

Mathematical Modeling and Equilibrium Analysis of Early Life Stress Using a Modified SEIR Framework

Abstract

Early life stress (ELS) exerts profound effects on brain development in children and mental health in later life. We formulate and solve a deterministic SEIR-type model to describe the effects of ELS on child populations in urban areas. The model involves environmental, genetic, neurobiological, and resilience factors with crucial dynamics and relapse. Equilibrium states are derived, and the local and global stability of the equilibriums are rigorously investigated. The model identifies significant patterns of progression of stress and provides a foundation for evaluating intervention approaches. Mathematical analysis reveals the Stress Propagation Index R_0 and shows that resilience and environmental exposure are key determinants of the dynamics of mental health. The findings are of relevance to mental health professionals, public health policy makers, educational and social services, and the general public.

Keywords: SEIR Model, Early Life Stress, Mental Health, Stability Analysis, Stress Propagation Index.

1 Introduction

Early life stress (ELS) is a major risk factor for adverse mental health outcomes. The timing and accumulation of stress during critical developmental periods can alter neurobiological systems, increasing vulnerability to psychiatric disorders such as anxiety disorders, depression, PTSD, and drug abuse [5] [2].

Over 60% Studies in Kenya have highlighted the high prevalence of adverse childhood experiences (ACEs) among Kenyan children and adolescents, with significant associations to mental health issues. Additionally, maternal mental health has been shown to mediate the relationship between maternal ACEs and child mental health problems, emphasizing the intergenerational impact of early life stress. Most of urban populations live in informal settlements where children are routinely exposed to chronic stressors such as food insecurity, poor housing conditions, exposure to violence and lack of access to mental health care. The Nairobi Urban Health and Demographic Surveillance System (NUHDSS) has

consistently reported high levels of psychosocial stress and limited mental health support structures in informal settlements [1].

Early life stress measurement employs a multi-faceted approach combining standardized assessment tools, biological markers, and behavioral observations to create a comprehensive understanding of stress exposure and its impacts. The assessment tools, including the Childhood Trauma Questionnaire (CTQ), Early Life Stress Inventory and Parental Bonding Instrument, provide structured evaluation of traumatic experiences, adverse childhood events, and parent-child relationship quality through validated questionnaires and interviews. These subjective measures are complemented by objective biological markers, where cortisol levels in hair or saliva samples offer insights into chronic stress exposure, heart rate variability measurements indicate stress response system development and inflammatory markers in blood samples reveal physiological stress effects at the cellular level.

Mathematical modeling provides a structured framework for understanding the transmission and persistence of stress effects within a population, integrating neuroimaging studies, longitudinal cohort data and psychological assessments [6]. However, there remains a gap in comprehensive models that holistically integrate genetic predispositions, environmental influences and psychosocial factors. This paper develops a modified SEIR model for ELS and analyzes its equilibrium behavior and stability. It contributes to the identification of thresholds and provides intervention strategies to reduce the burden of mental health in children.

2 Literature Review

This chapter reviews existing research on the effects of early life stress (ELS) on brain development and mental health trajectories.

[Euchus et al \(2021\)](#) Impact of ELS on brain development and behaviour used zebrafish as a model organism to study the impact of ELS on brain development and behavior. The model leverages the genetic and neuroanatomical similarities between zebrafish and humans to examine stress responses. A common formulation used in this type of model involved modeling stress-induced changes in gene expression using a system of differential equations:

$$\frac{dG(t)}{dt} = \beta - \gamma G(t) + \delta S(t) \quad (1)$$

Where: $G(t)$ represents the level of a specific gene expression at time t .

β is the baseline gene expression rate.

γ is the decay rate of gene expression.

δ represents the effect of stress $S(t)$ on gene expression.

This equation helps to understand how ELS influences gene expression patterns related to stress and developmental outcomes. Translating findings from zebrafish to human neurodevelopment requires careful consideration of species-specific differences in stress responses.

