

CoSoGMIR: A Social Graph Contagion Diffusion Framework using the Movement-Interaction-Return Technique

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Abstract: Besides the inherent benefits of exchanging information and interactions between nodes on a social graph, they can also become a means for the propagation of knowledge. Social graphs have also become a veritable structure for the spread of disease outbreaks. These and its set of protocols are deployed as measures to curb its widespread effects as it has also left network experts puzzled. The recent lessons from the COVID-19 pandemic continue to reiterate that diseases will always be around. Nodal exposure, adoption/diffusion of disease(s) among interacting nodes vis-a-vis migration of nodes that cause further spread of contagion (concerning COVID-19 and other epidemics) has continued to leave experts bewildered towards rejigging set protocols. We model COVID-19 as a Markovian process with node targeting, propagation and recovery using migration-interaction as a threshold feat on a social graph. The migration-interaction design seeks to provision the graph with minimization and block of targeted diffusion of the contagion using seedset(s) nodes with a susceptible-infect policy. The study results showed that migration and interaction of nodes via the mobility approach have become an imperative factor that must be added when modeling the propagation of contagion or epidemics.

Keywords: COVID-19; SI-graph; pandemic propagation; diffusion models; small-world graphs; SIS; SIR.

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1. Introduction

A disease refers to a disorder that impedes performance or a deviation in the structure and functioning of a system [1]. It is also a harmful impediment to a system's structural norms or function. Commonly associated with physical injury, it occurs in a specified location [2] and often yields specific symptoms that appear [3] to indicate abnormal conditions inherent in such a system [4], [5]. Diseases can morph into contagion, spreading via a medium that includes fomites, oral (injection), direct contact, aerosol, and vector-borne [6]. Its diffusion spread within a society (comprised of various actors/agents) – often yields a regular spread of contagion, epidemic, or pandemic. It can become an epidemic directly impacting society [7]–[9]. Studies today are geared towards modeling propagation tasks on a social graph of connected actors to investigate relations, the structure, and underlying features of interest [10]–[13]. It can exist in 4-forms: hereditary, non-hereditary, deficiency, and infectious [14].

Movement of these actors/agents – plays a critical and significant role in the transformational process [15], [16] of the social graph to help de(escalate) local disease(s) outbreaks as they morph into epidemic cum pandemics, and vice versa. And thus, it continues to necessitate the inclusion of human migration and interaction into every contagion model – to effectively simulate disease cum epidemic outbreaks and to implement a dependable, dynamic framework as future preventive programs and policies for any contagion [17], [18]. With the

unfettered, sporadic movement of nodes (on a time-variant, social-graph with the probability distribution of nodal observations), these can portend some issues to include: (a) nodal exposure to diseases (i.e., targeting), (b) diffusion (i.e., spread of infection), and (c) rate of recovery (i.e., time taken for infected nodes to recover depending on their adoption/susceptibility level(s) [19].

Our study wishes to address such problems using the movement-interaction-return ensemble that models nodal targeting and exposure to disease/contagion to seek its rate of propagation with exposure to seed-nodes on the social graph as well as compute the expected recovery rate and time of susceptible-infect (SI) nodes vis-à-vis the nature of the disease cum contagion.

2. Review of Related Literature(s)

2.1. Agent-Based Modeling on Social Networks

A social graph is a collection of networked nodes or actors whose interaction with each other ripples formal changes in the nodal observations in relation to their exposure to an idea or innovation. Thus, it advances learning as the innovation diffuses within the structure's nodes [20]. A social graph ripples learning changes propagated across nodal relations as it seeks to explain structural theories of observed patterns and analyze nodal (local) features that eventually morph into global trends [21] via nodal interactions that finally yield a social convergence. These global trends manifest explicitly as nodal ties. Thus, as learning occurs, each node and its relations seek to (re)shape via social interactions – each graph over time to enable the adoption of innovation (i.e., in this case, disease cum contagion) [22].

