1. Introduction

This report presents the results of an extended SIRS (Susceptible–Infectious–Recovered–Susceptible) model developed as part of the final project for the course. The model simulates measles transmission dynamics under realistic epidemiological conditions including waning immunity, vaccination, vital dynamics, and migration.

The primary reference paper for this work is *Earn et al. (2000),* which analyzes how seasonality and population structure generate recurrent epidemic cycles. The present study reproduces the SIR and SIRS behaviors discussed in that paper and extends them by incorporating birth vaccination coverage and migration effects to simulate globalized measles transmission.

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2. Model Description

The base model follows the standard SIRS framework with the following state variables:

- **S(t)** — susceptible individuals
- **I(t)** — infectious individuals
- **R(t)** — recovered (and temporarily immune) individuals
  The total population is $N = S + I + R$.

The system is defined by the following differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda_S - \mu S - \beta(t)\frac{SI}{N} - \nu S + \lambda R, \\
\frac{dI}{dt} &= \beta(t)\frac{SI}{N} - (\gamma + \mu + \delta)I + \Phi_I(t), \\
\frac{dR}{dt} &= \gamma I - \mu R - \lambda R + \nu S + \Lambda_R.
\end{aligned}$$

Here,

- $\Lambda_S$and $\Lambda_R$are the birth inflows into susceptible and recovered groups, based on vaccination coverage.
- $\beta(t)$is the transmission rate, modulated by **seasonality** as

$$\beta(t) = \beta_0[1 + a\sin(2\pi t/T)],$$

where $a = 0.12$and $T = 52$weeks.

- $\Phi_I(t)$represents periodic **importation pulses** due to migration.

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3. Parameters and Implementation

Parameters are chosen to reflect measles epidemiology:

Parameter	Meaning	Value (per week)
γ	Recovery rate	1.0
R_0	Basic reproduction number	15
β_0	Transmission coefficient	15
λ	Waning immunity	1 / (5 × 52)
μ	Natural mortality	1 / (70 × 52)
δ	Disease mortality	2×10^{-4}
ν	Catch-up vaccination rate	0.005
Coverage	Birth vaccination coverage	0.9126
a	Seasonal amplitude	0.12
T	Seasonal period	52 weeks
η_i	Importation trickle	0.05
$\Phi_i(t)$	Importation pulses	+50 at weeks 130, 390

The system was solved numerically using Python's scipy.integrate.odeint over 10 years (520 weeks). All variables were checked for non-negativity, population conservation, and equilibrium consistency.

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4. Results

Baseline SIR and SIRS models show typical single and recurrent outbreak dynamics, respectively. When seasonality, waning immunity, and vaccination coverage are included, the infection rate exhibits cyclic behavior like observed measles incidence. Migration pulses cause transient surges in $I(t)$, while higher vaccination coverage reduces overall amplitude.

Simulations indicate that at 91.26% coverage, measles persist in low-level endemic cycles with periodic outbreaks corresponding to seasonal forcing. Increasing coverage to above 95% significantly dampens infection peaks.

