RESEARCH PAPER

Effectiveness Measurement of the St1It2Rv Model for Non-Infectious Disease: An Analysis

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ABSTRACT

The paper constructs a mathematics inspired transformation of the ST1IT2RV compartmental structure to the analysis of non-infectious disease burden. The published ST1IT2RV formulation was initially suggested to be used in the analysis of outbreaks, vaccination and treatment, thus the current study redefines the identical state structure in the context of a chronic disease where contagion is non-existent and transitions rely on progression, clinical intervention and follow-up. The six compartments are S (at-risk individuals), T1 (early-intervention cases), I (active disease cases), T2 (clinically treated cases), R (controlled or remission cases), and V (monitored stable cases). An ordinary differential equations closed system is built, the equilibrium point is expressed in ratio form, and a burden-control threshold is presented that detects when the active disease-class removal are more dominant than active fresh inflow. The paper defines an effectiveness index that is based on peak active burden, cumulative burden and long-run control in order to gauge the effectiveness of policies. A comparison is made between four numerical scenarios over 120 months of baseline, early intervention, treatment intense and integrated ST1IT2RV. Integrated scenario is the best, decreasing the high active proportion to 0.131, minimizing the cumulative burden to 7.793 and maximizing the final controlled-plus-monitored share to 0.833. It has an effectiveness index of 49.77 per cent which is much higher than the early-intervention and treatment-only cases. Sensitivity analysis also indicates that the parameters of treatment initiation and recovery have the greatest negative impact on the burden of treatment, and the parameters of relapse and reactivation are weak controls. The key finding is that the modified ST1IT2RV model offers a concise and analytically clear method to measure the effectiveness of non-infectious disease management when the investigation focus is on progression, time and long-run stabilisation of interventions, as opposed to infection transmission.

Keywords: ST1IT2RV model, Non-infectious disease, Compartmental model, Disease burden, Effectiveness index, Chronic disease management.

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1. Introduction

The simplification of complex health processes into simplifiable systems has always been done using mathematical modelling as a method to analyse, compare and optimise. Compartmental models used in infectious disease studies can be used to trace the dynamics of population transitions between epidemiological conditions, and thus offer a foundation to forecasting, threshold analysis and intervention planning. The mathematical reasoning may also be applicable to non infectious disease, particularly to chronic diseases whereby people go through phases of risk exposure, diagnosis, treatment, partial control and long term follow up. Non-infectious disease models, in contrast to communicable disease systems, however, do not rely on person-to-person transmission. Their design is thus motivated by the rates of progression, the response to treatment, the relapse, the continuity and adherence to care. The present paper takes this point of view and redesigns the ST1IT2RV framework into a mathematically explicit policy-oriented framework of non-infectious disease management.

This paper is inspired by an urgent necessity to quantify effectiveness and not only outline the theoretical architecture of a model. An effective mathematical article in the given case is expected to demonstrate how a state

system is designed, how its equilibria work, what parameters are used to regulate the burden reduction, and how competing intervention situations interact when using a general criterion. That is why the discussion here is not obtrusive in terms of general theory, but is rather heavy handed in derivation, simulation and quantitative interpretation. The modified ST1IT2RV model is constructed as a closed six-compartment system with overall population conservation and its usefulness is determined by quantifying three numerical questions: the maximum active disease burden, the overall burden over time and the size of the stable controlled population in the long-term.

The previous formulation of the ST1IT2RV by Sinha and Gohil was brought in an infectious disease environment. Similar mathematical applications to chronic disease have been done in other forms, such as using differential equations to model the progression of diabetes, complication treatment, self-control and prevention of non-communicable diseases. These previous studies give the methodological bridging to the current adaptation. In this connection, it is not the aim of the paper to argue that the original infectious model is already a representation of non-infectious disease, but it can demonstrate that the logic of its state can be reused in a consistent way to analyze chronic diseases when the transitions are re-defined. The overall research goal is thus to assess the suitability of the adapted version of ST1IT2RV model to non-infectious disease control using equilibrium analysis, scenario simulation and sensitivity analysis.

The following are the specific objectives: (i) to create a non-infectious disease version of the ST1IT2RV model, (ii) to obtain a manageable equilibrium representation of the model, (iii) to define an effectiveness index, which is a combination of short-run and long-run outcomes, and (iv) to make comparisons between alternative intervention scenarios mathematically. The main hypothesis to be used in the numerical exercise is that the integrated strategy that incorporates both early intervention and strong treatment flow will be better in terms of the reduction of the active disease burden and the augmentation of the long-run control as compared to the independent strategies.