[Reza et al\(2022\)](#) Backward bifurcation in a two strain model of Heroine Addiction[8], they introduced and analyzed a two-strain epidemic model with the superinfection for modeling the effect of prescribed opioids abuse on heroin addiction. Among the various causes of heroin addiction, the use of prescription opioids is one of the main reasons. The model contains the impact of relapse of individuals under treatment/rehabilitation

to drug abuse in each strain and was formulated as follows;

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - \frac{\beta_1 S I_1}{N} - \frac{\beta_2 S I_2}{N} - \mu S \\
 \frac{dI_1}{dt} &= \frac{\beta_1 S I_1}{N} - \delta_2 \frac{\beta_2 I_1 I_2}{N} + \delta_1 \frac{\beta_1 I_1 I_2}{N} - (\mu + \alpha_1) I_1 + \gamma_1 \frac{R I_1}{N} \\
 \frac{dI_2}{dt} &= \frac{\beta_2 S I_2}{N} + \delta_2 \frac{\beta_2 I_1 I_2}{N} - \delta_1 \frac{\beta_1 I_1 I_2}{N} - (\mu + \alpha_2) I_2 + \gamma_2 \frac{R I_2}{N} \\
 \frac{dR}{dt} &= \alpha_1 I_1 + \alpha_2 I_2 - \mu R - \gamma_1 \frac{R I_1}{N} - \gamma_2 \frac{R I_2}{N}
 \end{aligned} \tag{2}$$

Kalabu et al (2020) Modeling of substance abuse and its progression stages[4] , a model was developed on Substance-Abuse and Its Progression Stages. Substance-abuse mathematical model was proposed to study the dynamism of substance abuse and its various stages of progression from the justification that substance abuse leads to substance addiction with the development of tolerance and dependence.

The model was formulated under the following assumptions that, total population comprises Susceptible (S), Substance Abusers (A), Substance Addicts (Q), substance Tolerants (T), substance Dependents (D) and Rehabilitants (R) with the assumptions that new recruitment into the system are in the susceptible class, rehabilitation does not provide permanent recovery therefore rehabilitated persons could become susceptible again and that the rehabilitation center is strictly inpatient so as to avoid relapsing. The model equations were formulated as follows:

$$\begin{aligned}
 \frac{dS}{dt} &= \mu + \gamma R - \left(\frac{\beta_1 A}{N} + \alpha \right) S \\
 \frac{dA}{dt} &= \frac{\beta_1 S A}{N} - (\delta_1 + \beta_2 + \alpha + \rho_1) A \\
 \frac{dQ}{dt} &= \beta_2 A - (\delta_2 + \beta_3 + \alpha + \rho_2) Q \\
 \frac{dT}{dt} &= \beta_3 Q - (\delta_3 + \beta_4 + \alpha + \rho_3) T \\
 \frac{dD}{dt} &= \beta_4 T - (\delta_4 + \alpha + \rho_4) D \\
 \frac{dR}{dt} &= \delta_1 A + \delta_2 Q + \delta_3 T + \delta_4 D - (\gamma + \alpha + \rho_5) R
 \end{aligned} \tag{3}$$

where $N = S + A + Q + T + D + R$,

subject to the initial conditions;

$t = 0$, $S(0) = S_0$, $A(0) = A_0$, $Q(0) = Q_0$, $T(0) = T_0$, $D(0) = D_0$, $R(0) = R_0$

with $\mu, \gamma, \alpha, \beta, \delta, \rho$ as parameters.

In this research, epidemiological approach has been adopted in the mathematical model which enables scientists to study the dynamism of substance-abuse and its various stages of progression. With this in mind, a comprehensive mathematical model is crucial for understanding the mechanisms underlying the effects of early life stress on brain development and mental health trajectories using epidemiological approach in the model development.

3 Model Development

This SEIR model is designed to study how early life stress (ELS) impacts brain development and mental health. It categorizes individuals into four groups: susceptible (S), exposed (E), impacted (I), recovered (R), and models how individuals transition between these states based on the population changes over time. The dynamics are influenced by vital demographic processes (births and deaths) and modulated by biological, genetic, and psychological factors.

We define four compartments:

$S(t)$: Susceptible children (not yet exposed to ELS)

$E(t)$: Exposed children (experienced ELS, no symptoms)

$I(t)$: Impacted children (symptomatic mental health burden)

$R(t)$: Recovered children (temporary resolution of symptoms)

The model equations incorporating birth-death dynamics and relapse are:

$$\begin{aligned}\frac{dS}{dt} &= \Lambda p_1 - \beta S E_f (1 - R_f) - \mu S \\ \frac{dE}{dt} &= \Lambda p_2 + \beta S E_f (1 - R_f) - \alpha E (N_f + G_f) - \mu E \\ \frac{dI}{dt} &= \Lambda p_3 + \alpha E (N_f + G_f) - \sigma I + \gamma R_f I - \mu I \\ \frac{dR}{dt} &= \sigma I - \gamma R_f I - \mu R\end{aligned}\tag{4}$$

