

Global Stability Analysis of a SEIR Epidemic Model with Saturation Incidence Rate

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ABSTRACT

The global stability of a SEIR epidemic model with saturating incidence rate is investigated. A threshold R_0 is identified which determines the outcome of the disease. If $R_0 \leq 1$, the infected fraction of the population disappears and the disease dies out while if $R_0 > 1$, the infected fraction persists and a unique equilibrium state is shown under a careful restriction of parameters. Dulac's criterion plus Poincare'-Bendixson theorem and Lyapunov functions are used to prove the global stability of the disease free and endemic equilibria respectively. Numerical simulation illustrates the main results in the paper.

Keywords-SEIR model, saturating incidence rate, global stability, Lyapunov function, Dulac's criterion, Poincare-Bendixson.

1. INTRODUCTION

Epidemiological models with latent or incubation period have been studied by many authors, because many diseases such as influenza and tuberculosis have a latent incubation period, during which the individual is said to be infected but not infectious [3]. This period can be modeled by incorporating or introducing an exposed class [2]. The incidence of a disease is the number of new cases per unit time, and it plays an important role in the study of mathematical epidemiology. Many diseases such as influenza, measles and sexually transmitted diseases are easily spread among the population. In many studies on epidemic models, the global stabilities of SEIRS and SEIS have long been conjectured and was solved in 1995 by [4] using the Poincare-Bendixson properties of competitive systems in dimensions three combined with sophisticated use of compound matrices. The global stability of SEI model is studied in [1]. [6] provided the global stability of an SEIR model with nonlinear incidence rate. Zhang and Ma [5] systematically analyze the global dynamics of SEIR model with saturating incidence rate. Sun et al. [7] investigated the global properties of an SEIRS model with saturating contact rate. They showed that a unique endemic equilibrium exists for some conditions.

In this paper, the global dynamics of the disease free equilibrium is resolved through the use of Lyapunov function and the global stability of the endemic equilibrium is done through Dulac's criterion plus Poincare'-Bendixson theorem.

This paper is organized as follows. The model is stated in section 2 and the basic reproduction number R_0 is calculated using the next generation matrix. Local stability and global stability of the disease-free equilibrium is established in section 3. In Section 4, local stability and global stability using Dulac's criterion plus Poincare Bendixson Theorem are established. The numerical simulations for the model are carried out in section 5.

1.1 The Mathematical Model

$$\begin{aligned}\frac{dS}{dt} &= \pi - \frac{\beta SI}{1+mI} - \mu S \\ \frac{dE}{dt} &= \frac{\beta SI}{1+mI} - (\epsilon + \mu)E \\ \frac{dI}{dt} &= \epsilon E - (\gamma + \alpha + \mu)I \\ \frac{dR}{dt} &= \gamma I - \mu R\end{aligned}$$

In our model, we have divided the population into four compartments (susceptible, exposed, infected and recovered) depending on the epidemiological status of individuals. We denote the population of those who are susceptible as S, who

exposed as E, who are infected by I and those who are subsequently removed as R. Our assumptions on the dynamical transfer of the population are demonstrated in the diagram below.

The parameter $\pi > 0$ is the rate for natural birth and $\mu > 0$ is that of natural death. The parameter α is the rate for disease-induced death, γ is the rate for recovery and ϵ is the rate at which the exposed individuals become infective so

that $\frac{1}{\epsilon}$ is the mean latent period. The recovered individuals are assumed to acquire permanent immunity; there is no transfer from the R class back to the S class. The force of infection is $\frac{\beta I}{1+mI}$ where β the effective per capita contact rate of infective individuals and the saturating incidence rate is $\frac{\beta SI}{1+mI}$ and mis the saturation term for the infected individuals.

Using the above definition and assumptions, we derive the following SEIR model with saturating incidence rate.

$$\begin{aligned}\frac{dS}{dt} &= \pi - \frac{\beta SI}{1+mI} - \mu S \\ \frac{dE}{dt} &= \frac{\beta SI}{1+mI} - (\epsilon + \mu)E \\ \frac{dI}{dt} &= \epsilon E - (\gamma + \alpha + \mu)I \\ \frac{dR}{dt} &= \gamma I - \mu R\end{aligned}\quad (1)$$

Where $\pi, \mu, \beta, \gamma, \epsilon, \alpha, m$ are assumed to be positive constant.

