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Optimal Control and Uncertainty Analysis of an SEIRS Model for Online Shopping Addiction among University Students

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Abstract

Online shopping addiction is a growing behavioural problem among university students. This study formulates, analyses, and applies optimal control to an SEIRS model of online shopping addiction, calibrated with questionnaire data from students of the Faculty of Mathematics and Natural Sciences, Universitas Negeri Makassar. Writing the model in proportions makes its dynamics invariant to population size and so reconciles the calibration sample ($n = 98$) with the target population ($N_p = 3{,}028$). We prove that the feasible region is positively invariant, that the addiction-free equilibrium is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$ and globally asymptotically stable when $\mathcal{R}_0 \leq 1$, and that a unique endemic equilibrium exists precisely when $\mathcal{R}_0 > 1$; verifying the Castillo-Chavez–Song conditions, for which we obtain closed forms, shows the transition at $\mathcal{R}_0 = 1$ to be a forward transcritical bifurcation for all admissible parameters. Two bounded controls — prevention acting on transmission and education-and-counselling acting on the addicted class — are characterised by Pontryagin's minimum principle after establishing existence. At the point estimates $\mathcal{R}_0 \approx 1.03$. Because the rates are transformations of mean Likert scores rather than binomial proportions, sampling uncertainty is quantified by a stratified parametric bootstrap over respondent-level scores reconstructed from the reported subscale means, giving a median $\mathcal{R}_0$ of 1.05 with a 95% interval of [0.50, 1.97] and $P(\mathcal{R}_0 > 1) = 0.56$: the data cannot resolve on which side of the threshold the system lies, so the policy conclusions rest on the transient burden instead. That benefit is robust. Over ten years the combined strategy lowers the peak addicted population by 41.5% and cumulative addicted student-years by 66.4%, and across 200 resampled parameter draws the burden reduction never falls below 49%. Partial rank correlation identifies the progression rate, not the transmission rate, as the dominant driver of the transient peak, which explains why prevention alone reduces that peak by only 7.2% against 42.7% for counselling: the near-term peak is fed by an already-exposed cohort that prevention cannot reach.

Keywords: Basic reproduction number; Global sensitivity analysis; Online shopping addiction; Optimal control; Pontryagin's minimum principle; SEIRS model.

1. Introduction

The rapid development of information and communication technology has transformed consumption patterns. E-commerce platforms such as Shopee, Tokopedia, and Lazada have made shopping

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possible without spatial or temporal limits [1], and easy internet access, fast transactions, and promotions such as discounts, flash sales, and free shipping have raised public interest in online shopping; in Indonesia internet penetration has reached 78.19 percent of the population [2]. Students are among the most active users, with more than sixty percent of student respondents reporting regular online shopping [3], and the same convenience fosters excessive consumptive behaviour because digital marketing pushes individuals toward impulsive purchases without regard to actual needs [1]. Such behaviour can develop into online shopping addiction, a pattern of repeated and compulsive buying that is difficult to control even when the individual is aware of its consequences [4]: addicted individuals spend excessive time browsing, purchase unneeded goods, and use shopping as an escape from stress [5], with financial problems, declining academic performance, and mental health disturbances following [6]. Clinically, the behaviour is closely related to compulsive buying–shopping disorder, whose conceptualisation, measurement, and internet-based manifestations have received growing attention [7, 8].

The spread of online shopping addiction exhibits characteristics resembling the dynamics of infectious-disease transmission. Individuals who are not yet affected may be influenced by their social environment and by exposure to digital advertising, entering an exposed phase, then developing addiction, and potentially recovering through certain interventions. This similarity is reinforced by empirical evidence that behaviours and states such as obesity, smoking, and even happiness spread through social networks in a contagion-like manner [9]. Consequently, compartmental epidemiological models, whose mathematical foundations were established by classical epidemic theory [10], provide a natural framework for the quantitative analysis of addictive behaviour.

Several previous studies have applied compartmental models to addiction phenomena. Anwar [11] used an SEIRS model to analyse online game addiction among mathematics students at Universitas Negeri Makassar, while Siregar and Panjaitan [12] applied an SEIRS model to online game addiction by age group in Indonesia. Girsang et al. [13] built a mathematical model of online shopping addiction on Shopee, and Ahfa [14] applied optimal control to an SEIPQ model for online game addicts. Internationally, Li and Guo [15] and Guo and Li [16] developed online game addiction models with optimal control and family-education factors, following a broader tradition of optimal control applied to behavioural epidemics such as smoking dynamics [17].

Position of the present study. Equilibrium analysis, computation of a basic reproduction number, and optimal control are by now standard components of behavioural-epidemic modelling, and several of the works cited above already contain them: reproduction numbers and stability analyses appear in [11, 12, 15, 16], optimal control appears in [14–16], and primary questionnaire data were used for calibration in [11]. We therefore do not claim novelty for any of these ingredients individually. What is new is the following combination, of which the last two elements are, to the best of our knowledge, absent from the addiction-modelling literature cited above: (i) an SEIRS formulation applied to online *shopping* rather than online *game* addiction, with waning of behavioural protection modelled explicitly, whereas the existing online shopping model [13] contains neither a threshold analysis nor control; (ii) a two-control optimal-control problem in which prevention acts on the transmission term and education-and-counselling on the addicted class, so that the intervention structure matches the two distinct policy levers a university actually has; (iii) analytical rather than numerical resolution of the threshold structure — positive invariance, global stability of the addiction-free equilibrium when $\mathcal{R}_0 \leq 1$, and a forward transcritical bifurcation at $\mathcal{R}_0 = 1$, all valid for *all* admissible parameter values; and (iv) a formal uncertainty and global sensitivity analysis that propagates questionnaire sampling error into $\mathcal{R}_0$, into the transient outcomes, and — unusually for this literature — through the optimal-control problem itself, so that the reported benefit of intervention is a distribution rather than a single number.

Section 2 details the methodology, Section 3 presents the results and discussion, and Section 4

concludes.

2. Methods

This study is quantitative research employing a mathematical modelling approach, conducted at the Faculty of Mathematics and Natural Sciences (FMIPA), Universitas Negeri Makassar. The work is analytical and computational: it derives the invariant region, equilibria, stability conditions, basic reproduction number, and optimal control characterisation of an SEIRS model, and then evaluates them numerically using questionnaire data together with a formal uncertainty analysis.

2.1. Population, Sample, Instrument, and Classification

The study population comprises all active students of the faculty in the 2022–2024 cohorts, $N_p = 3{,}028$ students. The sample size was determined using the Slovin formula $n = N_p/(1+N_p e^2)$ with $e = 0.10$, yielding a minimum of 97 respondents; $n = 98$ students were selected by proportional stratified random sampling across study programmes.

The instrument is a 20-item questionnaire with five-point Likert responses (1 = strongly disagree, 5 = strongly agree), adapted from the compulsive-buying literature [7, 8] and covering four indicator domains: shopping frequency, expenditure relative to monthly income, self-control over purchasing, and interference with academic activity. Validity was assessed by Pearson product-moment correlation, all items exceeding the critical value $r_{\text{table}} = 0.199$ at the five percent level for $n = 98$; reliability was confirmed by Cronbach's alpha in the high category ($\alpha = 0.87 > 0.70$).

Classification rule. The allocation rule was as follows. Let $Q_j \in [20, 100]$ be the total score of respondent j, and let $\bar{Q}$ and s_Q be the sample mean and standard deviation. A respondent was classified as addicted (I) if $Q_j \geq \bar{Q} + s_Q$ *and* both screening items — on loss of control and on academic interference — were answered affirmatively; as recovered (R) if $Q_j < \bar{Q}$ *and* the respondent reported having previously experienced the addicted pattern and deliberately reduced it; as exposed (E) if $\bar{Q} \leq Q_j < \bar{Q} + s_Q$ with no affirmative screening item, that is, frequent exposure and elevated interest but retained control; and as susceptible (S) if $Q_j < \bar{Q}$ with no reported history of the addicted pattern. This rule produced $(S, E, I, R) = (50, 33, 3, 12)$ respondents. Two features of this classification should be borne in mind when reading the results: the addicted cell contains only three respondents, so the recovery rate has to be estimated on the pooled ever-addicted group $I \cup R$, and small sub-sample sizes are the dominant source of the parameter uncertainty quantified in Subsection 2.7; and the rule is score-based rather than clinical, so I should be read as "meets a screening threshold for problematic online shopping", not as a diagnosis of compulsive buying–shopping disorder.

Ethics. Participation was voluntary, written informed consent was obtained from every respondent, no personally identifying information was recorded, and responses were analysed only in aggregate. Ethical clearance for this survey-based study was granted by the Research Ethics Committee of Universitas Negeri Makassar (approval no. XXX/UN36/LP/YYYY, dated DD Month YYYY).

2.2. Model Formulation

The population at time t is partitioned into $S(t)$, $E(t)$, $I(t)$, and $R(t)$ with $N(t) = S(t) + E(t) + I(t) + R(t)$. The compartmental structure is shown in Fig. 1; all four are head-counts of students, and N is the constant faculty total. The interpretation of the compartments requires care, and we state it before the equations.