A graph $G = (E, V, w)$ is a structure where each node $i \in V$, with a corresponding set of links (i.e., ties) where each $m \in E$, and has a corresponding assigned weight (w) called the cost or penalty of interaction. Its ties/link, measured using dyads D [23], can either be self-looped, singly-linked, or multi-linked. Each graph G is poised with 2-points: (a) to objectify how these nodal structures evolve, and (b) to understudy local features that cascade social processes and specify how we can exploit/explore these local features via nodal interaction(s) [24]. With the society as our case in point, nodal migration and interactions (among a set of V) are imperative as they will provide lessons and measures to help combat diseases/contagions (i.e., innovation) as these nodes interact (via a set of links/ties E), and ripples within G – a series of corresponding associated costs (w) therein [25]. As local properties morph onto global patterns – modeling migration-interactions can provide crucial knowledge in planning the future states cum scale of an event [26], [27]. Such models become tools to yield insight about the future, with predictions to yield timely insights about the velocity and veracity of a probable event [28]. The reliability of the generated results is best seen as prophecies/fore-warnings rather than predictions and is open to questioning as either myths or fictions [29]–[32].

At the crux of nodal interactions is the agent-based model (ABM). This is best illustrated with a flock of birds flying as a single system in tight formation. With no leader bird, they gracefully choreograph their movement such that each bird responds to the flight pattern of its immediate neighbor; And results in a highly-nonlinear patterned, hypnotic rhythm [33], [34]. We model such interaction as a sum of local feats represented in 3-rules, namely: (a) separation or collision avoidance in which each bird distances itself from others, (b) alignment for which each bird's direction and velocity are matched by its immediate neighbor, and (c) cohesion for which each bird's preserves a perceived equidistance of both the flock and its immediate neighbor to realize such flight formation with a steady paced, velocity and direction. Each bird is thus, as an actor or agent, modeled with local features whose interaction with the others yields such realistic flight formation or pattern [35]–[40].

Thus, an ABM can yield or be used to represent a dynamic, self-organized, complex, and non-linear framework that cascades local, nodal properties as processes learned by interacting actors/agents in response to a variety of external/internal influences (shocks) – to yield a global pattern eventually [41]–[43]. ABM's potential focuses on emerging processes to yield a structure that (a) posits a dynamic ensemble shaped via observing nodal interactions, and (b) it tests learning as a social convergence that manipulates the network structure using features such as its nodal stratification, graph topology etc [27], [44]–[47].

2.2 Diffusion Framework Models

For a social network, innovation (ideas or products) can diffuse across a graph via seed-node actions for tight clusters on a typical susceptible-infect (SI) case. The seed-node is a set of infected actors that have adopted an innovation (in this case, a disease). Other nodes in G , yet to adopt the innovation, are initialized, and can take any 3-states, namely: (a) susceptible – here, a node can be exposed to an innovation; But has a threshold limit for which aids it to either accept/reject an innovation, (b) infected – here, the node is exposed, and is influenced by its immediate neighbor to accept the innovation having exceeded its threshold limits. Thus, it becomes infected [48], [49] and has the ability (i.e., external shock) to influence other nodes to either accept/reject the innovation and (c) be removed – here, the infected node is placed on treatment, so that in time – the infected node recovers; The same treatment is placed on all other immediate neighbors that are infected till either a stop criterion is reached or no more infected node(s) exists on the social graph and/or network [50], [51].