It is easy to see that the region $\{(S, E, I, R) | S > 0, E \geq 0, I \geq 0, R > 0\}$ is positively invariant for the model (1).

Summing up the four equations in model (1), we have

$$\frac{d}{dt}(S + E + I + R) = \mu \left[\frac{\pi}{\mu} - (S + E + I + R) \right]$$

Then, $\lim_{t \rightarrow \infty} \sup(S + E + I + R) \leq \frac{\pi}{\mu}$. So we study the dynamical behavior of the model (1) on the region.

$\Sigma = \{(S, E, I, R) | S > 0, E \geq 0, I \geq 0, R > 0, S + E + I + R \leq \frac{\pi}{\mu}\}$. Also observe that the variable R does not appear in the first three equations of (1). This allows us to attack (1) by studying the subsystem

$$\begin{aligned}\frac{dS}{dt} &= \pi - \frac{\beta SI}{1+mI} - \mu S \\ \frac{dE}{dt} &= \frac{\beta SI}{1+mI} - (\epsilon + \mu)E \\ \frac{dI}{dt} &= \epsilon E - (\gamma + \alpha + \mu)I\end{aligned}\quad (2)$$

From biological considerations, we study (2) in the closed set

$$\Gamma = \{(S, E, I) \in \mathbb{R}_+^3 | S + E + I \leq 1\} \quad (3)$$

Where \mathbb{R}_+^3 denotes the nonnegative core of \mathbb{R}^3 including its lower dimensional faces.

Corresponding to E=I=0 model (2) always has a disease-free equilibrium

$$P_0\left(\frac{\pi}{\mu}, 0, 0\right)$$

Let $X = (S, E, I)^T$. Then the model (2) can be written as $\frac{dx}{dt} = f(x) - v(x)$, where

$$F(x) = \begin{pmatrix} \frac{\beta SI}{1+mI} \\ 0 \\ 0 \end{pmatrix} \quad V(x) = \begin{pmatrix} -\pi + \mu S \\ \epsilon E - \mu E \\ \gamma I + \alpha I + \mu I - \sigma E \end{pmatrix}$$

We have

$$F = \begin{pmatrix} 0 & \beta S_0 \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \epsilon + \mu & 0 \\ -\epsilon & \gamma + \alpha + \mu \end{pmatrix}$$

So

$$V^{-1} = \begin{pmatrix} \frac{1}{\epsilon + \mu} & 0 \\ \frac{\epsilon}{(\epsilon + \mu)(\gamma + \alpha + \mu)} & \frac{1}{\gamma + \alpha + \mu} \end{pmatrix}$$

In [8], the basic reproduction number is defined as the spectral radius of the next generation matrix $FV^{-1}(\rho(fV^{-1}))$. So, according to theorem 2 in [8], the basic reproduction number of model (2), denoted R_0 , is

$$R_0 = \rho(FV^{-1}) = \frac{\epsilon \beta \pi}{\mu(\epsilon + \mu)(\gamma + \alpha + \mu)}$$

2. LOCAL AND GLOBAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM

Theorem 1: The disease-free equilibrium P_0 is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.

Proof: The linearized problem corresponding to (2) is

$$\frac{dx}{dt} = Jx, \text{ where}$$

$$x = (x_1, x_2, x_3)^T, (x_1, x_2, x_3) \in \mathbb{R}_+^3$$

And

$$J(P_0) = \begin{pmatrix} -\mu & 0 & \beta S_0 \\ 0 & -(\epsilon + \mu) & \beta S_0 \\ 0 & \epsilon & -(\gamma + \alpha + \mu) \end{pmatrix}$$

with eigenvalue $\lambda_1 = -\mu$, and the roots of the quadratic equation

$$f(\lambda) = \lambda^2 + (\alpha + \gamma + \epsilon + 2\mu)\lambda + \mu\alpha + \mu\gamma + \mu^2 + \epsilon\alpha + \epsilon\gamma + \epsilon\mu - \beta S_0$$

Because all the model parameter values are assumed positive, so it follows that $\lambda_1 < 0$. Obviously, if $R_0 < 1$, then the roots of $f(\lambda)$ have negative real parts, therefore P_0 is locally asymptotically stable when $R_0 < 1$; if $R_0 > 1$, then the roots of $f(\lambda)$ are real and one is positive, so that P_0 is unstable.

