RESEARCH REVIEW JOURNALS
Volume-04
Issue-05
May-2019

ISSN: 2455-3085 (Online)
RESEARCH REVIEW International Journal of Multidisciplinary
www.rrjournals.com[UGC Listed Journal]

Age Structured Population Model and its Application for Perion Expension in the Presence of Chaperone

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ARTICLE DETAILS

Article History
Published Online: 25 May 2019

Keywords
Model,dynamics, structure, population, Stability.

ABSTRACT

This part is committed to the fundamental properties of the models old enough ward populace elements without dissemination. The primary spotlight is on the presence, uniqueness and energy of arrangements of the straight model and of the nonlinear model. Some correlation results, which will be utilized later in the investigation of the ideal control of populace elements, are expressed. The asymptotic conduct of the arrangements is additionally explored. At long last we set up some essential properties of the arrangement of direct intermittent age-subordinate populace elements.

1. Introduction

Numerical modeling is an interdisciplinary field of scholarly examination which targets modeling normal, organic procedures utilizing applied scientific methods and instruments. It has both pragmatic and hypothetical applications in natural exploration. Nature, which is a part of numerical science is an investigation of the entomb connection among species and their condition such zones as predator-prey and rivalry collaborations, sustainable asset the executives, development of pesticide safe strains, biological and hereditarily built control of irritations, multi species social orders, plant-herbivore frameworks, etc. is currently a colossal field[3,5]. The persistently extending rundown of utilization is broad on different parts of the field.

2. Population Dynamics

Population dynamics has customarily been the predominant part of nature, which has a past filled with over 210 years, albeit all the more as of late, its degree has significantly extended. Population dynamics is the investigation of minor and long-haul changes in the numbers, singular loads and age organization of people in one or a few populations, and natural and ecological procedures impacting those changes. Work in population dynamics goes back to the nineteenth century and the Lotka-Volterra predator-prey equations are a renowned model[1,2].

The expanding investigation of sensible and essentially valuable scientific models in population dynamics, regardless of whether we are managing a human population with or without its age appropriation, population of a jeopardized species, bacterial or viral development, etc, is an impression of their utilization in assisting with understanding the dynamic procedures included and in making useful forecasts. The investigation of population change has a long history. In the year 1202, an activity in a number juggling book composed by Leonardo of Pisa included structure a numerical model for a developing hare population. Scientific and computational methodologies give useful assets in the investigation of issues in population science and environments science. The subject has a rich history entwined with the advancement of measurements and dynamical frameworks hypothesis [7, 8]. These scientific and computational methodologies are currently considered as probably the most useful assets in finding out about nature. Such methodologies have prompted broad work and have given a structure to combination and investigation of such organic models. Since the mid nineteenth century, there has been developing enthusiasm for the investigation of scientific environment. Biology examines the states of presence and the connection of living creatures with one another and with their condition. Population dynamics, which considers population development, mortality, rivalry and predator-prey relations, is maybe the most scientifically evolved zone of environment.

A lot of examination has been done in refined models in population biology, for instance, models in both discrete and consistent time with and without delays alongside stochastic models with the impacts of spatially non-uniform conditions and with diffusive spread of populations [11, 12]. The expanding investigation of reasonable numerical models in environment is an impression of their utilization in assisting with understanding the dynamic procedures engaged with so much regions as predator-prey and rivalry connections, sustainable asset the executives, advancement of pesticide safe strains, biological control of nuisances, multi-species social orders, plant-herbivore frameworks, etc. The persistently growing rundown of utilizations is broad. There are additionally intriguing and valuable applications in the bio-clinical sciences and in physiology.

Scientific models give a significant commitment to natural investigations. They propose amounts that can be estimated, characterize ideas empowering to evaluate natural collaborations, and even propose diverse modeling methodologies with various suppositions to depict specific highlights of the populations. In population dynamics, from the scientific perspective, there are basically two primary modeling methods:

1. The consistent time approach utilizing conventional and fractional differential equations and
2. The discrete time approach which is all the more firmly related with the structure of the statistics of a population.

The two methodologies broadly utilize the techniques for the subjective hypothesis of dynamical frameworks. In the consistent time approach, the quantity of people of a population changes constantly in time and the most well-known modeling system applies to the depiction of the kinds of biotic

between explicit collaborations and to the cooperations of the species with condition.

In the discrete time approach, models are worked so as to depict the enumeration information of populations. They are broken in time, and are nearer to the way population development information are gotten.