S is the class of individuals *currently at risk*: students who are neither exposed nor addicted at time t. Because the model contains the transition $R \to S$, this class is *not* restricted to individuals who have never been addicted; it contains both never-affected students and formerly recovered students whose behavioural protection has lapsed, and statements below about "preserving the susceptible population" should be read in that sense. E is the class of individuals regularly exposed to online-shopping stimuli, showing elevated consumptive interest but retaining control; I the class meeting the screening threshold for addiction; and R the class of individuals who have reduced the addicted pattern and currently hold the acquired behavioural safeguards, such as budgeting habits, notification management, or counselling support.
The parameter δ governing $R \to S$ therefore represents the *waning of behavioural protection*: recovered individuals gradually lose the safeguards they acquired and re-enter the at-risk pool, from which they may be re-exposed and become addicted again along the path $S \to E \to I$. It is not a direct relapse rate. A direct $R \to I$ relapse transition is a plausible refinement that we deliberately do not include, and we return to it in the limitations.

Assumption 1 (Model hypotheses)**.**

(A1) Individuals enter the faculty at rate ΛN and leave it at per-capita rate μ; all entrants join S. Taking $\Lambda = \mu$ gives $dN/dt = 0$, so the total population is *constant*. It is not closed: entry and exit occur continuously and $1/\mu$ is the mean time a student spends in the faculty. We use "demographically stationary" rather than "closed" throughout.

(A2) Behavioural transmission is frequency-dependent, at rate $\beta SI/N$: the chance that an at-risk student is drawn into the exposed class is proportional to the *prevalence* of addicted peers, not to their absolute number. This is the appropriate scaling for a social-contact process in which the number of relevant contacts per student does not grow with faculty size.

(A3) Exposed individuals progress to addiction at constant rate σ; addicted individuals recover spontaneously at constant rate γ; recovered individuals lose protection at constant rate δ. All sojourn times are exponentially distributed.

(A4) Two bounded controls act on the system: a prevention control $u_1(t)$ that reduces the effective transmission term by the factor $1 - u_1$, and an education-and-counselling control $u_2(t)$ that adds to the recovery rate of the addicted class.

Under Assumption 1 the controlled model is

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda N - (1-u_1)\frac{\beta SI}{N} - \mu S + \delta R,\\
\frac{dE}{dt} &= (1-u_1)\frac{\beta SI}{N} - (\sigma+\mu)E,\\
\frac{dI}{dt} &= \sigma E - (\gamma+\mu)I - u_2 I,\\
\frac{dR}{dt} &= (\gamma+u_2)I - (\mu+\delta)R,
\end{aligned} \tag{1}$$

with $S(0), E(0), I(0), R(0) \geq 0$. Summing the equations gives $dN/dt = (\Lambda - \mu)N = 0$. The uncontrolled system is Eq. (1) with $u_1 \equiv u_2 \equiv 0$.

2.3. Normalisation and the Sample–Population Correspondence

Both reviewers ask how the calibration sample $n = 98$ relates to the target population $N_p = 3{,}028$. The model itself answers this question, and we make the answer explicit.

Set $s = S/N$, $e = E/N$, $i = I/N$, $r = R/N$, so that $s + e + i + r = 1$.

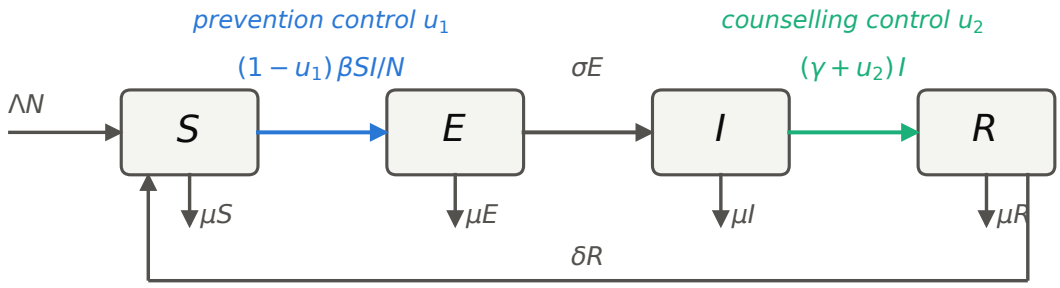

Fig. 1: Compartmental structure of the controlled SEIRS model Eq. (1). Recruitment ΛN enters the at-risk class S; every class leaves at per-capita rate μ; transmission is frequency dependent and is reduced by the prevention control u_1; the education-and-counselling control u_2 augments the recovery flow γI; and δR returns recovered individuals to the at-risk class as behavioural protection wanes.

Lemma 1 (Scale invariance). *Under Assumption 1 the proportions satisfy*

$$\begin{aligned} \frac{ds}{dt} &= \mu - (1-u_1)\beta si - \mu s + \delta r, & \frac{de}{dt} &= (1-u_1)\beta si - (\sigma+\mu)e, \\ \frac{di}{dt} &= \sigma e - (\gamma+\mu)i - u_2 i, & \frac{dr}{dt} &= (\gamma+u_2)i - (\mu+\delta)r, \end{aligned} \tag{2}$$

which contains no occurrence of N. Consequently two populations of different sizes but identical initial proportions *and identical parameters follow identical proportional trajectories.*

Proof. Divide each equation of Eq. (1) by the constant N and use $\Lambda = \mu$ together with $(\beta SI/N)/N = \beta si$. □

Lemma 1 settles the reconciliation. The questionnaire is used for two things only: it supplies the initial *proportions* $(s_0, e_0, i_0, r_0) = (50, 33, 3, 12)/98$ and it supplies the parameter estimates of Subsection 2.4. The population size enters nowhere in the dynamics; it is used solely to convert proportions into head-counts for reporting. Throughout Section 3, all trajectories are computed from Eq. (2) and all head-counts are obtained by multiplying by $N_p = 3{,}028$, which is the population the university would actually act upon. The corresponding initial head-counts are $(S(0), E(0), I(0), R(0)) = (1544.9,\ 1019.6,\ 92.7,\ 370.8)$, and the objective functional of Subsection 2.6 is likewise stated per capita and scaled by N_p when expressed in student-years. The sampling uncertainty induced by the finite sample $n = 98$ is not ignored: it is carried into every reported quantity through Subsection 2.7.

2.4. Parameter Estimation, Units, and Identifiability

Time unit. All rates are per year (yr^{-1}). The unit is fixed by the demographic parameter: the nominal duration of the undergraduate programme is four years, so $1/\mu = 4$ yr and $\mu = \Lambda = 0.25\ \text{yr}^{-1}$.

An identifiability caveat, stated first. A single cross-sectional survey identifies compartment *occupancies*; it does not identify transition *rates*. Recovering $\beta, \sigma, \gamma, \delta$ from one wave of data therefore requires an additional assumption, and it is more useful to state that assumption plainly than to present the resulting numbers as measurements. Two routes are conceivable. The first is to treat the observed cross-section as the endemic equilibrium and invert the equilibrium relations; this fails here, because the observed ratios $E/I = 11.0$ and $R/I = 4.0$ are incompatible with the equilibrium relation $E^*/I^* = (\gamma+\mu)/\sigma$ for any parameter set that also reproduces the observed S fraction, confirming that the surveyed cohort is in a transient rather than a stationary

state. The second route, adopted here, is to map item-level responses onto annual transition probabilities through an explicit and stated transformation. We use it, and we treat the resulting values as *nominal* estimates carrying substantial uncertainty rather than as measurements.

Mapping. For a transition $X \to Y$, let $\bar{q}_{XY} \in [1, 5]$ be the mean score, over the respondents currently classified in X, of the questionnaire subscale that measures propensity for that transition. Normalise to a probability scale by

$$p_{XY} = \frac{\bar{q}_{XY} - 1}{4} \in [0, 1], \tag{3}$$

interpreting p_{XY} as the probability that the transition occurs within one year, and convert to the exponential rate consistent with (A3),

$$\theta_{XY} = -\ln(1 - p_{XY}) \ \text{yr}^{-1}. \tag{4}$$

This yields σ, γ, and δ directly. The transmission rate is not a probability and requires the usual decomposition $\beta = c\, p_{SE}$, where c is the mean number of shopping-relevant social or promotional exposure episodes per student per year and p_{SE} is the per-episode probability of entering the exposed class. We fix $c = 12$ yr^{-1} (one relevant episode per month) as a stated structural assumption; because c is not estimated from the data, the uncertainty intervals reported below are, in this respect, optimistic.

Table 1 records the inputs and outputs of this procedure, and Table 2 lists the resulting parameter set. Every downstream number in this paper is produced by the script described in Subsection 2.8 and will update automatically if these inputs change.

Table 1: Conversion of questionnaire responses into transition rates. Sub-sample denotes the respondents over whom the subscale mean is taken; p follows from Eq. (3) and the rate from Eq. (4). The interval is the 95% interval of the parametric respondent-level resampling of Subsection 2.7; it is model-based, not empirical.

Rate	Transition	Sub-sample (n)	$\bar{q}$	p	Estimate (yr^{-1})	95% CI
β	$S \to E$	S (50)	1.45	0.1125	1.35	[0.72, 1.92]
σ	$E \to I$	E (33)	1.66	0.1647	0.18	[0.10, 0.29]
γ	$I \to R$	$I \cup R$ (15)	2.04	0.2592	0.30	[0.14, 0.48]
δ	$R \to S$	R (12)	1.56	0.1393	0.15	[0.06, 0.26]

$\beta = c\, p_{SE}$ with $c = 12$ yr^{-1}; $\mu = \Lambda = 0.25$ yr^{-1} from the four-year nominal study duration, 95% range [0.20, 0.28]. Intervals under the maximal-dispersion bound of Subsection 2.7 are roughly 1.6 times wider.