From the above definitions and descriptions, the resulting diagram for the model is given as;

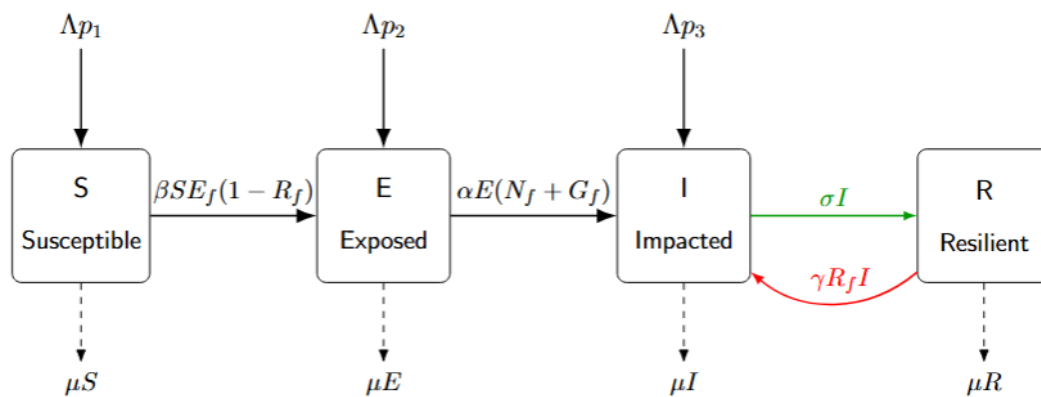


Figure 1: Schematic diagram of the SEIR model for Early Life Stress (ELS)

Model Parameters:

$\beta = 0.28$: Stress exposure rate [1]

$E_f = 0.65$: Environmental factor index [1]

$R_f = 0.35$: Resilience factor [5]

$N_f = 0.52, G_f = 0.40$: Neurobiological and genetic factors [5]

$\alpha = 0.18$: Incubation rate [10]

$\sigma = 0.12$: Recovery rate [10]

$\gamma = 0.06$: Relapse rate [10]

$\Lambda = 0.025$, $\mu = 0.015$: Birth and death rates [11]

$p_1 = 0.70$, $p_2 = 0.25$, $p_3 = 0.05$: Birth distribution proportions [1]

4 Model Analysis.

The well-posedness of the differential equations for the model formulated in this study was demonstrated by proving several theorems on the feasibility, boundedness, equilibria, local and global stabilities.

4.1 Positivity

From the model equations, we establish that the model is biologically meaningful by showing that the state variables (representing population sizes) remain non-negative and bounded for all time, given non-negative initial conditions. Positivity ensures that the population sizes are non negative, for it to be realistic due to resource limitations or other constraints.

If $S = 0$ at some time t_0 , then

$$\left. \frac{dS}{dt} \right|_{S=0} = \Lambda p_1 > 0 \quad (4)$$

assuming $\Lambda > 0$ and $p_1 > 0$. This ensures S cannot become negative.

If $E = 0$ at some time t_0 , then

$$\left. \frac{dE}{dt} \right|_{E=0} = \Lambda p_2 + \beta S E_f (1 - R_f) \geq \Lambda p_2 > 0 \quad (5)$$

assuming $\Lambda > 0$ and $p_2 > 0$. This ensures E remains non-negative.

If $I = 0$ at some time t_0 , then

$$\left. \frac{dI}{dt} \right|_{I=0} = \Lambda p_3 + \alpha E (N_f + G_f) \geq \Lambda p_3 > 0 \quad (6)$$

assuming $\Lambda > 0$ and $p_3 > 0$. This ensures I remains non-negative.

If $R = 0$ at some time t_0 , then

$$\left. \frac{dR}{dt} \right|_{R=0} = \sigma I \geq 0 \quad (7)$$

since $I \geq 0$ and $\sigma > 0$. This ensures R remains non-negative.

Therefore, the system preserves positivity of all state variables, making it biologically meaningful. The solution trajectories that start in the non-negative orthant \mathbb{R}_+^4 remain in \mathbb{R}_+^4 for all $t \geq 0$.

4.2 Boundedness

For a biologically realistic model, we need to show that the total population remains bounded over time. Let $N = S + E + I + R$ be the total population in the system.