With this in mind, the susceptible-infect (SI) diffusion ensemble can be represented in 2-classes, namely: (a) SI-Susceptible (SIS) and (b) SI-Remove (SIR) [52]–[55]:

1. SI-Susceptible: At time $t = 0$, G inserts into it a set of nodes (seed-nodes) (i.e., infected and have accepted/adopted the innovation), such that an infected node x has only a chance at any time t , to be exposed its immediate neighbor node y . With y exposed – the probability that n the node x convinces node y yields P_{xy} – so that if x succeeds, then y becomes infected at time $t + 1$; Else, y may reject now – only to accept later (i.e., accept either from x or from another node z). Also, y can reject at time $t + 1$ and then, later, accept at time $t+2$. This can go on and stop after either n -steps, or if there exists no node(s) to be exposed. We can also note that a node can be exposed any number of times such that even after such node is treated and has recovered – when exposed, it becomes susceptible again to be infected [56], [57]. Thus, if G has M -nodes with d -seedset (M_d) placed on G , at complete diffusion – it yields $S(M_d, G)$ as the maximum expected number of adopters as in Equation (1) As G evolves, the innovation either propagates or dies; And each actor accepts the innovation with a probability λ at rate $\frac{\lambda}{\delta}$, or rejects with probability δ This yields a threshold $\frac{\lambda}{\delta}$ that sufficiently aids quick recovery of the nodes in G [5], [58]–[62].
2. SI-Remove: At time $t = 0$, G inserts into it, a set of nodes (seednodes), such that an infected node x has only a chance at any time t , to be exposed its immediate neighbor node y . When an infected node x is removed (treated), it is no longer susceptible and can never become infected again even if/when re-exposed to an infected neighbor. Thus, node y if exposed may not become infected (only if it has been previously treated); Yet, if node y has never been exposed; it remains susceptible until treated of the innovation. The probability that node x infects y remains P_{xy} – at time $t+1$. Again, with treatment placed on all the nodes that have(not) been exposed, recovery commences even as diffusion goes on. This goes on and stops after either n -steps, or if there exists no node(s) to be exposed. This implies that each node, once recovered, can never be susceptible, and each node is exposed exactly once. Thus, if G has M -nodes with d -seedset (M_d) placed on G , it yields $S(M_d, G)$ as the maximum expected number of adopters – based on random choices of the SI-diffusion model and the nature of G in use. Equation (1) yields the maximum expected number of adopters.

$$S_d(G) = \max_{M_d} S(M_d, G) \tag{1}$$

$A_d = \arg \max_{M_d} S(M_d, G)$ does an adaptive seedset make a choice, And $S_d(G)$ is the rate of contagion diffusion in G . Thus, it yields Equation (2) that shows the contagion spread with the randomized seedset to denote the expected propagation and spread over all possible positions with d -innovations placed on G as [39], [63].

$$S'_d(G) = E_{M_d}[S(M_d, G)] \tag{2}$$

3. Proposed Material and Method

3.1. Data Gathering

Nigeria is arguably in Africa, the most strategic and influential nation – in lieu of her being the most populous black nation, her vast natural resources of hydrocarbon(s) vis-à-vis her government’s commitment to anti-corruption and to the unity of Africa. The abundance of minerals has continued to foster Nigeria’s heavy dependence on her oil, which accounts for over 41% of her Gross Domestic Product and 90% of her export revenues [51], [64]–[66]. The study uses the COVID-19 contagion (as its innovation), and models Delta State as a small-world graph with diffusion time as the lockdown from April 2020 to March 2022. Of the 270-wards in Delta State, we use 27-wards (as clusters) to introduce the seedset as in Table 1.

Table 1. Network Parameters with sampled COVID-19 epidemiological features

Network Features	Corona Virus Contagion
Total number of Clusters	27 wards (cluster)
The age range in Population	456,342 persons/contacts
Introduced Seedset	5-seedset nodes per ward
Diffusion Time ($t = 0, 1, 2, 3, \dots, n - 1$)	April 2020 to March 2021
Probability Density Distribution	$\mu = 0.5346$ and $\delta = 0.34$
Average Time of Adoption	Six weeks
Lowest/Highest Saturation (Final number of Adopters)	36% / 80% at $t > 0$ and $t = t - 1$ respectively