Table 2: Parameters of the SEIRS online shopping addiction model

Parameter	Description	Unit	Value	Source
Λ	Recruitment rate of new students	yr^{-1}	0.25	Set equal to μ
μ	Exit rate ($1/\mu$ = mean time in faculty)	yr^{-1}	0.25	Four-year study duration
β	Effective transmission rate	yr^{-1}	1.35	Questionnaire, Eq. (3)
σ	Progression rate, exposed to addicted	yr^{-1}	0.18	Questionnaire, Eq. (4)
γ	Spontaneous recovery rate	yr^{-1}	0.30	Questionnaire, Eq. (4)
δ	Waning rate of behavioural protection	yr^{-1}	0.15	Questionnaire, Eq. (4)
u_1	Prevention control (scales transmission)	—	$[0, 0.6]$	Control variable
u_2	Counselling control (augments recovery)	yr^{-1}	$[0, 1]$	Control variable

2.5. Threshold and Stability Analysis

Equilibria are obtained by setting the right-hand sides of Eq. (2) with $u_1 = u_2 = 0$ to zero. Local stability is assessed from the eigenvalues of the Jacobian, using the Routh–Hurwitz criterion where the characteristic polynomial factorises. Global stability of the addiction-free equilibrium is established by a Lyapunov function combined with LaSalle's invariance principle [18]. The basic reproduction number is computed by the next-generation matrix method [19], and normalised forward sensitivity indices $\Upsilon_p^{\mathcal{R}_0} = (\partial \mathcal{R}_0 / \partial p)(p/\mathcal{R}_0)$ follow [20].

2.6. Optimal Control Problem

Let T be the planning horizon and let the admissible control set be

$$\mathcal{U} = \left\{ (u_1, u_2) \in (L^\infty[0,T])^2 : \; 0 \le u_1(t) \le u_{1\max}, \; 0 \le u_2(t) \le u_{2\max} \text{ a.e.} \right\}.$$

We minimise, per capita,

$$J(u_1, u_2) = \int_0^T \left(A_1 \, i(t) + \tfrac{1}{2} b_1 u_1^2(t) + \tfrac{1}{2} b_2 u_2^2(t) \right) dt \tag{5}$$

subject to Eq. (2) and the given initial state. Quadratic control costs are the standard convexifying choice and encode diminishing returns to programme intensity [21].

Choice of the weights, and what they mean. The following scheme makes the weights dimensional rather than arbitrary. We take $A_1 = 1$ as the numéraire, so that J is measured in addicted student-years per student and $N_p J$ in addicted student-years. The control weights then carry the unit of population: writing $B_k = b_k N_p$, the instantaneous programme cost at intensity u_k is $\frac{1}{2} B_k u_k^2$ student-years per year, so $\frac{1}{2} B_k$ is the number of addicted student-years the institution is willing to spend one year of full-intensity programme k to avert. We set

$$b_1 = 0.010 \; (\, B_1 = 30.3 \text{ students}\,), \qquad b_2 = 0.050 \; (\, B_2 = 151.4 \text{ students}\,),$$

so that a year of prevention at full intensity is valued at 15 averted addicted student-years and a year of counselling at full intensity at 76. The bound $u_{1\max} = 0.6$ encodes the judgement that no campus programme can suppress more than about sixty percent of exposure to e-commerce marketing, while $u_{2\max} = 1 \text{ yr}^{-1}$ allows counselling to at most quadruple the baseline recovery rate $\gamma = 0.30$. Because all four of these are judgements rather than measurements, Subsection 3.8 reports how the conclusions move as each of b_1 and b_2 varies over a 32-fold range, each of $u_{1\max}$ and $u_{2\max}$ over a fourfold range, and T over $\{4, 10, 20\}$ years.

Choice of horizon. We take $T = 10$ yr as the baseline. This is 2.5 mean residence times, long enough for the transient dynamics of interest to run their course, and short enough to be a meaningful institutional planning horizon. As Subsection 3.4 shows, the slowest eigenvalue at the endemic equilibrium corresponds to a relaxation time of about 150 years, so no horizon that is meaningful for a university reaches the asymptotic regime, and reporting transient rather than asymptotic quantities is the honest choice.

2.7. Uncertainty and Global Sensitivity Analysis

Because $\mathcal{R}_0$ is estimated to lie close to its threshold, a point estimate alone is not informative, and we quantify uncertainty formally following standard practice for compartmental models [22].

Why a binomial model is not appropriate here. The index p_{XY} of Eq. (3) is an affine transformation of a *mean subscale score* on a five-point scale, not a binomial proportion: no respondent contributes a Bernoulli outcome, and the sampling distribution of $\bar{q}_{XY}$ is governed by the dispersion of the individual scores rather than by $p(1-p)$. Attaching a Beta posterior to p_{XY}, as one would to a count of successes, therefore misstates the sampling model. The natural remedy is to resample the objects that were actually sampled — the respondents — which is what we do.

What the reported intervals are, and are not. One qualification must be stated before the procedure, because it governs how the intervals should be read. A respondent-level bootstrap needs the individual responses. Those responses exist but are not part of the record analysed here: the manuscript works from the four subscale *means* of Table 1, so the individual scores have to be *reconstructed* from those means through an assumed response distribution. What we report is therefore a *parametric* bootstrap over reconstructed respondent-level scores, and the resulting intervals are model-based rather than purely empirical: they quantify the sampling variability implied by an assumed response distribution with the observed mean, not variability read off the observed responses themselves. We say so wherever the intervals are quoted, and we mitigate the assumption in two ways — by choosing the least-committal response distribution for the primary analysis and by reporting a second, provably worst-case distribution alongside it. The accompanying software performs the ordinary nonparametric bootstrap instead, with no change to any other step, whenever an item-level response file is supplied; replacing the reconstruction by the real responses is the single change that would make these intervals empirical.

The resampling scheme. Each replicate draws, independently within each compartment, a sample of respondents of the same size with replacement; every subscale mean is then recomputed from the *same* resampled respondents and the rates re-derived through Eq. (3)–Eq. (4). Resampling respondents once per replicate, rather than each rate separately, matters because the sub-samples overlap: γ is estimated on $I \cup R$ and δ on R, so the two estimates share the twelve recovered respondents and are necessarily dependent. The scheme reproduces that dependence automatically, giving a correlation of 0.85 between $\hat{\gamma}$ and $\hat{\delta}$; treating the marginals as independent, as a naive scheme would, misses it entirely. We use $B = 4{,}000$ replicates. For μ, which is administrative rather than questionnaire-derived, we draw the mean study duration $1/\mu$ by stratified sampling of the range $[3.5, 5.0]$ yr.

The two response distributions. The primary analysis assigns each compartment the maximum-entropy distribution on $\{1, \dots, 5\}$ with the observed mean, which is the least-committal reconstruction given that only the mean is known [23]. As a bound we repeat everything with the two-point distribution on $\{1, 5\}$ with the same mean, which maximises the response variance and therefore the width of every interval; no reconstruction consistent with the observed means can give wider ones. That bound is also instructive, since it has $\mathrm{Var}(q) = 16\,p(1-p)$ with $p = (\bar{q} - 1)/4$ and hence $\mathrm{SE}(\hat{p}) = \sqrt{p(1-p)/n}$, exactly the binomial standard error: a binomial sampling model is recovered as the extreme, maximal-dispersion case of the present procedure. Reporting both distributions means the reader sees the range within which the reconstruction assumption can move the answer, and Subsection 3.7 shows that the qualitative verdict does not move at all. One further caveat: because a reconstructed panel of n_X integer scores matches a prescribed mean only to within $1/n_X$, the resampling distributions are centred within about two percent of the point estimates of Table 2.

Propagation. We report the induced distribution of $\mathcal{R}_0$, of the peak addicted head-count, and of the ten-year cumulative burden $N_p \int_0^T i\,dt$ in addicted student-years. Partial rank correlation coefficients (PRCC) between each input and each output measure monotone influence after

controlling for the remaining inputs, with significance from the associated t statistic on $B-k-1$ degrees of freedom. Finally, the full optimal-control problem is re-solved for 200 of the bootstrap parameter vectors, so that the *benefit of intervention* is itself reported as a distribution rather than as a single number [24].

2.8. Numerical Implementation

The state system is integrated forward by the classical fourth-order Runge–Kutta method, the adjoint system backward from its terminal condition by the same scheme, and the pair is coupled by the forward–backward sweep of Lenhart and Workman [21]. Starting from $u_1 \equiv u_2 \equiv 0$ on the uniform grid $t_k = kh$, $h = T/M$, each iteration integrates Eq. (2) forward from $x(0)$, integrates Eq. (11) backward from $\lambda(T) = 0$, evaluates the projected minimisers Eq. (12), and updates each control by the convex combination $u_k \leftarrow \omega\tilde{u}_k + (1-\omega)u_k$; iteration stops when the relative ℓ^1 increment $\sum_k \|\Delta u_k\|_1 / \sum_k \|u_k\|_1$ falls below tol. The relaxation is what makes the sweep converge here: the pure update ($\omega = 1$) oscillates.