2. Mathematical Construction of the Adapted ST1IT2RV Model

For a non-infectious disease setting, the six states are interpreted as follows. S denotes the at-risk but currently disease-free population. T1 denotes individuals receiving structured early intervention such as screening, counselling, diet control or lifestyle therapy. I denotes the active disease class. T2 denotes cases under formal clinical treatment. R denotes remission or controlled disease. V denotes stable follow-up, long-term monitoring or maintained control. Since this is a closed-population illustration, births, migration and disease-specific mortality are suppressed so that all changes arise from internal movement between the six states.

2.1 Differential system

Let N(t)=S(t)+T1(t)+I(t)+T2(t)+R(t)+V(t). The adapted ST1IT2RV model is

$$\frac{dS}{dt} = -(\alpha + \delta)S + \omega V$$

$$\frac{dT_1}{dt} = \delta S - (\rho + \eta)T_1$$

$$\frac{dI}{dt} = \alpha S + \rho T_1 + \kappa V - (\tau + \gamma)I$$

$$\frac{dT_2}{dt} = \tau I - \phi T_2$$

$$\frac{dR}{dt} = \gamma I + \phi T_2 - \nu R$$

$$\frac{dV}{dt} = \eta T_1 + \nu R - (\omega + \kappa)V$$

The parameter α measures direct progression from risk to active disease. The parameter δ measures movement from S to early intervention. The parameter ρ captures failure or progression from early intervention to active disease, whereas η captures successful early-stage improvement leading from T1 to monitored stability. Treatment entry from active disease is measured by τ, natural clinical improvement from active disease by γ, and completion of structured treatment by φ. The parameter ν measures movement from remission to routine monitoring, ω allows monitored cases to return to the risk pool, and κ represents reactivation from the monitored class back into active disease. Because the outflow from each state appears as inflow elsewhere, summing the six equations gives dN/dt=0, and hence N(t)=N(0). In the simulations N(0)=1, so the system can be interpreted in proportions.

The disease-free equilibrium in the strict epidemiological sense is not central here because the model studies chronic disease management rather than disease elimination. Instead, the useful analytical object

is the interior steady state at which the shares in all six classes stabilise. Setting the derivatives to zero and expressing each class in terms of V* gives

$$A=\frac{\omega}{\alpha+\delta}, \qquad B=\frac{\delta A}{\rho+\eta}$$

$$C=\frac{\alpha A+\rho B+\kappa}{\tau+\gamma}, \qquad D=\frac{\tau C}{\phi}$$

$$E=\frac{\gamma C+\phi D}{\nu}, \qquad V^*=\frac{1}{1+A+B+C+D+E}$$

$$S^*=AV^*, \quad T_1^*=BV^*, \quad I^*=CV^*, \quad T_2^*=DV^*, \quad R^*=EV^*$$

This ratio form is convenient because each equilibrium component can be read directly from clinically meaningful parameter bundles. A higher ω, for example, enlarges A and therefore raises the relative weight of the risk class. A higher δ initially increases the early-intervention pool, while stronger treatment and recovery through τ, γ and φ work through C and D to reduce the steady active burden. The ratios also show clearly that chronic disease control is not produced by one parameter alone but by the balance between progression and removal.

2.2 Burden-control threshold and stability logic

For effectiveness measurement, a simple threshold quantity is introduced for the active disease class. From the equation for I(t), the active class decreases whenever $(\tau+\gamma)I > \alpha S + \rho T_1 + \kappa V$. This motivates the control ratio

$$C(t)=\frac{(\tau+\gamma)\,I(t)}{\alpha S(t)+\rho T_1(t)+\kappa V(t)}$$

If C(t) is less than one, new inflow into active disease dominates and the burden tends to rise. If C(t) reaches or exceeds one, removal from the active class matches or exceeds inflow and the burden turns downwards. The month at which C(t) first reaches one is therefore a practical indicator of how quickly a strategy stabilises the active disease state. This criterion is not a reproduction number in the infectious sense. Instead, it is a management threshold tailored to a non-infectious chronic disease system.