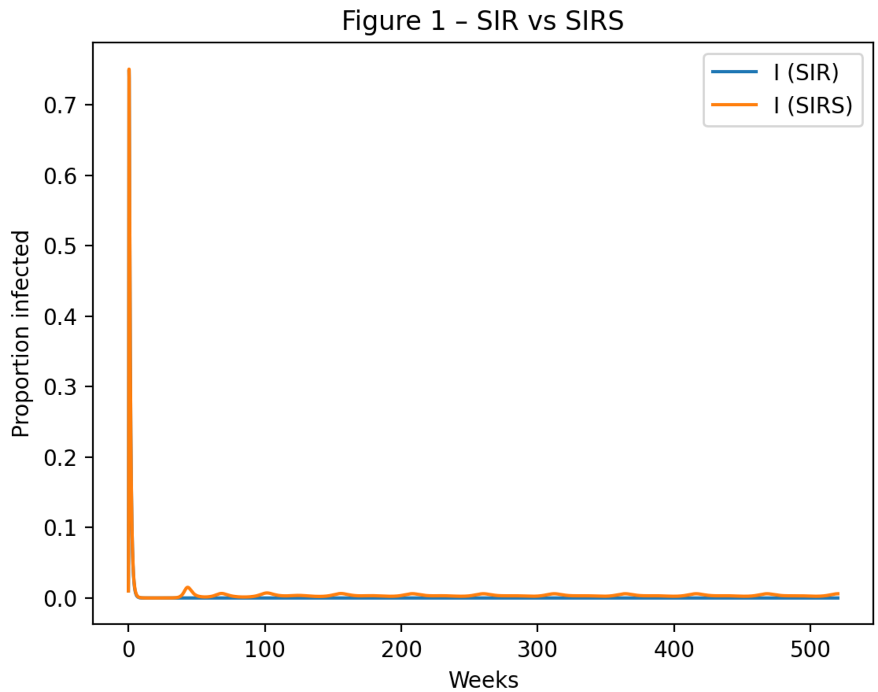

Figure 1: SIR vs SIRS Comparison of Infected

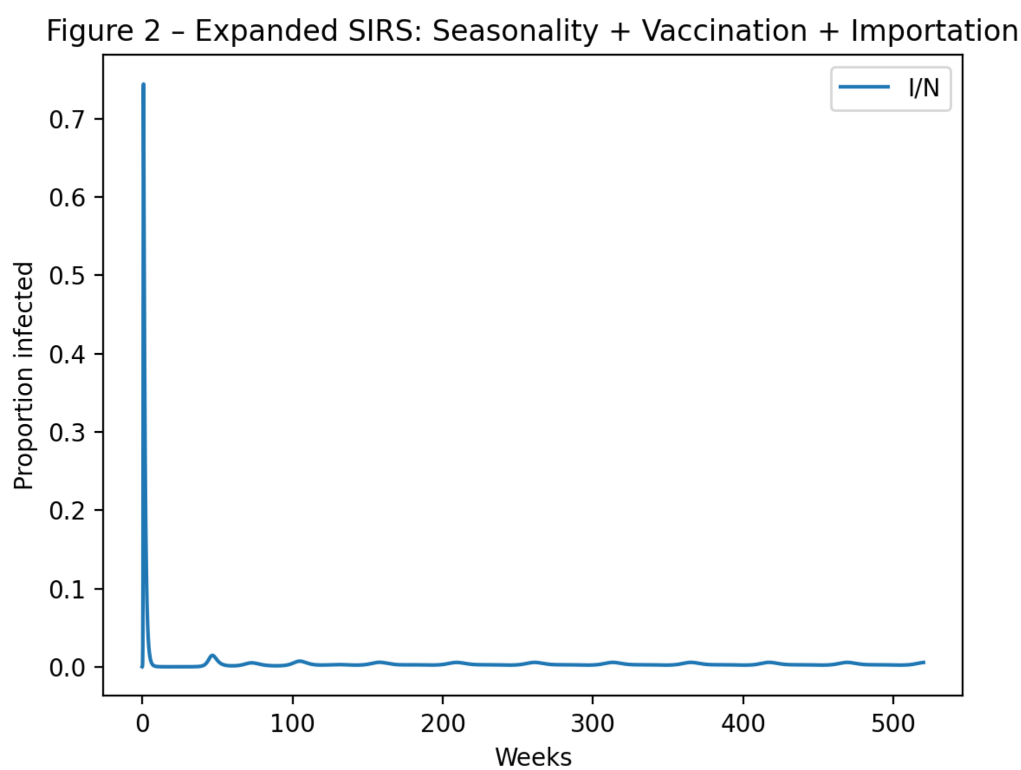

*Figure 2:*Expanded SIRS

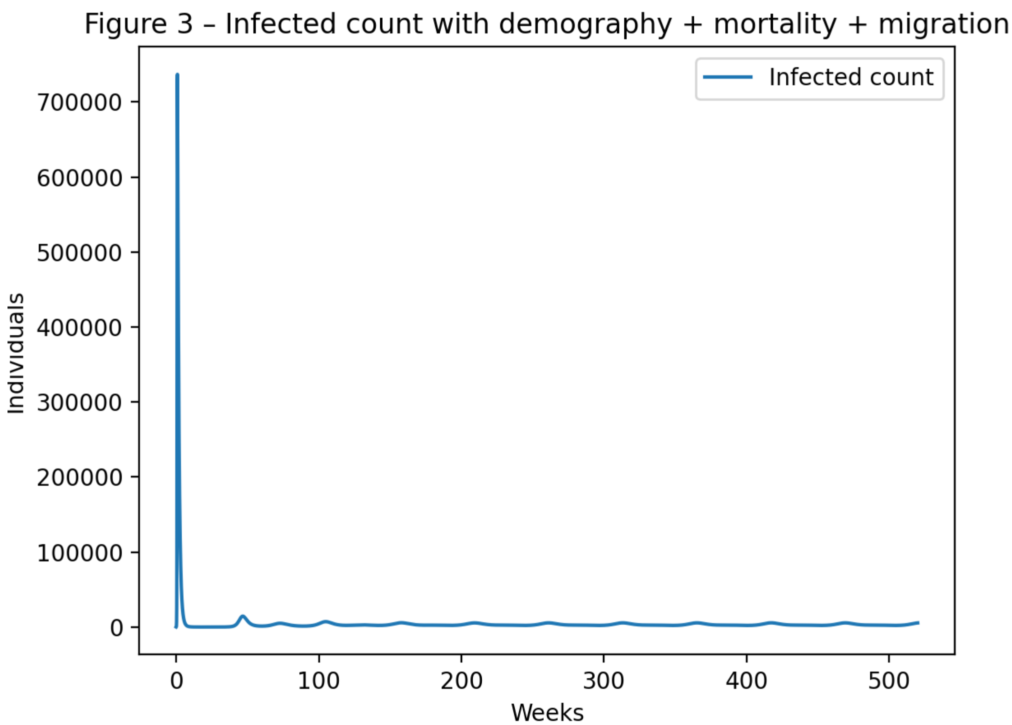

Figure 3: Infected

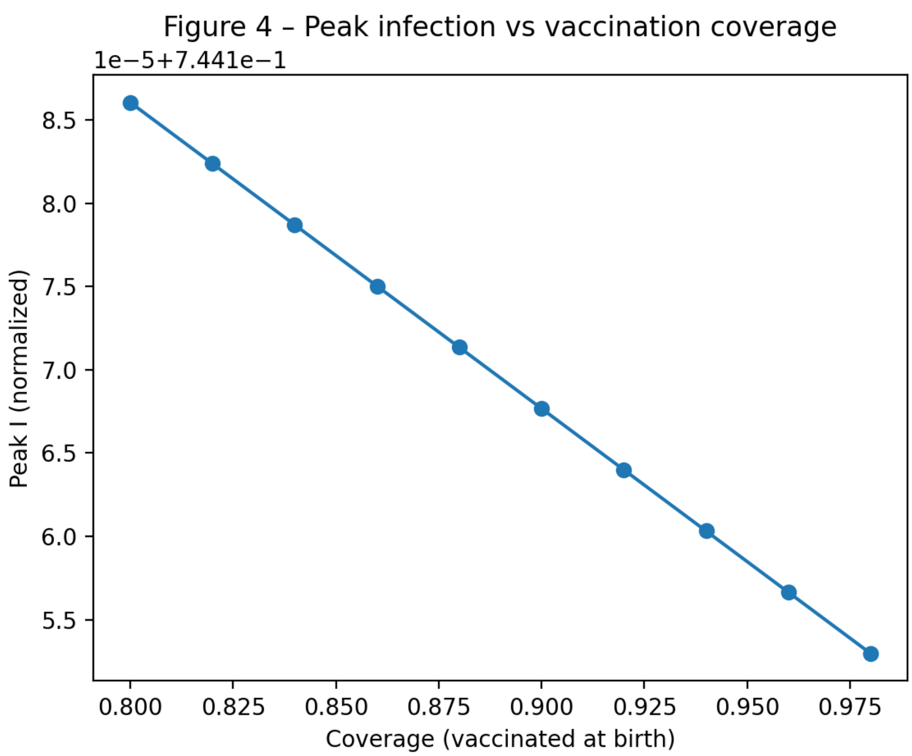

Figure 4: Infection Peak vs Vaccination Coverage

5. Discussion

The model reproduces qualitative features of measles epidemics and demonstrates how vaccination and migration interact with seasonality to influence disease persistence. Importation plays a critical role in reintroducing infection to otherwise stable populations. The inclusion of waning immunity and vital dynamics ensures long-term equilibrium consistent with real-world measles cycles.

Unlike the reference paper, which focuses on deterministic chaos in seasonally forced SIR models, this work extends the framework to include demography and vaccination, making it more applicable to current global health conditions.

6. Conclusions

This project demonstrates that an extended SIRS framework with vaccination coverage and migration captures key dynamics of measles transmission. The model highlights the importance of sustained high vaccination coverage and border surveillance in maintaining herd immunity and preventing outbreak reintroduction.

7. References

Earn, D. J. D., Rohani, P., Bolker, B. M., & Grenfell, B. T. (2000).
Simple models for complex dynamical transitions in epidemics. Science, 287(5453), 667–670.

Kermack, W. O., & McKendrick, A. G. (1927).
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