The settings needed to reproduce every result are as follows: uniform grid with $M = 4{,}000$, so $h = 2.5 \times 10^{-3}$ yr; relaxation $\omega = 0.3$; stopping tolerance tol $= 10^{-4}$, reached in 14–25 iterations; terminal conditions $\lambda_j(T) = 0$; $B = 4{,}000$ resampling replicates and 200 re-solved control problems under parameter uncertainty, all from the fixed seed 20260816; and the weight and bound sweeps of Subsection 3.8 covering a 32-fold range in b_1 and b_2 and a fourfold range in the two bounds. Two convergence checks were run against the finest grid $M = 8{,}000$: the objective changes by 3.3×10^{-8} in relative terms at $M = 4{,}000$ and by only 2.7×10^{-6} at the coarsest grid $M = 500$, so the reported values are not grid artefacts; and the converged objective agrees to six significant figures across $\omega \in \{0.1, 0.2, 0.3, 0.5, 0.8\}$ even though the iteration count varies from 14 to 64. All computations used Python 3.11 with NumPy, SciPy, and Matplotlib; the complete script accompanies this article.

3. Results and Discussion

This section presents the analytical and numerical results of the proposed model, followed by a discussion of their implications for the dynamics and control of online shopping addiction.

3.1. Well-Posedness and Invariant Region

Proposition 1 (Positivity and invariance)**.** *The simplex* $\Omega = \{(s,e,i,r) \in \mathbb{R}^4_+ : s+e+i+r = 1\}$ *is positively invariant for Eq. (2) for every measurable control pair in* $\mathcal{U}$*, and solutions starting in* Ω *exist and are unique for all* $t \geq 0$*.*

Proof. The right-hand side of Eq. (2) is polynomial in the state and affine in the controls, hence locally Lipschitz in the state uniformly in $(u_1, u_2) \in \mathcal{U}$, so local existence and uniqueness follow from Picard–Lindelöf. Summing the four equations gives $d(s+e+i+r)/dt = \mu(1-(s+e+i+r))$, so the hyperplane $s+e+i+r = 1$ is invariant. For positivity, evaluate each vector field component on the corresponding face: $ds/dt|_{s=0} = \mu + \delta r \geq \mu > 0$; $de/dt|_{e=0} = (1-u_1)\beta si \geq 0$; $di/dt|_{i=0} = \sigma e \geq 0$; and $dr/dt|_{r=0} = (\gamma + u_2)i \geq 0$. Hence the vector field never points out of $\mathbb{R}^4_+$ and Ω is positively invariant. Boundedness on Ω then gives global existence. □

3.2. Equilibria, Threshold, and Bifurcation Structure

Setting the derivatives of the uncontrolled Eq. (2) to zero, the addiction-free equilibrium is $\mathcal{E}_0 = (1,0,0,0)$, corresponding to $(N_p, 0, 0, 0) = (3{,}028,\ 0,\ 0,\ 0)$ students. Taking (e,i) as the

infected subsystem and linearising at $\mathcal{E}_0$ gives new-infection and transition matrices

$$\mathcal{F} = \begin{pmatrix} 0 & \beta \\ 0 & 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} \sigma + \mu & 0 \\ -\sigma & \gamma + \mu \end{pmatrix},$$

whence

$$\mathcal{R}_0 = \rho\left(\mathcal{F}\mathcal{V}^{-1}\right) = \frac{\beta\sigma}{(\sigma+\mu)(\gamma+\mu)}. \tag{6}$$

The factor $\sigma/(\sigma+\mu)$ is the probability that an exposed student progresses to addiction before leaving the faculty, and $1/(\gamma+\mu)$ is the mean time spent addicted; graduation therefore acts as a genuine removal mechanism, which is why μ appears in both denominators.

Proposition 2 (Existence and uniqueness of the endemic equilibrium)**.** *For all positive $\beta, \sigma, \gamma, \delta, \mu$, system Eq. (2) with $u_1 = u_2 = 0$ possesses a unique endemic equilibrium $\mathcal{E}^* = (s^*, e^*, i^*, r^*) \in \operatorname{int}\Omega$ if and only if $\mathcal{R}_0 > 1$, given by*

$$s^* = \frac{1}{\mathcal{R}_0}, \qquad e^* = \frac{(\gamma+\mu)\, i^*}{\sigma}, \qquad r^* = \frac{\gamma\, i^*}{\mu+\delta}, \qquad i^* = \frac{\mu\,(1 - 1/\mathcal{R}_0)}{D}, \tag{7}$$

where $D = \dfrac{(\sigma+\mu)(\gamma+\mu)}{\sigma} - \dfrac{\delta\gamma}{\mu+\delta} > 0$. Moreover i^* is continuous and strictly increasing in $\mathcal{R}_0$ with $i^* \to 0^+$ and $\mathcal{E}^* \to \mathcal{E}_0$ as $\mathcal{R}_0 \to 1^+$, so the endemic branch emerges continuously from the addiction-free equilibrium into the interior of Ω.

Proof. From $di/dt = 0$ we get $e = (\gamma+\mu)i/\sigma$, and from $dr/dt = 0$ we get $r = \gamma i/(\mu+\delta)$. Substituting into $de/dt = 0$ with $i > 0$ gives $\beta s = (\sigma+\mu)(\gamma+\mu)/\sigma$, i.e. $s^* = 1/\mathcal{R}_0$. Adding $ds/dt = 0$ and $de/dt = 0$ and using $\Lambda = \mu$ yields $\mu(1-s) + \delta r = (\sigma+\mu)e$, and substituting the expressions for e and r gives $\mu(1-s^*) = D\, i^*$.
Positivity of D holds for all positive parameters: since $(\sigma+\mu)/\sigma > 1$ we have $(\sigma+\mu)(\gamma+\mu)/\sigma > \gamma+\mu > \gamma$, while $\delta\gamma/(\mu+\delta) < \gamma$ because $\delta/(\mu+\delta) < 1$; hence $D > \gamma - \gamma = 0$. Therefore $i^* > 0$ if and only if $1 - s^* = 1 - 1/\mathcal{R}_0 > 0$, that is, if and only if $\mathcal{R}_0 > 1$; and then $e^*, r^* > 0$ and $s^* \in (0,1)$, so $\mathcal{E}^* \in \operatorname{int}\Omega$ and is unique because each component is determined by i^*, which solves a linear equation. Finally, $\mathcal{R}_0 \mapsto i^* = \mu(1 - 1/\mathcal{R}_0)/D$ is continuous and strictly increasing on $(1, \infty)$ with $i^* \to 0^+$ as $\mathcal{R}_0 \to 1^+$, and the remaining components are positive multiples of i^*, so $\mathcal{E}^* \to \mathcal{E}_0$ in that limit. □

Remark 1. Proposition 2 establishes that a positive equilibrium branch exists precisely on the far side of the threshold and emerges continuously from $\mathcal{E}_0$. On its own this is a statement about equilibria, not a classification of the bifurcation; the classification requires the non-degeneracy conditions and is proved separately as Theorem 3, after the spectral information supplied by Theorem 1 is available.

3.3. Stability of the Addiction-Free Equilibrium

The Jacobian of the uncontrolled Eq. (2) is

$$J(s,e,i,r) = \begin{pmatrix} -\beta i - \mu & 0 & -\beta s & \delta \\ \beta i & -(\sigma+\mu) & \beta s & 0 \\ 0 & \sigma & -(\gamma+\mu) & 0 \\ 0 & 0 & \gamma & -(\mu+\delta) \end{pmatrix}. \tag{8}$$

Theorem 1 (Local stability, sharp threshold). *$\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$, for all positive parameter values.*

Proof. At $\mathcal{E}_0 = (1, 0, 0, 0)$ the first column of Eq. (8) is $(-\mu, 0, 0, 0)^\top$ and the last column is $(\delta, 0, 0, -(\mu + \delta))^\top$, so $J(\mathcal{E}_0)$ is block triangular with eigenvalues $\xi_1 = -\mu < 0$, $\xi_2 = -(\mu + \delta) < 0$, and the roots of

$$\xi^2 + (\sigma + \gamma + 2\mu)\,\xi + (\sigma + \mu)(\gamma + \mu)\,(1 - \mathcal{R}_0) = 0. \tag{9}$$

The coefficient of ξ is positive, so by the Routh–Hurwitz criterion both roots have negative real part if and only if the constant term is positive, that is, if and only if $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$ the constant term is negative and Eq. (9) has one positive and one negative real root, so $\mathcal{E}_0$ is a saddle. □

Theorem 2 (Global stability when $\mathcal{R}_0 \le 1$). *If $\mathcal{R}_0 \le 1$ then $\mathcal{E}_0$ is globally asymptotically stable in Ω.*

Proof. By Proposition 1 the set Ω is compact and positively invariant, so LaSalle's invariance principle [18] applies on Ω. Let $L(e, i) = \sigma e + (\sigma + \mu)i$, so that $L \ge 0$ on Ω with $L = 0$ if and only if $e = i = 0$. Along solutions of the uncontrolled Eq. (2),

$$\dot{L} = \sigma\,[\beta s i - (\sigma + \mu)e] + (\sigma + \mu)\,[\sigma e - (\gamma + \mu)i] = (\sigma + \mu)(\gamma + \mu)\,i\,(\mathcal{R}_0\, s - 1)\,. \tag{10}$$