By adding all equations in our system, we obtain:

$$\frac{dN}{dt} = \frac{d(S + E + I + R)}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \quad (8)$$

$$= \Lambda p_1 + \Lambda p_2 + \Lambda p_3 - \mu S - \mu E - \mu I - \mu R \quad (9)$$

$$= \Lambda(p_1 + p_2 + p_3) - \mu(S + E + I + R) \quad (10)$$

Assuming that $p_1 + p_2 + p_3 = 1$ (i.e., the birth rates into different compartments sum to 1), we have:

$$\frac{dN}{dt} = \Lambda - \mu N \quad (11)$$

This is a linear first-order ordinary differential equation which can be solved using standard techniques. Therefore, the total population $N(t)$ is bounded above by $\frac{\Lambda}{\mu}$, regardless of the initial conditions. This constant $\frac{\Lambda}{\mu}$ represents the carrying capacity of the population in this model. We define a positively invariant region:

$$\Omega = \left\{ (S, E, I, R) \in \mathbb{R}_+^4 : S + E + I + R \leq \frac{\Lambda}{\mu} \right\} \quad (12)$$

If $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$ for all $t \geq 0$.

If $N(0) > \frac{\Lambda}{\mu}$, then $N(t)$ will eventually be less than or equal to $\frac{\Lambda}{\mu}$ as t increases.

This boundedness property ensures that the solutions of the system are Well-posedness and biologically meaningful. Positivity and boundedness of the SEIR-ELS model has been established thus making it biologically meaningful.

4.3 Stress Propagation Index R_0

We define the infected state vector:

$$x = \begin{bmatrix} E \\ I \end{bmatrix}^T$$

We separate the system into:

$\mathcal{F}(x)$: the rate of new infections

$\mathcal{V}(x)$: the rate of transfer in/out of infected compartments (not due to new infections)

New Infection Terms

$$\mathcal{F} = \begin{bmatrix} \beta S E_f (1 - R_f) \\ 0 \end{bmatrix}$$

$$\mathcal{V} = \begin{bmatrix} \alpha(N_f + G_f)E + \mu E \\ -\alpha(N_f + G_f)E + (\sigma - \gamma R_f + \mu)I \end{bmatrix}$$

$$F = \frac{\partial \mathcal{F}}{\partial x} \Big|_{DFE} = \begin{bmatrix} \beta \frac{p_1 \Lambda}{\mu} E_f (1 - R_f) & 0 \\ 0 & 0 \end{bmatrix}$$

$$V = \frac{\partial \mathcal{V}}{\partial x} \Big|_{DFE} = \begin{bmatrix} \alpha(N_f + G_f) + \mu & 0 \\ -\alpha(N_f + G_f) & \sigma - \gamma R_f + \mu \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\alpha(N_f + G_f) + \mu} & 0 \\ \mu(\gamma R_f + \lambda + \sigma + \mu) & \frac{1}{\sigma + \mu - \gamma R_f} \end{bmatrix}$$

Next Generation Matrix (FV^{-1})

$$FV^{-1} = \begin{bmatrix} \frac{\beta S_0 E_f (1 - R_f)}{\alpha(N_f + G_f) + \mu} & 0 \\ 0 & 0 \end{bmatrix}$$

$$R_0 = \frac{\beta S_0 E_f (1 - R_f)}{\alpha(N_f + G_f) + \mu}$$

Substituting $S_0 = \frac{p_1 \Lambda}{\mu}$

$$R_0 = \frac{\beta E_f (1 - R_f) \cdot \Lambda p_1}{\mu(\alpha(N_f + G_f) + \mu)}$$

This represents the expected number of new symptomatic cases caused by stressors in a fully susceptible population.

4.4 Sensitivity Analysis of the Stress Propagation Index R_0

Sensitivity analysis is performed to evaluate the impact of model parameters on the transmission dynamics of early life stress (ELS). The normalized forward sensitivity index for each parameter x is defined as:

$$\Upsilon_x^{R_0} = \frac{\partial R_0}{\partial x} \cdot \frac{x}{R_0}$$

A positive index indicates that an increase in the parameter increases R_0 , while a negative index suggests that increasing the parameter decreases R_0 .

Parameter	Symbol	Sensitivity Index
Stress transmission rate	β	+1.000
Environmental factor	E_f	+1.000
Resilience factor	R_f	-0.538
Incubation rate	α	-0.917
Neurobiological factor	N_f	-0.518
Genetic factor	G_f	-0.399
Natural death rate	μ	-0.083

Table 1: Normalized sensitivity indices of R_0 with respect to model parameters.

