator of steadiness in the network structure. Subsequently, vaccination can target the detected hubs and central nodes to slow the spread of the disease. Before that, given the high connection probability, intervention, especially vaccination, might be noneffective.

The structural transition identified by the curvature measures at day 180 carries important implications for the timing and targeting of public health interventions. The pretransition regime (days 0–180) exhibits highly negative node curvatures and a near-unity connection probability across a broad range of hyperbolic distances, indicating a network in a far-from-equilibrium state where the disease is rapidly expanding geographically. In this regime, the network is highly responsive to the introduction of infections, with each new case capable of seeding onward transmission to previously unconnected localities. Consequently, vaccination campaigns launched during this early phase may appear to have limited effect, as the highly connected, nonstationary structure allows the virus to bypass immunized nodes through alternative pathways, akin to the effect observed when interventions are applied to networks with high effective temperature. The observed transition to a stable scale-free topology after day 180 marks a regime where the network has settled into a reproducible structure, characterized by a clear hierarchy of hubs and a reduced connection probability at large hyperbolic distances. In this stabilized phase, targeted vaccination strategies can be more effective: the identified hubs, localities with the highest betweenness centrality, can be prioritized for immunization, as their removal or protection would significantly disrupt the transmission pathways. More precisely, by identifying hubs in this phase, public health officials can locate localities that act as connectors between communities, analogous to bridges in a transportation network, whose immunization would fragment the network into smaller, more controllable components. However, while

curvature provides a powerful diagnostic tool for detecting structural changes and identifying influential nodes, it should not be used in isolation for operational decision-making. The curvature measure reflects the geometric structure of the transmission network as inferred from infection data, which is inherently lagging behind the real-time epidemiological situation. Furthermore, curvature-based recommendations must be contextualized with other critical factors, including health-care capacity, population demographics, vaccine availability, logistical constraints, and the emergence of new variants with different transmissibility profiles. Instead, curvature analysis should be viewed as a complementary layer in a multi-metric framework that combines geometric indicators with traditional epidemiological surveillance (such as reproduction numbers, hospitalization rates, and genomic surveillance) to inform dynamic, data-driven vaccination prioritization and targeted lockdown strategies, particularly during periods of network stabilization where structural interventions are most likely to succeed.

Finally, curvatures introduce another metric to assess risk in dynamics over networks and confirm that structural changes

can be detected using geometrically inspired indicators to inform policy in public health.

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DATA AVAILABILITY

The data that support the findings of this article are not publicly available because they are owned by a third party and the terms of use prevent public distribution.

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