Since $0 \le s \le 1$ on Ω and $\mathcal{R}_0 \le 1$, we have $\mathcal{R}_0 s - 1 \le \mathcal{R}_0 - 1 \le 0$, hence $\dot{L} \le 0$ on Ω.
Step 1: identification of $\{\dot{L} = 0\}$. By Eq. (10), $\dot{L} = 0$ if and only if $i = 0$ or $\mathcal{R}_0 s = 1$. In the second case $\mathcal{R}_0 \le 1$ and $s \le 1$ force $\mathcal{R}_0 = 1$ and $s = 1$, and then $e = i = r = 0$ because the components are nonnegative and sum to one; that point is $\mathcal{E}_0$, which also has $i = 0$. Therefore

$$E := \left\{x \in \Omega : \dot{L}(x) = 0\right\} = \{x \in \Omega : i = 0\}\,.$$

Step 2: the largest invariant subset of E. This is the step that requires care. The slice $\{(s, 0, 0, r) \in \Omega\}$ lies entirely in E and is forward invariant, so observing that trajectories in E satisfy $i \equiv 0$ does not by itself locate $\mathcal{E}_0$; one must use that invariance in LaSalle's principle means invariance in *both* time directions. Let $\mathcal{M} \subseteq E$ be the largest invariant set and let $x(\cdot)$ be a solution with $x(t) \in \mathcal{M}$ for all $t \in \mathbb{R}$. Then $i \equiv 0$, so $0 = \dot{i} = \sigma e$ and hence $e \equiv 0$. The fourth equation then reduces to $\dot{r} = -(\mu + \delta)r$, so that

$$r(t) = r(0)\,e^{-(\mu+\delta)t}, \qquad t \in \mathbb{R}.$$

If $r(0) > 0$ then $r(t) \to +\infty$ as $t \to -\infty$, contradicting $r(t) \le 1$ for all t, which holds because $x(t) \in \Omega$. Hence $r \equiv 0$, and therefore $s \equiv 1$. Thus $\mathcal{M} = \{\mathcal{E}_0\}$, and LaSalle's principle gives $x(t) \to \mathcal{E}_0$ for every solution starting in Ω.
Step 3: Lyapunov stability. Global attractivity alone does not give stability, and L does not control the r component, so we argue directly. Let $\eta > 0$ and suppose $e(0) + i(0) \le \eta$ and $r(0) \le \eta$. Since $\sigma < \sigma + \mu$ we have $L(0) \le (\sigma + \mu)\eta$, and $\dot{L} \le 0$ gives $L(t) \le L(0)$ for $t \ge 0$, whence

$$e(t) \le \frac{\sigma + \mu}{\sigma}\,\eta, \qquad i(t) \le \eta \qquad (t \ge 0).$$

Feeding this into $\dot{r} = \gamma i - (\mu + \delta)r \le \gamma\eta - (\mu + \delta)r$ and applying the comparison lemma,

$$r(t) \le r(0)\,e^{-(\mu+\delta)t} + \frac{\gamma\eta}{\mu + \delta} \le \eta\left(1 + \frac{\gamma}{\mu + \delta}\right) \qquad (t \ge 0),$$

and finally $|s(t)-1| = e(t)+i(t)+r(t) \le C\eta$ with $C = (\sigma+\mu)/\sigma + 1 + 1 + \gamma/(\mu+\delta)$. Every component is therefore bounded by a fixed multiple of η, so for any prescribed neighbourhood of $\mathcal{E}_0$ a sufficiently small η keeps the whole trajectory inside it. This is Lyapunov stability, which together with Step 2 gives global asymptotic stability. □

Theorem 3 (Forward transcritical bifurcation at $\mathcal{R}_0 = 1$)**.** *Take β as the bifurcation parameter and let $\beta^* = (\sigma+\mu)(\gamma+\mu)/\sigma$ be its value at $\mathcal{R}_0 = 1$. Then the uncontrolled system Eq. (2) undergoes a transcritical bifurcation at $\mathcal{E}_0$ as β crosses β^*, and the bifurcation is forward (supercritical): for β slightly below β^* the addiction-free equilibrium is locally asymptotically stable and no positive equilibrium exists, whereas for β slightly above β^* it is unstable and a locally asymptotically stable endemic equilibrium exists nearby. This holds for all positive $\sigma, \gamma, \delta, \mu$.*

Proof. We verify the hypotheses of Theorem 4.1 of Castillo-Chavez and Song [25]. Put $x = (x_1, x_2, x_3, x_4) = (s-1, e, i, r)$, so that the uncontrolled Eq. (2) becomes $\dot{x} = f(x, \beta)$ with

$$f_1 = -\beta(1+x_1)x_3 - \mu x_1 + \delta x_4, \qquad f_3 = \sigma x_2 - (\gamma+\mu)x_3,$$
$$f_2 = \beta(1+x_1)x_3 - (\sigma+\mu)x_2, \qquad f_4 = \gamma x_3 - (\mu+\delta)x_4,$$

and $f(0,\beta) = 0$ for every β, so $x = 0$ is an equilibrium for all values of the parameter.
Spectrum. $A := D_x f(0, \beta^*)$ is the matrix Eq. (8) evaluated at $\mathcal{E}_0$ with $\beta = \beta^*$. By the factorisation used in the proof of Theorem 1, its eigenvalues are $-\mu$, $-(\mu+\delta)$ and the roots of Eq. (9), whose constant term $(\sigma+\mu)(\gamma+\mu)(1-\mathcal{R}_0)$ vanishes at $\beta = \beta^*$; those roots are 0 and $-(\sigma+\gamma+2\mu)$. Hence A has a simple zero eigenvalue and three real negative eigenvalues, as the theorem requires.
Null vectors. Direct computation gives a right null vector w and a left null vector v of A,

$$w = \left(-\frac{D}{\mu},\ \frac{\gamma+\mu}{\sigma},\ 1,\ \frac{\gamma}{\mu+\delta}\right)^{\top}, \qquad v = \left(0,\ \frac{\sigma}{\sigma+\mu},\ 1,\ 0\right),$$

with D as in Proposition 2; in particular $w_1 = -D/\mu < 0$. Since $v \cdot w = (\sigma+\gamma+2\mu)/(\sigma+\mu) > 0$, we may rescale v so that $v \cdot w = 1$, which replaces v_2 by $\sigma/(\sigma+\gamma+2\mu)$.
Bifurcation coefficients. The components f_3 and f_4 are linear, and $v_1 = v_4 = 0$, so the only second derivative that contributes is $\partial^2 f_2/\partial x_1 \partial x_3 = \beta$. Therefore

$$a = \sum_{k,i,j} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, \beta^*) = 2\beta^* v_2 w_1 w_3 = -\frac{2(\sigma+\mu)(\gamma+\mu)\,D}{\mu\,(\sigma+\gamma+2\mu)},$$

using $\beta^*\sigma = (\sigma+\mu)(\gamma+\mu)$, and

$$b = \sum_{k,i} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(0, \beta^*) = v_2 w_3 = \frac{\sigma}{\sigma+\gamma+2\mu}.$$

Because $D > 0$ for all positive parameters by Proposition 2, we have $a < 0$ and $b > 0$ unconditionally. Theorem 4.1 of [25] then yields a forward transcritical bifurcation at $\beta = \beta^*$ with the stated exchange of stability. At the estimated parameter values, $a = -2.3194$ and $b = 0.1837$. □

Remark 2. Two consequences of Theorem 3 are worth isolating. First, the sign of a is governed entirely by D, the same quantity that controls existence of the endemic equilibrium

in Proposition 2; since $D > 0$ always, the model admits no backward bifurcation, so $\mathcal{R}_0 < 1$ is sufficient for elimination and no subcritical endemic branch can coexist with a stable $\mathcal{E}_0$. Second, this is a structural property of the compartmental scheme rather than of the estimated parameters: it would fail if a direct relapse route $R \to I$ were added, which is one reason we flag that extension in the limitations. We do not claim a corresponding global result for $\mathcal{E}^*$. At the estimated parameter values the eigenvalues of $J(\mathcal{E}^*)$ are -0.9826, -0.3982, -0.2500, and -0.0066, all negative, so $\mathcal{E}^*$ is locally asymptotically stable *at this parameter set*; we make no assertion beyond it. Note the smallest eigenvalue in modulus: the associated relaxation time is $1/0.0066 \approx 150$ years. The endemic equilibrium is thus a mathematical reference point rather than a state the faculty would ever be observed in, which is the main reason the analysis below emphasises transient quantities.

3.4. Baseline Dynamics at the Point Estimates

Substituting Table 2 into Eq. (6) gives

$$\mathcal{R}_0 = \frac{(1.35)(0.18)}{(0.43)(0.55)} = \frac{0.2430}{0.2365} \approx 1.0275,$$

and Eq. (7) gives $D = 1.2014$ and

$$\mathcal{E}^* = (0.97325,\ 0.01701,\ 0.00557,\ 0.00417),$$

that is (2947.0, 51.5, 16.9, 12.6) students out of 3,028, equivalently (95.38, 1.67, 0.55, 0.41) per 98 students. Consistently with Theorem 1, the eigenvalues at $\mathcal{E}_0$ are -0.9866, -0.4000, -0.2500, and $+0.0066$, so $\mathcal{E}_0$ is unstable.

Fig. 2 shows the resulting trajectories in head-counts, and Table 3 summarises them. The uncontrolled trajectory deserves comment. Starting from the surveyed state, the addicted population *rises* from 93 to a peak of 193 students at $t = 2.32$ yr and then declines slowly, reaching 129 students at ten years and approaching the endemic level of 17 students only over the 150-year timescale noted above. In other words, the model does not predict unbounded growth of addiction; it predicts a pronounced multi-year transient surge driven by the large exposed pool already present at baseline ($E_0 = 1020$ students, a third of the faculty), followed by a very slow relaxation. The policy-relevant object is that transient, not the asymptotic state, and the correct reading of $\mathcal{R}_0 > 1$ here is that addiction does not self-extinguish, not that it grows without bound.