4.5 Disease-Free Equilibrium (DFE)

The Disease-Free Equilibrium represents a state of mental health system where no active progression of stress-related mental health issues occurs. Its stability is crucial in understanding how early life stress can potentially trigger or prevent long-term mental health trajectories.

At the DFE, $E = I = R = 0$, and

$$S^* = \frac{\Lambda p_1}{\mu}, \quad E^* = I^* = R^* = 0$$

4.6 Local Stability of Disease-Free Equilibrium

We compute the Jacobian matrix of the system at the DFE:

$$J = \begin{bmatrix} -\beta E_f(1 - R_f) - \mu & 0 & 0 & 0 \\ \beta E_f(1 - R_f) & -\alpha(N_f + G_f) - \mu & 0 & 0 \\ 0 & \alpha(N_f + G_f) & -(\sigma - \gamma R_f + \mu) & 0 \\ 0 & 0 & \sigma + \gamma R_f & -\mu \end{bmatrix}$$

so its eigenvalues are the diagonal entries:

$$\begin{aligned} \lambda_1 &= -\beta E_f(1 - R_f) - \mu < 0 \\ \lambda_2 &= -\alpha(N_f + G_f) - \mu < 0 \\ \lambda_3 &= -(\sigma - \gamma R_f + \mu) < 0 \\ \lambda_4 &= -\mu < 0 \end{aligned}$$

By Routh-Hurwitz Stability Criterion, all eigenvalues of the Jacobian have negative real parts thus when

$R_0 < 1$, we have:

$$\text{Tr}(J) < 0, \quad \det(J) > 0$$

This ensures that the recovery-driven feedback (relapse loop) is not stronger than the combined forces of recovery and natural death. Thus, when $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable, and this means that small perturbations from the DFE will decay over time, leading the system back to the disease-free state.

4.7 Global Stability of Disease-Free Equilibrium

To establish the global stability of DFE, we apply the Castillo-Chavez and Song theorem [3]. Consider the subsystem with $E = I = 0$:

$$\frac{dS}{dt} = \Lambda p_1 - \mu S, \quad \frac{dR}{dt} = -\mu R$$

:

$$S(t) \rightarrow \frac{\Lambda p_1}{\mu}, \quad R(t) \rightarrow 0 \quad \text{as } t \rightarrow \infty$$

$$fx = \begin{bmatrix} E \\ I \end{bmatrix}$$

$$F = \begin{bmatrix} \beta S E_f(1 - R_f) \\ 0 \end{bmatrix}$$

$$V(\mathbf{x}) = \begin{bmatrix} \alpha E(N_f + G_f) + \mu E \\ -\alpha E(N_f + G_f) + (\sigma - \gamma R_f + \mu)I \end{bmatrix}$$

$$D_x V = \begin{bmatrix} \alpha(N_f + G_f) + \mu & 0 \\ -\alpha(N_f + G_f) & \sigma - \gamma R_f + \mu \end{bmatrix}$$

Using the Castillo-Chavez approach [3], if $R_0 < 1$, then the disease-free equilibrium is globally asymptotically stable.

4.8 Endemic Equilibrium (EE)

At the endemic equilibrium:

$$\begin{aligned} S^* &= \frac{\Lambda p_1}{\mu + \beta E_f(1 - R_f)} \\ E^* &= \frac{1}{\alpha(N_f + G_f) + \mu} [\Lambda p_2 + \beta S^* E_f(1 - R_f)] \\ I^* &= \frac{1}{\sigma + \gamma R_f + \mu} [\Lambda p_3 + \alpha E^*(N_f + G_f)] \\ R^* &= \frac{\sigma + \gamma R_f}{\mu} I^* \end{aligned}$$

4.9 Local Stability of Endemic Equilibrium

The eigenvalues are the diagonal entries of the Jacobian matrix.

$$\lambda_1 = -\beta E_f(1 - R_f), \lambda_2 = -\alpha(N_f + G_f) - \mu, \lambda_3 = -\sigma + \gamma R_f - \mu, \lambda_4 = -\mu$$

$$\text{Tr}(J) = -\beta E_f(1 - R_f) - \alpha(N_f + G_f) - \sigma + \mu - \gamma R_f - \mu$$

The stability of the endemic equilibrium depends on the value of R_0 and the biological parameters.