Theorem 2: The disease-free equilibrium $P_B = \left(\frac{\pi}{\mu}, 0, 0 \right)$ of (2) is globally asymptotically stable in P if $R_0 \leq 1$, it is unstable; if $R_0 > 1$, and the solutions of (2) starting sufficiently close to P_0 is Γ and move away from P_0 except that those starting on the invariant S-axis approach P_0 along this axis.

Remark: The global stability of P_0 with $R_0 < 1$ shall be proved by the method of Lyapunov functions.

$$\text{Set } L = \sigma E - (\epsilon + \mu)I$$

By differentiating L, we have

$$\begin{aligned} L^1 &= \epsilon E^1 - (\epsilon + \mu)I^1 \\ L^1 &= \epsilon \left(\frac{\beta SI}{1+mI} - (\epsilon + \mu)E \right) + (\epsilon + \mu) [\sigma E - (\gamma + \alpha + \mu)I] \\ L^1 &= \frac{\epsilon \beta SI}{1+mI} - (\epsilon + \mu)(\gamma + \alpha + \mu)I \\ &= I \left[\frac{\epsilon \beta SI}{1+mI} + (\epsilon + \mu)(\gamma + \alpha + \mu) \right] \\ &= I \left(\frac{R_0}{1+mI} - 1 \right) \end{aligned}$$

Therefore, $L^1 \leq 0$ for $R_0 \leq 1$ and $L^1 = 0$ if and only if $I = 0$. Further, substituting $I = 0$ into the equation for S, E, I shows that $S(t) \rightarrow \frac{\pi}{\mu}$, $E(t)$ as $t \rightarrow \infty$. It follows that, by the LaSalle's invariance principle, every solution to the system (2) with initial conditions in D approaches P_0 as $t \rightarrow \infty$. Thus since the region P is positively invariant, the disease free-equilibrium, P_0 is globally asymptotically stable in P if $R_0 \leq 1$.

Its epidemiological implication is that the infected fraction (the sum of the exposed and the infections fractions) of the population vanishes in time so the disease dies out.

3. LOCAL AND GLOBAL STABILITY OF THE ENDEMIC EQUILIBRIUM

Theorem 3: When $R_0 > 1$, the endemic equilibrium P^* of the system (2) is locally asymptotically stable if $b_3 > 0$ and $b_1 b_2 b_3 > 0$, where b_1, b_2 and b_3 are presented in the following proof.

Proof: the Jacobian matrix of (2) of P^* is

$$J(P_0) = \begin{pmatrix} -(\beta I_* + \mu) & 0 & \beta S_0 \\ \beta I_* & -(\epsilon + \mu) & \beta S_0 \\ 0 & \epsilon & -(\gamma + \alpha + \mu) \end{pmatrix}$$

The characteristic equation of the matrix is

$$\lambda^3 + b_1 \lambda^2 + b_2 \lambda + b_3 = 0 \text{ where}$$

$$b_1 = \beta I_* + 3\mu + \alpha + \epsilon + \gamma$$

$$b_2 = 2\beta I_* \mu + \beta I_* \alpha + \beta I_* \epsilon + \beta I_* \gamma + 3\mu^2 + 1\alpha \mu + 2\epsilon \mu + 2\mu \gamma + \gamma \epsilon + \epsilon \alpha$$

$$b_3 = \beta I_* \epsilon \gamma + \beta I_* \epsilon \mu + \beta I_* \alpha \mu + \beta I_* \gamma \mu + \beta I_* \mu \alpha + \beta I_* \mu^2 + \mu \epsilon \gamma + \mu^2 \epsilon + \mu \alpha \epsilon + \mu^2 \gamma + \mu^2 \alpha + \mu^3$$

Obviously, $b_1 > 0$. Based on Hurwitz criterion, when $R_0 > 1$, the endemic equilibrium P^* of the system (2) is locally asymptotically stable if $b_3 > 0$ and $b_1 b_2 b_3 > 0$.