3.2. Problem Formulation

The propagation spread size of COVID-19 on society is a function of nodal migration and return, social interaction of (un)participatory nodes and other accompanying epidemiological features. The contagion size is that fraction of the population that remains infected even if and when the contagion has reached a stationary state (i.e., time has expired, and there are no more nodes to infect). A sample contagion size is a seedset introduced into the framework as in Table 1 [67]–[69]. Nodes are represented as migrants and residents via relations to yield the appropriate/requisite ties and tie-strength to G . Residents yield stronger ties; while, migrants yields weaker ties with nodes on G . Tie-strengths are measured using dyads d , and exists in G to propagate cluster formations as they morph into communities. Ties are all the pair of interactions between n-nodes with m-ties to form the matrix T of $n \times m$ in G [70], [71]. Each tie formed helps each node to overcome data issues of retrospective accuracy and acts as data feedback to benefit nodes during migration and return. Thus, we compute tie-strength (as a precursor to acceptance/adoption of an innovation) modeled as a linear combiner to yield Equation (3) – with N_i as the structure of G , R_i is all predictive variables, EI_i are all shocks in pairs dyadic relations, e_i is error term for maximization, and D_i is all dyadic pairs.

$$S_i = \alpha + \beta R_i + \gamma D_i + N_i + EI_i + e_i \tag{3}$$

3.2. Proposed Movement-Interaction-Return (MIR) Contagion Framework

We model the movement-interaction-return contagion framework as a variant of the Markov Model. It models graph G as a set of nodes with state transition ties (i.e., a series of chaotic and dynamic chain sequences with assigned probabilities as the cost associated with state transitions). This variant addresses the fundamental issues raised therein the Markovian process via the (a) use of positional data (alignment) in the sequences (i.e., resident and migrant nodes) and (b) use of a null transition state(s) to match insert/delete (migration) sequences for observed transition [72], [73]. Thus, for the nodal observations, the probability distribution cum state transition for the generated sequences of the task at hand includes [51], [74]–[78]:

1. Targeting with seedset: Given observations m and n , we seek to determine $\lambda = (A, B, \pi)$ that best fits the sequence. We train the ensemble to fit the data using the

features of interest. HMM training requires no aprior assumptions about the model other than outline parameter N and M , which specifies the model's size.

2. Rate of Infection Diffusion: We compute the probability that the model produces a sequence given that Equation (1) and (2) holds true with $= (A, B, \pi)$ and compute $P(O/\lambda)$.
3. Task-3: We uncover HMM $\lambda = (A, B, \pi)$ and observation sequence O to determine the most likely sequence of states $X = (x_1, x_2, \dots, x_T)$ that could have produced the sequence.

We propose the extended movement-interaction-return (MIR) framework to consist of three phases, namely: (a) movement, (b) interaction, and (c) return, respectively [79]–[81]:

1. Movement – G is populated with actors from a variety of geographical locations. Each can migrate (to new positions) across G with a probability P , and are etched with behavioral feats/roles for residents/migrants in a small-world graph to indicate their mobility pattern of choice. G uses the parameter $M_j^i k$ to denote nodal movement cum migration such that R_2^1 (as in Figure 1b) denotes a node in cluster-1 migrating to cluster-2; while, R_1^3 denotes node in cluster-3 migrating to cluster-1; And so on. This governs migration flow to describe the rate of nodal mobility i from the sub- $G j$ to another k as in Equation (4).

$$M_j^i k = \delta_j^i R_k^j + (1 + \delta_j^i) \delta_k^j \tag{4}$$

This feature ensures that resident nodes can migrate, and migrant nodes not in their place of residence must be forced to stay at their temporal residence until they are re-migrated home. As such, movement holds – both migrant and resident nodes can/may exchange innovation (data) and learn via interaction given in Figure 1a and 1b, respectively.