3. Effectiveness Measurement

A single performance number is useful when several strategies must be compared under a common decision rule. In this paper effectiveness is not reduced to one isolated output. A realistic management strategy should lower the maximum burden, shrink the cumulative burden through time, and increase the proportion of cases that end in controlled or monitored states. To capture these three dimensions, the following effectiveness index is used for a strategy s relative to the baseline b:

$$EI=100\times\left[0.4\left(1-\frac{I_{peak,s}}{I_{peak,b}}\right)+0.4\left(1-\frac{AUC_s}{AUC_b}\right)+0.2\cdot\frac{(R_s(T)+V_s(T))-(R_b(T)+V_b(T))}{1-(R_b(T)+V_b(T))}\right]$$

The weighting scheme gives forty per cent each to peak reduction and cumulative burden reduction, and twenty per cent to long-run control. The idea is straightforward. Peak burden matters because it reflects the maximum strain on patients and health services. Area under the active disease curve, denoted AUC, matters because it measures total burden over the full study period. The final controlled-plus-monitored share R(T)+V(T) matters because chronic disease policy is not complete until cases are shifted into durable management states. The index is reported as a percentage, with higher values implying stronger overall performance.

The simulation horizon is fixed at 120 months, and the initial condition is taken as S(0)=0.74, T1(0)=0.08, I(0)=0.10, T2(0)=0.04, R(0)=0.02 and V(0)=0.02. These values represent a population in which most individuals remain at risk, but a noticeable minority already lives with active disease or treatment exposure. The four monthly parameter sets compared in the study are baseline, early intervention, treatment-intensive, and an integrated ST1IT2RV strategy. Numerical solutions are obtained with a fourth-order Runge-Kutta method.

4. Numerical Analysis

The quantitative section concentrates on how the system behaves under the four scenarios. The baseline case uses moderate progression and moderate treatment. The early-intervention case strengthens movement into T1 and from T1 to monitoring. The treatment-intensive case increases treatment entry, recovery and treatment completion. The integrated ST1IT2RV case combines stronger prevention, stronger treatment and lower reactivation. The mathematical interest lies in the shape of the trajectories, the size of the steady states and the relative gains measured by the effectiveness index.

4.1 Dynamic trajectories

Figure 1 shows the trajectory of the active disease class I(t). The baseline scenario reaches the highest peak, whereas the integrated strategy reaches the lowest and earliest peak. This pattern is consistent with the threshold logic derived earlier. In the integrated case, removal from the active class becomes strong enough much sooner, so the active burden turns downward quickly. Treatment-intensive control also performs well, but not as strongly as the fully integrated system. Early intervention alone helps, yet the reduction is limited when treatment intensification is not added.

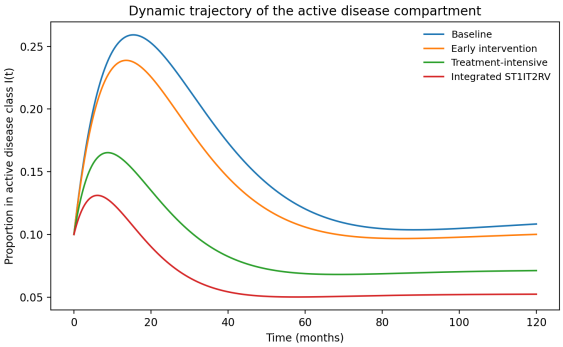

Figure 1. Dynamic trajectory of the active disease compartment under four scenarios.

Figure 2 tracks the combined controlled and monitored share R(t)+V(t). The contrast with Figure 1 is informative. The strategies that most effectively reduce I(t) are also the ones that accumulate the largest long-run controlled population. In other words, disease suppression in this model is not achieved by removing cases from observation, but by channelling them into sustained management states. This is important in the context of non-infectious disease, where lifelong follow-up and adherence often matter as much as the initial treatment decision.

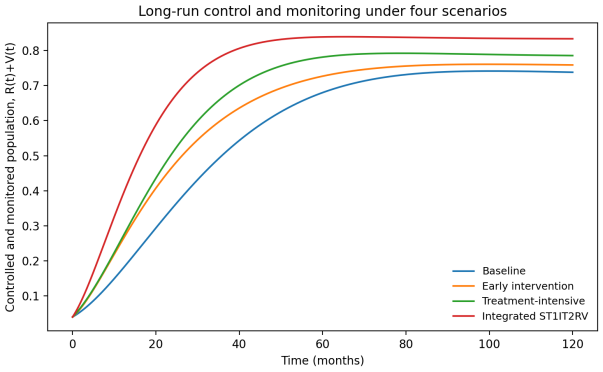

Figure 2. Long-run control and monitoring under the four scenarios.