In order to prove the global stability of the endemic equilibrium P^* of the equation (2) we apply Dulac's criterion plus Poincare-Bendixson Theorem.

Theorem 4: (Dulac's Criterion)

Consider the following general nonlinear autonomous system of de

$$x(t) = f(x), x \in E \quad (*)$$

Let $f \in C^1(E)$ where E is a simple connected region in \mathbb{R}^2 . If there exists a function $H \in C^1(E)$ such that $\nabla \cdot (Hf)$ is not identically zero and does not change sign in E , the system (*) has no closed orbit lying entirely in E . If A is an annular region contained in E on which $\nabla \cdot (Hf)$ does not change sign, then there is at most one limit cycle of the system (*) in A .

Theorem 5:(The Poincare-Bendixson Theorem): Suppose that $f \in C^1(E)$ where E is an open subset of \mathbb{R}^n and that the system (*) has a rejecting Γ contained in a compact subset f of E . Assume that the system (*) has only one unique equilibrium point x_0 in f , then one of the following possibilities holds.

- (a) $w(\Gamma)$ is the equilibrium point x_0
- (b) $w(\Gamma)$ is a periodic orbit
- (c) $w(\Gamma)$ is a graphic

Theorem 6: Let P_* be the unique positive equilibrium point of the system (2). If $R_0 > 1$, then P_* of the system (*) is globally asymptotically stable.

Proof: We use Dulac's criterion plus Poincaré-Bendixson Theorem to analyze the system (2). Consider.

$$H(S, E, I) = \frac{1}{SEI} \quad (**)$$

Where $S > 0, E > 0, I > 0$ then

$$\begin{aligned} \nabla \cdot (Hf) &= \frac{\partial}{\partial S} (H \cdot f_1) + \frac{\partial}{\partial E} (H \cdot f_2) + \frac{\partial}{\partial I} (H \cdot f_3) \\ &= \frac{\partial}{\partial S} \left[\frac{1}{SEI} \left(\pi - \frac{\beta SI}{1+mI} - \mu S \right) \right] + \frac{\partial}{\partial E} \left[\frac{1}{SEI} \left(\frac{\beta SI}{1+mI} - (E + \mu)E \right) \right] \\ &\quad + \frac{\partial}{\partial I} \left[\frac{1}{SEI} (\sigma E - \gamma I + \alpha I + \mu I) \right] \\ &= -\frac{\pi}{S^2 EI} - \frac{\beta}{E^2 (1+mI)} - \frac{E}{S^2 I} \\ &= -\left(\frac{\pi}{S^2 EI} - \frac{\beta}{E^2 (1+mI)} - \frac{E}{S^2 I} \right) < 0 \end{aligned}$$

Hence, by the Dulac's criterion, there is closed orbit in the first quadrant, therefore, the endemic equilibrium is globally asymptotically stable.

4. NUMERICAL SIMULATION

In order to illustrate the various theoretical results, numerical experiments (using maple) were carried out to compute the solutions of system (2).

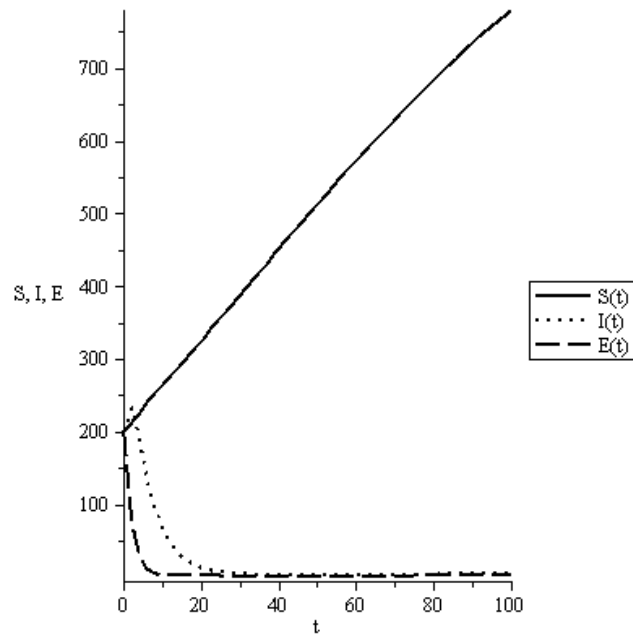


Figure 1: Graph of $S(t)$, $I(t)$ and $E(t)$ against time (t) when $\pi=8$, $\beta=0.0005$, $m=0.1$, $\mu=0.0027$, $\epsilon=0.5$, $\gamma=0.2$, $\alpha=0.0062$ when $R_0 < 1$