When

$$R_0 > 1, \quad \text{Tr}(J) < 0, \quad \det(J) > 0,$$

All eigenvalues have negative real parts when

$$\sigma + \mu > \gamma R_f, R_0 > 1$$

then

$$\text{Tr}(J) < 0, \det(J) > 0$$

so the endemic equilibrium is locally asymptotically stable.

4.10 Global Stability of Endemic Equilibrium

Let a Lyapunov function be:

$$V(E, I) = a(E - E^*)^2 + b(I - I^*)^2$$

where $a, b > 0$ are constants. Then $\dot{V} \leq 0$ when $R_0 > 1$. Hence, the endemic equilibrium is globally stable when stress is endemic in the population. [asa](#)

5 Discussion

This study developed a compartmental SEIR model to understand the effects of early life stress on childhood and adolescent mental health, incorporating key biological, environmental, and resilience-related parameters. Key findings showed that Stress Propagation Index R_0 is highly sensitive to Environmental factor index E_f , Resilience factor R_f , and Incubation rate α .

Increasing resilience factors significantly reduce the size and duration of the impacted population, while reducing environmental stressors delays symptom onset and flattened the progression curve. In contrast, increasing the incubation rate accelerated transitions from exposure to impact, underscoring the importance of early intervention.

Reducing environmental risk factors such as poverty, Parenting support and early childhood education (e.g., Baby-Friendly Initiatives, daycare subsidies), Community mental health programs (e.g., CHW-led counseling, school-based therapy), Violence prevention and legal support in low-income areas, Nutrition and housing assistance programs and promoting resilience through family support and psychosocial interventions could have significant public health impact. This aids in prioritizing interventions and resource allocation.

The model also demonstrated how births, deaths, and background stress levels influence population flow through the compartments over time. Overall, the results emphasize on the critical role of resilience-building, environmental improvement, and early intervention in mitigating long-term psychological impacts of early life stress.

6 Conclusion

A structured SEIR model of early life stress was developed and rigorously analyzed. Both local and global stability conditions were derived, offering insights into how resilience and environmental quality affect stress dynamics. The model is particularly suited for forecasting mental health outcomes in response to early adversity, making it a useful tool for public health and policy interventions in settings where large-scale interventions are needed. By incorporating both biological and social factors, the ELS model presents a holistic view of early life stress dynamics, offering important insights for future research and practical applications in mental health.

References

- [1] African Population and Health Research Center. (2021). *Nairobi Urban Health and Demographic Surveillance System*. Nairobi, Kenya.
- [2] Bindt, C., Guo, N., Schauer, M., Salm, A., & Teicher, M. H. (2017). Early life stress and its relation to mental health in adolescence and adulthood. *Current Psychiatry Reports*, 19 (10), 1–9.
- [3] C. Castillo-Chavez and Y. Song, Dynamical models of tuberculosis and their applications, *Mathematical Biosciences and Engineering*, vol. 1, no. 2, pp. 361–404, 2004.

- [4] Kalabu S. A., Usamatu U., Abdul A. I and Ibrahim Y. I., (2020). Modelling of Substance-Abuse and Its Progression Stages. North American Academic Research. Volume 3, Issue 12; 3(12) 12-29. <http://twasp.info/journal/home>
- [5] Kenya Medical Research Institute, *Neurobiological Biomarker Dataset*, Technical Report, Nairobi, Kenya, 2022.
- [6] Kumar, M., Huang, K. Y., Othieno, C., Wamalwa, D., Madeghe, B., Osok, J., ... & McKay, M. M. (2018). Adverse childhood experiences and child mental health: Mediating role of maternal depression and parenting stress. *Journal of Child and Adolescent Trauma*, 11(3), 345–356. <https://doi.org/10.1007/s40653-017-0176-3>
- [7] Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience* 10, 434–445
- [8] Reza Memarbashi1, Atena Ghasemabadi and Zahra Ebadi. (2022). Backward bifurcation in a two strain model of heroin addiction. *Computational Methods for Differential Equations*. Vol. 10, No. 3, 2022, pp. 656-673. <http://cmde.tabrizu.ac.ir>
- [9] P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences*, vol. 180, pp. 29–48, 2002.
- [10] University of Nairobi, *Psychiatry and Pediatrics Study on Mental Health and Early Life Stress*, Internal Study Report, Nairobi, Kenya, 2021.
- [11] World Health Organization (WHO). (2021). Global Health Observatory: Mortality rate estimates for urban populations. World Health Organization. <https://www.who.int/data/gho>