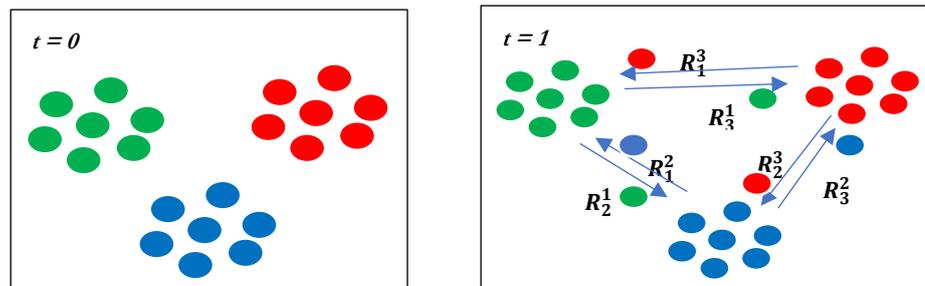


Figure 1. Nodal interaction (a) Nodal clusters at time $t = 0$; (b) Movement of nodes at $t = 1$.

2. Interaction: With a time-varying SIS in G , nodes interact with their immediate neighbors. Thus, if and when an infected seedset node x is exposed to y at $t + 1$, it has just one chance to infect node y . P is the probability rate at which the susceptible node y adopts the contagion from its neighbor x – given by the infectivity rate λ . Also, a contagion may die, node recovers with treatment, or may continue propagating (if untreated). The probability the contagion dies after treatment within its host carrier (i.e., infected node x) is given by the recovery rate μ as seen in Figure 2 [82].

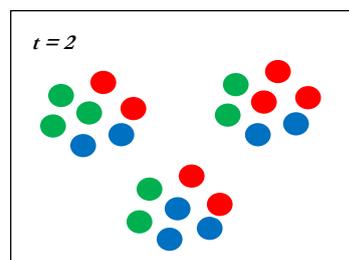


Figure 2. Nodal interaction with rates of infectious (λ) and rate of recovery (μ)

3. Return: Migrants are forced either to stay at their temporary location or return with a probability of γ^{-1} . This feat accounts for time convergence, where such migrants become destination node(s). With $\gamma^{-1} = 1(\gamma^{-1}) - G$ recovers to the original MIR form and state (i.e., migrants have returned). With $\gamma^{-1} \neq 1(\gamma^{-1}) -$ nodes lose data about their residence and become steadily re-distributed across neighbor locations [10] with $t - 1$ is as in Figure 3.

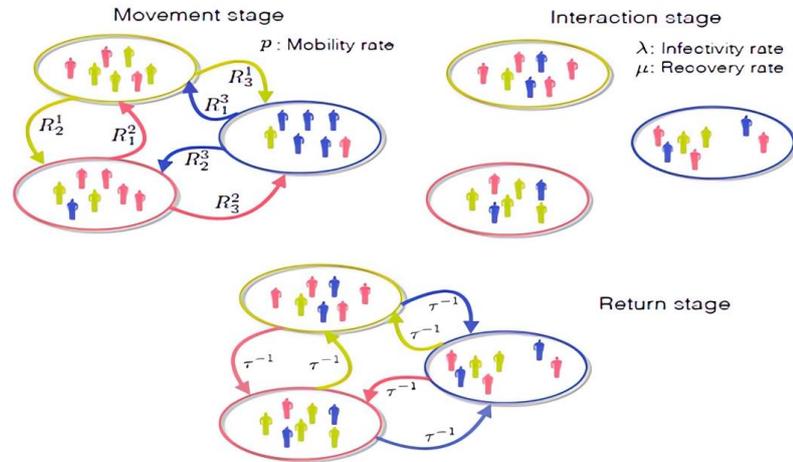


Figure 3. Schema model view of the Movement-Interaction-Return (MIR) Ensemble

3.3. Experimental Procedure

Each nodal exposure yields an updated, likely expected maximum number of adopters for the contagion. This implies that the ensemble yields an updated, optimal solution based on re-computed thresholds of exposed cum infected nodes. This update is achieved via the following steps as thus:

- a. Step-1: We normalize all nodal position(s) as in the algorithm listing 1, within the probability limits and range $[0 - 1]$,
- b. Step-2: We randomly swap each node's position using the roulette swap type to ascertain its rate of adoption/acceptance
- c. Step-3: With each nodal position reset, we re-computed the new threshold for G . This process continues till either all nodes are infected or the stop time criterion of 6 weeks is reached.