3.5. Optimal Control: Existence and Characterisation

Theorem 4 (Existence). *There exists an optimal pair $(u_1^*, u_2^*) \in \mathcal{U}$ minimising Eq. (5) subject to Eq. (2).*

Proof. We verify the hypotheses of the Fleming–Rishel existence theorem [26, Thm. III.4.1]. The set of admissible controls and corresponding state trajectories is nonempty, since $u_1 = u_2 = 0$ is admissible and, by Proposition 1, produces a bounded solution. The control set $\mathcal{U}$ is closed and convex, being a product of intervals. The right-hand side of Eq. (2) is bounded by a linear function of the state on Ω and is affine, hence continuous, in (u_1, u_2) for each fixed state. The integrand $A_1 i + \frac{1}{2} b_1 u_1^2 + \frac{1}{2} b_2 u_2^2$ is convex in (u_1, u_2) and satisfies the coercivity bound $A_1 i + \frac{1}{2} b_1 u_1^2 + \frac{1}{2} b_2 u_2^2 \geq c_1 \left(u_1^2 + u_2^2\right)^1 - c_2$ with $c_1 = \frac{1}{2} \min(b_1, b_2) > 0$ and $c_2 = 0$. The conclusion follows. □

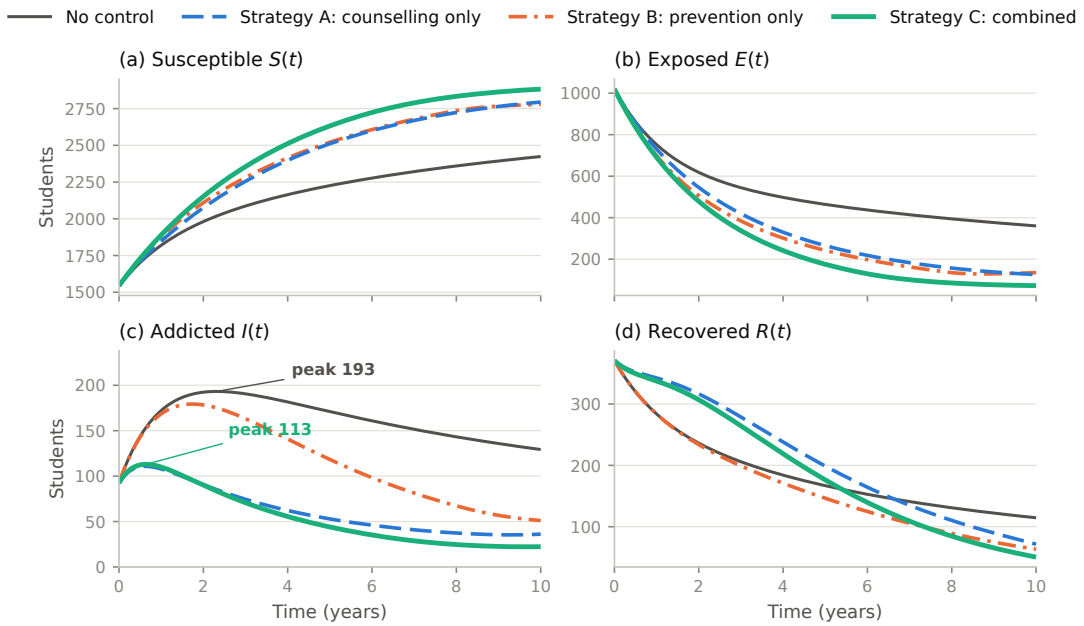

Fig. 2: Trajectories of the four compartments in head-counts for $N_p = 3{,}028$ over the ten-year horizon, without control and under the three intervention strategies. Line style and weight duplicate the colour coding so that the panels remain readable in greyscale. Panel (c) carries the main message: prevention alone (Strategy B) barely displaces the transient peak, because that peak is fed by students who were already exposed at $t = 0$ and whom prevention cannot reach.

Theorem 5 (Characterisation). *Let (u_1^*, u_2^*) be optimal with associated states (s, e, i, r). Then there exist adjoint variables $\lambda_1, \ldots, \lambda_4$ satisfying*

$$\begin{aligned}
\frac{d\lambda_1}{dt} &= (1 - u_1^*)\,\beta i\,(\lambda_1 - \lambda_2) + \mu\lambda_1, \\
\frac{d\lambda_2}{dt} &= (\sigma + \mu)\,\lambda_2 - \sigma\lambda_3, \\
\frac{d\lambda_3}{dt} &= -A_1 + (1 - u_1^*)\,\beta s\,(\lambda_1 - \lambda_2) + (\gamma + \mu + u_2^*)\,\lambda_3 - (\gamma + u_2^*)\,\lambda_4, \\
\frac{d\lambda_4}{dt} &= (\mu + \delta)\,\lambda_4 - \delta\lambda_1,
\end{aligned} \tag{11}$$

with transversality conditions $\lambda_j(T) = 0$, $j = 1, \ldots, 4$, and the optimal controls satisfy

$$\begin{aligned}
u_1^* &= \min\left\{u_{1\max},\ \max\left\{0,\ \frac{\beta s i\,(\lambda_2 - \lambda_1)}{b_1}\right\}\right\}, \\
u_2^* &= \min\left\{u_{2\max},\ \max\left\{0,\ \frac{i\,(\lambda_3 - \lambda_4)}{b_2}\right\}\right\}.
\end{aligned} \tag{12}$$

Proof. The Hamiltonian associated with Eq. (5) and Eq. (2) is

$$\begin{aligned}
H = {} & A_1 i + \tfrac{1}{2} b_1 u_1^2 + \tfrac{1}{2} b_2 u_2^2 + \lambda_1(\mu - (1 - u_1)\beta s i - \mu s + \delta r) + \lambda_2((1 - u_1)\beta s i - (\sigma + \mu)e) \\
& + \lambda_3(\sigma e - (\gamma + \mu)i - u_2 i) + \lambda_4((\gamma + u_2)i - (\mu + \delta)r)\,.
\end{aligned}$$

Pontryagin's minimum principle [21, 27] gives $d\lambda_j/dt = -\partial H/\partial x_j$ with $x = (s, e, i, r)$, which is Eq. (11), and $\lambda_j(T) = 0$ since the terminal state is free. On the interior of $\mathcal{U}$ the

stationarity conditions are

$$\frac{\partial H}{\partial u_1} = b_1 u_1 + \beta s i\,(\lambda_1 - \lambda_2) = 0, \qquad \frac{\partial H}{\partial u_2} = b_2 u_2 - i\,(\lambda_3 - \lambda_4) = 0,$$

giving the unconstrained minimisers in Eq. (12); projecting onto $[0, u_{k\max}]$ accounts for the bounds in the standard way. The Hessian of H in the controls is $\mathrm{diag}(b_1, b_2) \succ 0$, so H is strictly convex in (u_1, u_2) and these stationary points are minimisers. □

Remark 3. The two optimality conditions in Eq. (12) have a transparent reading. Prevention effort is proportional to the current incidence $\beta s i$ multiplied by the shadow-price gap $\lambda_2 - \lambda_1$ between an exposed and an at-risk student; counselling effort is proportional to the addicted head-count multiplied by the gap $\lambda_3 - \lambda_4$ between an addicted and a recovered student. Both vanish at $t = T$ because the adjoints do, which is why the profiles in Fig. 3 decay to zero at the horizon. Under the scaling $B_k = b_k N_p$ of Subsection 2.6, both expressions are invariant under a change of population size, consistently with Lemma 1.

3.6. Comparison of Intervention Strategies

We compare three strategies: A applies counselling only ($u_1 \equiv 0$), B applies prevention only ($u_2 \equiv 0$), and C applies both. Results are in Table 3, with trajectories in Fig. 2 and control profiles in Fig. 3.

Table 3: Outcomes over the ten-year horizon, head-counts for $N_p = 3{,}028$. Burden is the cumulative number of addicted student-years, $N_p \int_0^T i\,dt$. Reductions are relative to the uncontrolled trajectory.

Strategy	Peak I (students)	Peak t (yr)	Peak red. (%)	Burden (student-yr)	Burden red. (%)	J	Iter.
No control	193.2	2.32	—	1621.9	—	0.5356	—
A: counselling	110.8	0.63	42.7	617.4	61.9	0.2754	14
B: prevention	179.3	1.74	7.2	1155.9	28.7	0.3967	25
C: combined	113.0	0.64	41.5	544.9	66.4	0.2435	23

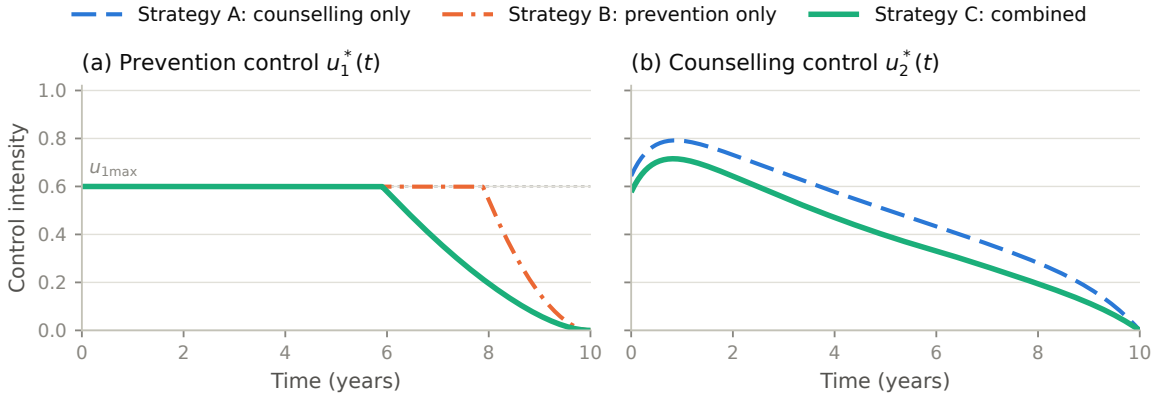

Fig. 3: Optimal control profiles obtained by the forward–backward sweep. Prevention rides its upper bound $u_{1\max} = 0.6$ for most of the horizon before decaying to zero, whereas counselling is front-loaded, peaking at 0.72–0.79 yr^{-1} within the first year while the addicted class is largest. Both decay to zero at $t = T$ in accordance with the transversality conditions.