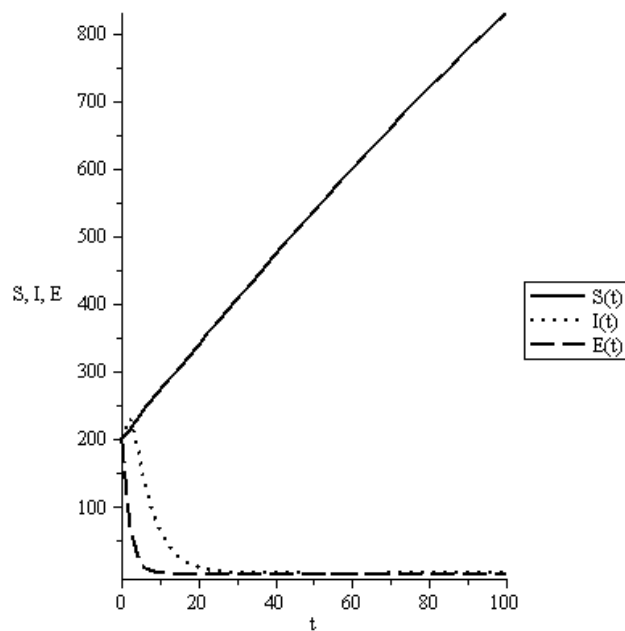


Figure 2: Graph of $S(t)$, $I(t)$ and $E(t)$ against time (t) when $\pi=8$, $\beta=0.0005$, $m=0.4$, $\mu=0.0027$, $\epsilon=0.5$, $\gamma=0.2$, $\alpha=0.0062$ when $R_0 < 1$

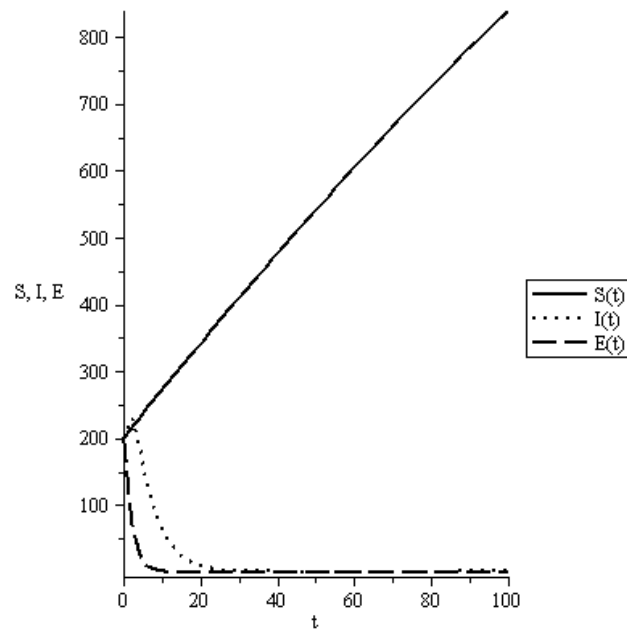


Figure 3: Graph of $S(t)$, $I(t)$ and $E(t)$ against time (t) when $\pi=8$, $\beta=0.0005$, $m=0.7$, $\mu=0.0027$, $\epsilon=0.5$, $\gamma=0.2$, $\alpha=0.0062$ when $R_0 < 1$