Algorithm 1. CoDiSoMIRE

INPUT: Number of nodes (N); Number of Ties (M).

OUTPUT: Print final adopters for network structure (N_iG).

- 1: Set initial Ties = 10+; Cluster Structure = 25+
- 2: Initialize G with the probability distribution $P_{\theta,G}(G = g) = \frac{\exp[\theta^t u(g)]}{c(\theta,G)}$
- 3: Node_position: set each with expected adopter Min-Max bounds $\rightarrow F(n - nodes * m - ties)$
- 4: Seedset choice: select randomly
- 5: **While** the node is not exposed
- 6: choose current_node position in seedset as the best nodal position in G
- 7: Dyadic and Predictive variable(s): set $(+D_i)$ and $(+R_i)$
- 8: Compute nodal-ties as function $(+R_i, +D_i, +N_iG, \text{ and } +Eli)$
- 9: **For** each n-node **Do**
- 10: **if** node_seedset \in nodal personal network **then**
- 11: approve_node_list (seedset)
- 12: **end if**
- 13: **end for-each**
- 14: Compute the adoption of contagion within the network structure using the linear combiner:

$$N_i(G) = P_{\theta,G} + \lambda_0 \mu_L + \lambda_1 Med_L + \sum_{t=0}^{t-1} \sum_{i \in L} \lambda_t (s - \mu_L)^t + \lambda_5 Min_L + \lambda_6 Max_L$$

- 15: Compute nodal disposition with exposure and infection/adaption of innovation at $t \geq 0$ as:

$$M_{new} = w \times M_{old} + c_1 \times rand() \times [(P_i)/T] + c_2 \times rand() \times [(P_n)/T]$$
- 16: Update nodal positions as: $P_{new} = (P_{old} - M_{new})$
- 17: // Until the stop criterion is met, or all nodes are exposed and infected: **Stop**

4. Results and Discussion

4.1. Result Findings

With each solution found, the ensemble restarts with a randomly selected seedset choice in G . Nodes with a threshold > 0.5 are chosen as the fit solution(s). The process stops if/when all nodes are exposed at the time $\geq t - 1$, or till a node with a threshold value ≤ 0.49 is reached. Thus, investigating and resolving criteria for targeting – as being the reasons to aid the adoption of an innovation vis-à-vis its propagation of COVID-19 – irrespective of the protocols in place, the results are shown in Table 2 using Equation (3).

Table 2. Simulated ties of expected maximum final acceptance/adoption

Variables	μ	$+R_i$	$+N_i$	$+D_i$	EL_i
Threshold Propagation	0.67	0.94	0.97	0.93	0.34
Distribution Density	0.47	0.87	0.88	0.91	0.37
Graph Support	0.43	0.90	0.92	0.95	0.21
Reciprocity	0.38	0.81	0.76	0.72	0.13
Structural Distance	0.09	0.80	0.82	0.86	0.34

The result agrees that the advent of the seedset yields a uniformly distributed acceptance of the COVID-19 contagion in G . This is seen from the values that with a mean structural distance of 0.09 (using approximately 10%) of population cluster as seedset, yields a threshold spread of 0.67 (i.e., 67%), with a dyadic interaction $+D_i$ of 0.93 (i.e., 93%) time convergence and a network structure $+N_i$ of 0.97 (i.e., 97% of infected nodes) as final adopters. This suggests that a highly clustered G will ease propagation time as a higher cluster coefficient (i.e., formation of cliques) further promotes spread ease along each node’s personal graph – even with the set protocols and measures to slow the propagation of COVID-19.