Three findings deserve emphasis, and the second of them speaks directly to a concern raised in review.

First, the combined strategy C attains the lowest objective value, $J = 0.2435$ against 0.2754 for A and 0.3967 for B, and the largest reduction in cumulative burden, 66.4%. It is not uniformly

best on every metric — A achieves a marginally lower peak, 110.8 against 113.0 students — which is expected rather than anomalous, since the optimiser minimises Eq. (5) over the whole trajectory and both programme costs, not the peak alone.

Second, prevention alone reduces the peak by only 7.2% while counselling alone reduces it by 42.7%, even though prevention reduces cumulative burden appreciably (28.7%). The reason is structural: at baseline a third of the faculty is already exposed, and the transient peak in I is produced almost entirely by that cohort progressing through $E \to I$. Prevention acts on $S \to E$ and cannot reach students who have already made that transition; it can only reduce the inflow sustaining the later trajectory. The sensitivity analysis below confirms this independently, with the progression rate σ rather than the transmission rate β dominating the peak. Interventions targeting incoming students and those targeting currently addicted students are therefore not substitutes on the same timescale: prevention is a multi-year investment, counselling is what moves the near-term peak. The two levers are therefore complementary rather than interchangeable.

Third, the shapes in Fig. 3 are policy-legible. Counselling should be deployed most intensively in the first two years, when I is largest, and tapered thereafter; prevention should be sustained near its feasible maximum throughout, because its benefit accrues through the cumulative avoided inflow rather than through an immediate effect.

3.7. Uncertainty Quantification and Global Sensitivity

Fig. 4(a) shows the resampling distribution of $\mathcal{R}_0$. As set out in Subsection 2.7, these intervals are model-based: they propagate the sampling variability implied by a reconstructed response distribution with the observed subscale mean, not variability read off the individual responses. Under the primary response model the median is 1.05 with a 95% interval of [0.50, 1.97], and

$$P(\mathcal{R}_0 > 1) = 0.558.$$

Under the maximal-dispersion bound the interval widens to [0.24, 2.71] with median 0.98 and $P(\mathcal{R}_0 > 1) = 0.489$. The conclusion is the same either way, and it must be stated without varnish: *the data do not determine on which side of the threshold the system lies.* The point estimate $\mathcal{R}_0 \approx 1.03$ conveys false precision, and a conclusion of the form "since $\mathcal{R}_0 > 1$, addiction will persist" is not supported by a sample of 98 students containing three addicted respondents. That the two response models bracket the threshold from opposite sides — $P(\mathcal{R}_0 > 1)$ moves from 0.56 to 0.49 as the assumed dispersion is pushed to its maximum — is itself the clearest possible statement of the situation: the sample locates the system *at* the threshold and cannot place it on either side of it.

The corresponding transient quantities are far better determined: the peak addicted head-count has median 202 with 95% interval [121, 391] students, and the ten-year burden has median 1713 with interval [856, 3283] student-years. Under every draw the burden is substantial. This asymmetry — a threshold that cannot be resolved but a transient burden that is unambiguously large — is what justifies basing the policy discussion on the transient.

Table 4 reports both the local normalised sensitivity indices of $\mathcal{R}_0$ and the global PRCC values for three outputs. The two agree on $\mathcal{R}_0$: β has local index exactly $+1$ and PRCC $+0.93$, γ and μ are negative in both, and δ has no influence, consistent with its absence from Eq. (6).

The interesting divergence is between the outputs. For $\mathcal{R}_0$ the ranking is β (0.93) $>$ σ (0.85) $>$ μ (-0.75) $>$ γ (-0.68), whereas for the peak addicted head-count it is σ (0.95) $>$ γ (-0.70) $>$ μ (-0.68) $>$ β (0.60). The progression rate overtakes the transmission rate as soon as attention shifts from the asymptotic threshold to the transient, which is the quantitative counterpart of the structural argument given in the previous subsection: what drives the peak is how fast the existing exposed pool converts to addiction, not how fast new students are recruited into exposure. Any policy inference drawn from $\mathcal{R}_0$ sensitivities alone would have missed this.

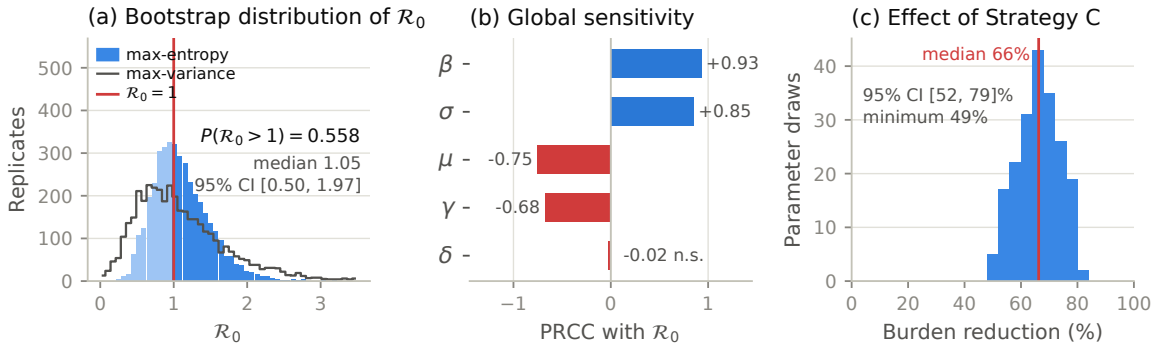

Fig. 4: (a) Parametric respondent-level resampling distribution of $\mathcal{R}_0$ ($B = 4{,}000$ replicates over reconstructed scores, hence model-based rather than empirical): filled bars use the maximum-entropy response model, the outline the maximum-variance bound. The threshold falls inside the bulk of both. (b) Partial rank correlation coefficients of $\mathcal{R}_0$ with each input; all significant at the one percent level except δ, which does not enter Eq. (6). (c) Reduction in ten-year cumulative burden under Strategy C when the control problem is re-solved for 200 bootstrap parameter vectors.

Two caveats apply. First, $\hat{\gamma}$ and $\hat{\delta}$ are correlated at 0.85 across replicates because they are estimated on overlapping respondents, and a partial correlation is hard to interpret when two inputs are nearly collinear; the non-significant PRCC of δ should be read as "no influence identifiable once γ is controlled for", which for $\mathcal{R}_0$ is exactly what Eq. (6) predicts. Second, PRCC values depend on the input ranges, so they measure influence over the uncertainty the data actually leave rather than intrinsic model sensitivity; that is why μ, whose local index is largest in magnitude, ranks below β and σ globally.

Table 4: Local normalised sensitivity indices of $\mathcal{R}_0$ and global partial rank correlation coefficients (PRCC) over the $B = 4{,}000$ resampling replicates, for three model outputs. All PRCC values are significant at $p < 0.01$ except those marked n.s.

Parameter	$\Upsilon_p^{\mathcal{R}_0}$ (local)	PRCC with $\mathcal{R}_0$	PRCC with peak I	PRCC with burden
β	$+1.000$	$+0.931$	$+0.601$	$+0.805$
σ	$+0.581$	$+0.852$	$+0.954$	$+0.941$
γ	-0.545	-0.676	-0.704	-0.756
μ	-1.036	-0.753	-0.683	-0.754
δ	0	-0.023 (n.s.)	-0.018 (n.s.)	$+0.000$ (n.s.)
Λ	0	—	—	—

3.8. Robustness of the Control Result

All four judgement-based quantities of Subsection 2.6 — the two cost weights, the two control bounds — and the horizon are varied here, together with the parameters themselves.

Counselling weight and bound. Fig. 5(a) varies b_2 over a 32-fold range and $u_{2\max}$ over a fourfold range. The peak reduction stays between 21.9% and 48.2%: it never approaches zero and never exceeds one half. The response is monotone and saturating in both directions, as it should be, since making counselling cheaper or raising its ceiling improves the outcome with diminishing returns.