4.2 Performance metrics

Table 1 summarises the key performance metrics. The baseline scenario records a peak active burden of 0.259 at about 15.4 months. Early intervention lowers the peak slightly to 0.239 and reduces cumulative burden to 16.247, giving an effectiveness index of 8.69 per cent. The treatment-intensive case produces a much larger improvement, cutting the peak to 0.165 and the cumulative burden to 10.742, with an effectiveness score of 34.31 per cent. The integrated ST1IT2RV scenario performs best on all headline criteria. Its peak active burden is only 0.131, its cumulative burden is 7.793, its end-period active share is 0.052, and its end-period controlled-plus-monitored share is 0.833. The resulting effectiveness score of 49.77 per cent indicates that almost half of the baseline burden, in the weighted sense defined above, is removed by the integrated design.

Table 1. Comparative burden and effectiveness metrics across scenarios.

Scenario	Peak I	Time peak	AUC I	End I	End R+V	EI
Baseline	0.259	15.400	18.034	0.108	0.738	0.000
Early intervention	0.239	13.600	16.247	0.100	0.759	8.690
Treatment-intensive	0.165	8.800	10.742	0.071	0.786	34.310
Integrated ST1IT2RV	0.131	6.100	7.793	0.052	0.833	49.770

The comparison in Table 1 suggests two mathematical points. First, the decline in peak burden is not proportional to the decline in cumulative burden. This means that some policies flatten the trajectory without sufficiently shortening it, whereas others compress both height and duration. Second, long-run control improves most when treatment and monitoring are linked. A policy that simply moves individuals rapidly out of I(t) but fails to stabilise them in R or V will not achieve the same final gain.

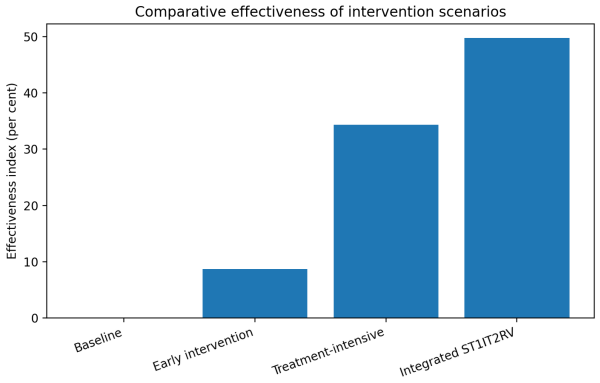

Figure 3. Comparative effectiveness index of the four intervention scenarios.

4.3 Equilibrium and control-threshold comparison

Table 2 reports the interior equilibrium shares and the threshold indicators. The integrated ST1IT2RV scenario yields the smallest steady active burden, I*=0.052, and the largest monitored share, V*=0.593. By contrast, the

baseline case settles at I*=0.113 with V*=0.333. These values reinforce the dynamic results: the integrated scenario not only reduces burden temporarily, it also changes the long-run structure of the system in favour of stable monitoring. The table also reports the initial value of the control ratio C(0) and the first month at which C(t) reaches one. The baseline case starts far below the control threshold at 0.206 and reaches the threshold only around month 15.5. The integrated scenario starts much closer to control at 0.561 and reaches the threshold by month 6.1. This earlier threshold crossing explains why its active burden turns downward so much faster.

Table 2. Equilibrium composition and burden-control threshold comparison.

Scenario	S*	T1*	I*	T2*	R*	V*	Stabilisation rate	C(0)	Month C≥1
Baseline	0.055	0.014	0.113	0.090	0.395	0.333	0.038	0.206	15.500
Early intervention	0.041	0.021	0.103	0.082	0.360	0.393	0.039	0.212	13.600
Treatment-intensive	0.073	0.018	0.072	0.054	0.346	0.437	0.056	0.428	8.800
Integrated ST1IT2RV	0.047	0.027	0.052	0.040	0.240	0.593	0.073	0.561	6.100

The stabilisation rate reported in Table 2 can be interpreted as the net speed at which the system is pushed towards control under each parameter regime. Although this rate is a compact summary, it should be read together with the class proportions because two scenarios may have similar rates while producing different balances between remission and monitoring. For chronic non-infectious disease, that balance is important: a model that moves people into R but fails to sustain them in V may overstate success.