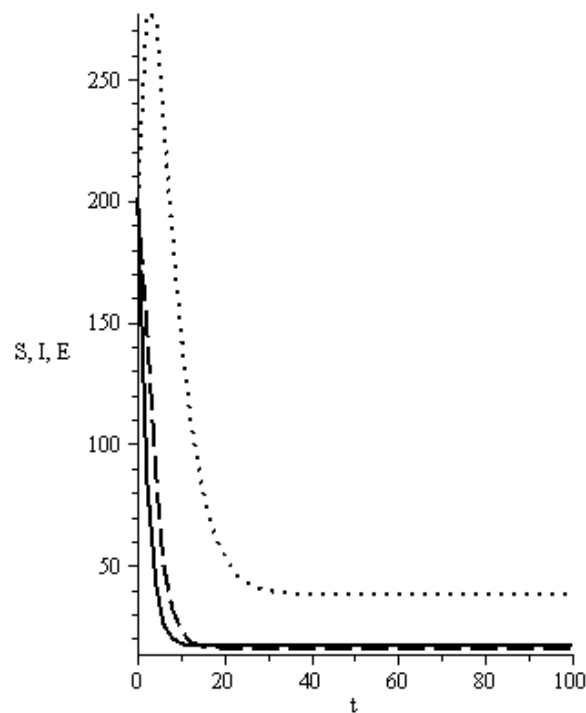


Figure 4: Graph of $S(t)$, $I(t)$ and $E(t)$ against time (t) when $\pi=8$, $\beta=0.48$, $m=0.1$, $\mu=0.0027$, $\epsilon=0.5$, $\gamma=0.2$, $\alpha=0.0062$ when $R_0 > 1$

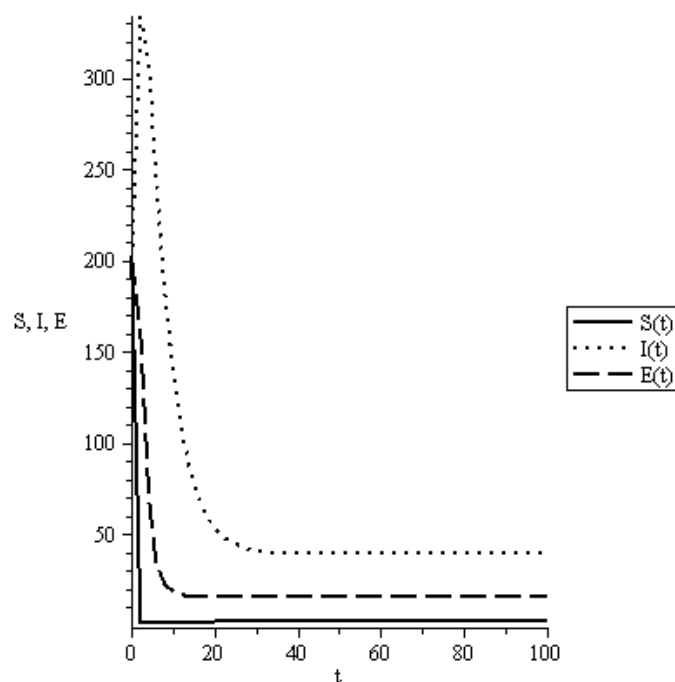


Figure 5: Graph of $S(t)$, $I(t)$ and $E(t)$ against time (t) when $\pi=8$, $\beta=0.48$, $m=0.1$, $\mu=0.0027$, $\epsilon=0.5$, $\gamma=0.2$, when $R_0 < 1$ and $\alpha=0$

5. DISCUSSION OF RESULTS

We considered the behavioural analysis of the model when $R_0 < 1$ and the effect of the saturation term (α) on the model. In figures 1-3, we discovered that the effect of the saturation term is not all that visible but the more the saturation term is close to 1, the faster the $E(t)$ and $I(t)$ approach zero. This means that the saturation term has a minimal effect in the eradication of the said disease in this model. In figure 4, we considered the model when $R_0 > 1$ and in the presence of disease-induced death, the susceptible and exposed classes approach zero while there is a noticeable increase on the infected class at a point. In figure 5, we looked at the model when $R_0 > 1$ and when there is no disease-induced death, we discovered that only the susceptible individuals approach zero, the exposed and the infected individuals are on the increase.

We could see that the absence of the disease-induced death increase the endemic nature of the exposed individual unlike where there is presence of disease-induced death.

7. CONCLUSION

A SEIR epidemic model with saturation incidence rate is investigated. The local and global stabilities of the disease-free equilibrium are analyzed while we analyzed the local stability of the endemic equilibrium using Routh-Hurwitz criterion and the global stability using Dulac's Criterion plus Poincaré-Bendixson theorem application. We also present the numerical simulation of the model when $R_0 < 1$ and $R_0 > 1$

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