Prevention weight and bound. The same question applies to b_1 and $u_{1\max}$, which are judgements of exactly the same kind. Fig. 5(b) varies b_1 over a 32-fold range and $u_{1\max}$ over a fourfold range and reports the reduction in cumulative burden, which is the outcome prevention actually moves. It stays between 62.9% and 66.6%, a spread of under four percentage points, while the peak reduction stays between 41.5% and 42.4%. Prevention is thus the *least* sensitive part of the

design, for a reason visible in Fig. 3(a): u_1^* rides its upper bound over most of the horizon at every weight tested, so b_1 influences only when the control peels off the bound near $t = T$, and lowering $u_{1\max}$ removes a benefit that accrues slowly in any case. We also checked the ranking of the three strategies at the four corners of that grid, $b_1 \in \{0.0025, 0.08\}$ and $u_{1\max} \in \{0.15, 0.60\}$: Strategy C attains the lowest objective in all four, with J between 0.2331 and 0.2700 against 0.2754 for Strategy A and between 0.3850 and 0.5019 for Strategy B. Neither the magnitude of the benefit nor the ordering of the strategies depends on how these two quantities are set.

Horizon. Varying T leaves the peak reduction essentially unchanged — 40.8%, 41.5%, and 41.7% for $T = 4$, 10, and 20 years — because under control the peak occurs within the first year and is insensitive to where the horizon is placed. The burden reduction grows with the horizon (46.3%, 66.4%, 77.8%), since a longer programme accumulates more benefit. The choice $T = 10$ yr is therefore not load-bearing.

Parameters. The strongest statement is Fig. 4(c), which re-solves the entire optimal-control problem for 200 parameter vectors drawn from the resampling distribution of Subsection 2.7. The reduction in ten-year cumulative burden under Strategy C has median 66.3%, 95% interval [52.1%, 78.8%], and *minimum* 48.9% *across all* 200 *draws*; every draw exceeds a 30% reduction. The peak reduction is more variable (median 42.1%, interval [21.3%, 64.9%], minimum 6.3%) because in draws with small σ the uncontrolled trajectory has no pronounced peak to suppress. Taken together with $P(\mathcal{R}_0 > 1) = 0.558$, this yields the paper's central practical message: whether the system sits above the epidemic threshold cannot be determined from these data, but the benefit of intervention does not depend on resolving that question.

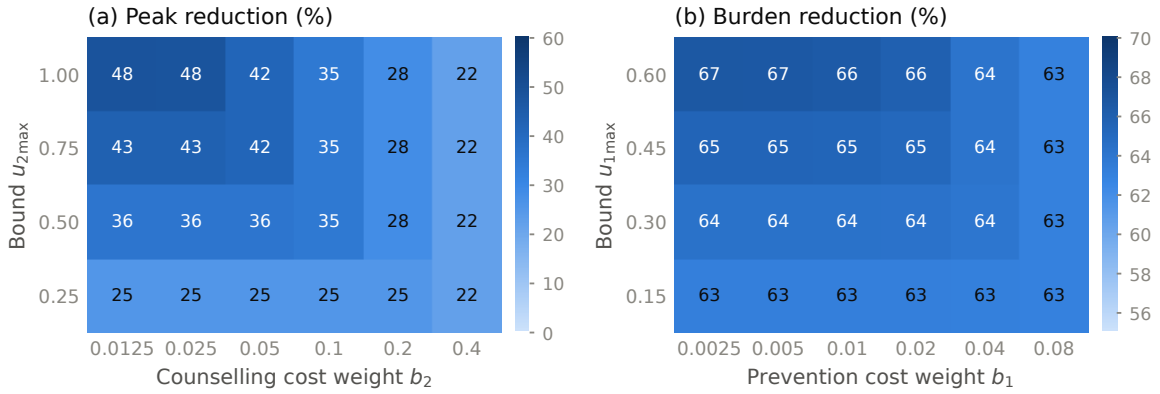

Fig. 5: Sensitivity of the optimal-control result to the judgement-based quantities of Subsection 2.6; cell values are percentages. (a) Peak reduction as the counselling cost weight b_2 and bound $u_{2\max}$ vary. (b) Reduction in ten-year cumulative burden as the prevention cost weight b_1 and bound $u_{1\max}$ vary; note the compressed colour range, the whole grid spanning under four percentage points.

3.9. Comparison with Previous Studies, Implications, and Limitations

Anwar [11] and Siregar and Panjaitan [12] both build SEIRS models of online *game* addiction with stability analysis and a reproduction number, and [11] also calibrates on primary questionnaire data, so none of those three ingredients is a point of novelty here. Girsang et al. [13] address online *shopping*, but without a threshold analysis, a bifurcation result, or control. The present study is distinguished by four things in combination: a bifurcation classification proved for all admissible parameters, a two-lever optimal-control problem, a formal uncertainty analysis carried through to the control result, and the application to online shopping rather than online gaming.

Implications. Three implications follow, each tied to a specific result rather than to the model in general. Because the transient peak is driven by the already-exposed cohort (Table 4, PRCC

of σ with peak I equal to 0.94), counselling capacity should be front-loaded rather than spread evenly, matching the profile in Fig. 3(b). Because prevention nonetheless reduces cumulative burden by 28.7% on its own and improves the combined objective, financial-literacy and digital-wellness content in new-student orientation is worth sustaining even though it will not visibly move the peak within the first two years; managing expectations about this lag is itself a policy recommendation. And because $\mathcal{R}_0$ cannot be resolved against its threshold, the case for intervention should be made on the burden — on the order of 1,600 addicted student-years over a decade at the point estimates — rather than on threshold rhetoric.

Limitations. The following limitations are substantive rather than formulaic. (i) A single cross-sectional survey does not identify the transition rates: the mapping Eq. (3)–Eq. (4) is a modelling assumption and the contact rate c in $\beta = cp_{SE}$ is assumed rather than measured, so the reported intervals understate the true uncertainty. Relatedly, the individual responses behind those intervals are reconstructed from the reported subscale means rather than observed, so they are model-based; archiving the item-level responses would turn the same computation into an ordinary nonparametric bootstrap and is the least costly improvement available, while a two-wave panel survey would identify the rates directly and is the most valuable one. (ii) The compartment sub-samples are small — three respondents addicted, twelve recovered — so γ and δ rest on very little data and are strongly correlated, which is the main reason $\mathcal{R}_0$ cannot be resolved and also limits how far the PRCC values can separate them. (iii) The model omits a direct relapse route $R \to I$; including it would permit a backward bifurcation, so that $\mathcal{R}_0 < 1$ would no longer guarantee elimination. (iv) Exponential sojourn times are a simplification for a process plausibly governed by cumulative exposure, and the parameters are constant whereas β almost certainly fluctuates with promotional events such as the 9.9 and 11.11 campaigns. (v) The data come from one faculty at one university and the classification is score-based rather than clinical, so head-counts should be read as counts of students meeting a screening threshold. (vi) The controls are continuous intensities with quadratic cost, whereas a real programme has fixed costs, capacity constraints, and discrete implementation levels.

4. Conclusion

We formulated an SEIRS model of online shopping addiction with two bounded controls and calibrated it with questionnaire data from 98 students of FMIPA Universitas Negeri Makassar. Writing the model in proportions makes its dynamics independent of population size, which resolves the relationship between the calibration sample and the target population of 3,028 students: the sample supplies the initial proportions and the parameters, and the population size converts proportions into head-counts.

Analytically, we proved that the feasible simplex is positively invariant, that the addiction-free equilibrium is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$ and globally asymptotically stable when $\mathcal{R}_0 \leq 1$, and that a unique endemic equilibrium exists exactly when $\mathcal{R}_0 > 1$. Verifying the Castillo-Chavez–Song conditions, whose coefficients we obtain in closed form as $a = -2(\sigma+\mu)(\gamma+\mu)D/[\mu(\sigma+\gamma+2\mu)] < 0$ and $b = \sigma/(\sigma+\gamma+2\mu) > 0$, shows the transition at $\mathcal{R}_0 = 1$ to be a forward transcritical bifurcation for all admissible parameters, so no backward bifurcation is possible in this model. Existence of an optimal control pair was established by the Fleming–Rishel theorem and the pair characterised by Pontryagin's minimum principle.

Numerically, the point estimates give $\mathcal{R}_0 \approx 1.03$, but a stratified parametric bootstrap over reconstructed respondent-level scores yields a 95% interval of $[0.50, 1.97]$ with $P(\mathcal{R}_0 > 1) = 0.558$, falling to 0.489 under a maximal-dispersion bound, so the threshold is not resolved by these data. The transient burden, by contrast, is unambiguous and large, and the benefit of intervention is robust: over ten years the combined strategy reduces the peak addicted population by 41.5% and cumulative addicted student-years by 66.4%; across 200 resampled parameter draws the burden reduction never falls below 48.9%, and across a 32-fold range of the prevention cost weight and a

fourfold range of its upper bound it varies by under four percentage points. Global sensitivity analysis shows that the progression rate σ, not the transmission rate β, dominates the transient peak, which explains why counselling reduces that peak by 42.7% while prevention reduces it by only 7.2%: the two levers act on different timescales and are complementary rather than interchangeable.

Future work should prioritise a two-wave panel survey to identify the transition rates directly, a larger multi-institution sample to sharpen γ, a direct relapse route with the associated backward-bifurcation analysis, and stochastic or seasonally forced formulations.

CRediT Authorship Contribution Statement

Muhammad Isbar Pratama: Conceptualization, Methodology, Formal Analysis, Software, Writing–Review & Editing, Supervision. **Wahidah Sanusi:** Methodology, Validation, Supervision. **Rhida Anggita Dasri:** Investigation, Data Curation, Writing–Original Draft, Visualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data and Code Availability

The questionnaire-derived parameters, initial values, and all numerical settings are reported in Table 1, Table 2, and Subsection 2.8.

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