However, with the migration pattern infused into such a tightly knit, cohesive G as well as accounting for each node’s personal graph vis-à-vis the loose network structure introduced with migration – this slows down the rate of infection as seen in Table 4 of final adopters, a 0.95-significance (95%-significance) using Equation (1) and (2).

Table 4. Tie strength on the time of final adoption

Dependent Variables	Shocks	Personal networks via direct ties					Final adopters
		μ	$+R_i$	$+N_i$	$+D_i$	EL_i	
Early	4.1	0.76	0.31	1.6	12.7	9.9	24.2%
Early Majority	20.1	0.57	0.23	8.8	11.2	10.6	30.6%
Late Majority	12.1	0.46	0.21	13.7	10.1	10.0	33.8%
Laggards	6.5	0.32	0.20	1.9	6.1	3.4	11.4%
Actor Network	42.8			27.3	30.1	42.6	100%

The result shows that even with the seedset, laggards account for 11.4% of G . It is found to have been skewed considerably from a proportion of non-adopters vis-à-vis the datasets used. Laggards were found not to be infected at time $t - 1$ (stop criterion) for the study. It was hoped that at this time, all nodes may completely adopt the innovation (COVID-19) even with the set protocols cum its adherence. It can be attributed to features that residents cannot abandon their homes just as migrants are quarantined (and cannot be allowed to move back to their homes). Some nodes were also found to remain indifferent to the innovation as they neither rejected nor adopted it due to higher threshold(s).

4.2. Discussion of Findings

At time $t \geq 0$ of propagation, nodes become exposed to seedset nodes infected with the contagion. Migration ensures that more nodes become exposed and easily adopt/accept an

innovation due to shocks (such as the external influence of the need for acceptance to the newly migrated society). It directly impacted the network's threshold, disposing G towards a positive trend (for nodes to accept the innovation); Although each nodal graph displays a stronger resolve to reject the COVID-19 contagion. The ease with cluster formation, consequently, eased structured learning and interaction as dyadic ties advanced improved nodal retention of data learned in nodal memory in the network's quest for optimal solution.

The random exchange of data in a node's personal graph encourages data swap – and, in time, yields an improved nodal disposition for all node-set. The more exposed each node becomes, the more interaction with learning and retaining knowledge that improves each node's disposition and personal network; And in turn, impacts the graph as community-based shocks (global patterns). This agrees with [20], [46], [83], [84]. The adoption of the MIR-ensemble has become imperative and crucial as a threshold parameter to study network structures vis-à-vis the lessons learned from the COVID-19 pandemic. A node's adoption time for a specific innovation is proportional to its threshold limits and shocks/influences impacted upon the node by its graph.

The advent of seedset rippled in by migration mobility ensured that over 80% of active nodes became infected prior the stop criterion as in Table 4 with the (early, early majority and later majority) adopters of the innovation as reflected by both the nodal and graph thresholds. This agrees with [85], [86]. Finally, a high threshold value indicates such a node/graph possess high tolerance to reject the contagion, as experienced with the laggard nodes [63], [87], [88].

5. Conclusions

Our contributions include thus: (a) we define ties as a dimension in all social-networks G , (b) we acknowledged and modeled the network structure as a linear function with the probability distribution of nodes in the search domain being considered, and (c) we used the MIR ensemble with mobility pattern as a predictor for both nodal and graph's threshold. Our result extends the realization that help modulate the social features within a graph vis-à-vis filtering actor relations through clusters/cliques, and with recourse to both the properties of the node and the connecting link itself that enables actors/nodes interact with each other. We defined these properties as dimensions in the network structure itself.

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Appendix: Notation List

– $+D_i$ as dyadic pair relations,
 λ as infectivity rate,
 γ as nodal return probability,
 μ as the rate of nodal recovery from contagion.

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