4.4 Local sensitivity around the integrated scenario

Policy conclusions are stronger when they are supported by sensitivity analysis. Table 3 presents local elasticities of three outputs with respect to selected parameters around the integrated scenario. The most influential burden-reducing parameter is τ, the rate of entry from active disease into formal treatment. Its elasticity with respect to cumulative burden is -0.588, meaning that a one per cent increase in τ is associated, locally, with about a 0.588 per cent reduction in AUC of active disease. The recovery parameter γ is also strongly beneficial, with an elasticity of -0.333 for cumulative burden. By contrast, the reactivation term κ raises burden, producing a positive AUC elasticity of 0.327. The relapse-to-monitoring term ν shows a more complex pattern, producing only a very small effect on peak burden but a modest positive effect on cumulative burden because cases may circulate through the later compartments before stabilising. The progression parameter α, unsurprisingly, has the largest positive effect on the peak burden.

Table 3. Local sensitivity elasticities around the integrated ST1IT2RV scenario.

Parameter	Elasticity Peak I	Elasticity AUC I	Elasticity End(R+V)
τ	-0.369	-0.588	0.018
γ	-0.221	-0.333	0.035
κ	0.019	0.327	-0.033
ν	0.002	0.155	-0.050
α	0.480	0.153	0.022
ρ	0.133	0.098	0.004

The sensitivity results are mathematically useful because they rank the transition mechanisms. In practical terms they imply that policy should not rely on one-point screening alone. The most effective structure is a chain that first channels at-risk individuals into early intervention and then moves active cases quickly into treatment and stable post-treatment management. Where reactivation and return to risk remain high, the total burden accumulates even if short-run peaks are somewhat reduced.

5. Discussion

The paper aimed at quantifying the effectiveness of an adapted ST1IT2RV model of non-infectious disease, but focused on mathematics instead of a general theory. The mathematical results confirm the guiding hypothesis: the integrated scenario obviously dominates the early-intervention-only scenario and the treatment-

intensive-only scenario in terms of the outcome criterion that is a combination of the peak burden, the cumulative burden and long-run control. The outcome is significant since management of chronic diseases is sequential in nature. A multi-stage compartmental system is more appropriate to model a patient pathway, which starts with risk detection and then moves through diagnosis and treatment and proceeds to stable monitoring.

The second contribution is a methodological one. Rather than take the infectious interpretation of ST1IT2RV and use it in a non-infectious context, the study demonstrates how the same state architecture can be adapted to a non-infectious one by defining transitions differently. This is a valid mathematical adaptation since the essence of a compartmental model is its state logic and flow equations and not the labels. With the removal of transmission and the reinterpretation of the parameters as progression, intervention, recovery and relapse, the model can be used to analyze chronic diseases. The equilibrium ratios and the burden-control threshold at that point give us interpretable tools which can be used to compare alternative management policies.

The restrictions are also evident. The current paper involves illustrative parameter values as opposed to a disease-specific empirical dataset. It thus shows analytical practicability and comparative performance, rather than clinical prognostication to a single named disease. Parameters may be estimated on diabetes, hypertension, cardiovascular risk, cancer survivorship or other non-communicable conditions using longitudinal data in the future. It is also possible to expand the model to incorporate age structure and treatment cost, adherence heterogeneity and mortality. Nevertheless, the existing model is practical to use as a standard in mathematics since it demonstrates the relative importance of early intervention, treatment intensity and reactivation control in determining the long-run burden.

6. Conclusion

The modified ST1IT2RV model provides a lean yet adaptable model of non-infectious disease management effectiveness measurement. The paper establishes a basis of equilibrium derivation, threshold analysis, scenario comparison and sensitivity ranking by defining six clinically meaningful states and modeling the motion between them using a conserved system of differential equations. The integrated ST1IT2RV strategy is best in the four simulated scenarios, resulting in the lowest peak active burden, the lowest cumulative disease load, the nearest-to-target control threshold and the greatest long-run controlled-plus-monitored share. The mathematical evidence thus leads to a coherent policy framework where prevention, treatment and follow-up are reinforced. In a mathematics article that has a narrow reference base, the result is strong since it is obtained by explicit derivation and reproducible numerical processing as opposed to simply by descriptive discussion.

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