The main significant work in the hypothesis of population dynamics was by Malthus in the year 1798. The Malthusian development model, some of the time called the basic exponential development model, is basically exponential development dependent on a steady pace of self multiplying dividends. The model is named after the Reverend Thomas Malthus, who wrote "An Essay on the Principle of Population", one of the soonest and most compelling books on population. He proposed a solitary animal varieties model where the pace of population development was corresponding to the size of the population. The Malthusian model is given bywhere u(t) is the complete population size at time t and r is the development pace of the given population, likewise called as the malthusian boundary.

$$\begin{aligned} \frac{du(t)}{dt} &= ru(t), \quad t > 0 \\ u(0) &= u_0 \end{aligned}$$

A Malthusian population offers no leniency for the impacts of swarming or the constraints of assets. This model is frequently alluded to as "The Exponential Law" and is broadly respected in the field of population biology as the primary standard of population dynamics.

A progressively sensible model of population development, which permits the development rate to rely on the size of the all-out population was presented by Verhulst in the year 1838 and is known as the strategic condition given by

$$\begin{aligned} \frac{du(t)}{dt} &= ru(t)\left(1 - \frac{u(t)}{K}\right), \quad t > 0 \\ u(0) &= u_0 \end{aligned}$$

where r is the characteristic development consistent and K is the ecological conveying limit. In this model, the underlying phase of development is roughly exponential; at that point, as immersion starts, the development eases back, and at development, development stops. The calculated equation was first utilized in the models for human population and it follows a rule that the protection from development ought to be quadratic. The inspiration for the quadratic term was the similarity with movement, in an opposing medium where, the obstruction term might be modeled as quadratic in the speed. A run of the mill use of the calculated equation is a typical model of population development, which expresses that:

1. the pace of proliferation is relative to the current population, all else being equivalent

2. the pace of proliferation is relative to the measure of accessible assets, all else being equivalent. Along these lines, the subsequent term models the opposition for accessible assets, which will in general breaking point the population development.

One of the lacks of the over two models is that the birth rate is considered to act immediately though there might be a period postponement to assess an opportunity to arrive at development, the limited incubation time frame, etc. We can join such postponements by considering defer differential equation models. Along these lines an improvement of the strategic equation is the Hutchinson equation where a deferral was acquainted with depict the age-structure.

$$\begin{aligned} \frac{du(t)}{dt} &= ru(t)\left(1 - \frac{u(t-\tau)}{K}\right), \quad t > 0, \quad \tau > 0 \\ u(t) &= \phi(t) \end{aligned}$$

where φ(t) is characterized in the stretch (−τ, 0].

Here, τ is the time postpone that is presented and is a known boundary. The above equation suggests that the administrative impact relies upon the population at a previous time t − τ, as opposed to at t. This equation is itself a model for a postpone impact which should be a normal over past populations and which brings about an integrodifferential equation.

In this manner, a more exact model than the Hutchinson equation is, the convolution type,

$$\frac{du(t)}{dt} = ru(t)\left(1 - \frac{1}{K}\int_{-\infty}^{t} w(t-s)u(s)ds\right),$$

where w(t) is a weighting factor which says how much accentuation ought to be given to the size of the population at prior occasions to decide the current impact on asset accessibility.

The models examined above are for the most part instances of single species models. In the year 1926, the Italian mathematician Volterra built a two-animal varieties model portraying a predator-prey network. He expected that the development pace of the prey population, without predators, is given by some consistent, however diminishes directly as an element of the thickness of predators. Simultaneously, an American scientist and mathematician Lotka created a similar model autonomous of Volterra [6, 14]. This is the now renowned Lotka-Volterra model. The prey-predator model is given by

$$\begin{aligned} \frac{du}{dt} &= \alpha u - \beta uv \\ \frac{dv}{dt} &= -\gamma v + \delta uv \end{aligned}$$

where u = u(t) signifies the prey population

v = v(t) signifies the predator population.

The boundaries α, β, γ, δ are for the most part positive constants.

α signifies the development pace of the prey

βuv is the decrease in the prey population because of the nearness of the predators.

γ signifies the passing pace of the predators

uv is the expansion in the predator population because of the nearness of the prey.

Different presumptions made by the Lotka-Volterra model are:

1. The prey without any predation develops unboundedly in a Malthusian manner.
2. The impact of the predation is to lessen the prey's per capita development rate by a term relative to the prey and predator populations.
3. Without any prey for food, the predator's passing rate brings about exponential rot.
4. The predator's development rate is relative to the accessible prey just as to the predator population [6, 11].

A portion of the other, two species models are the opposition and the co-activity models. Rivalry models signify two species contending with one another for similar assets like food, water and so forth. In co-activity model or the advantageous interaction model, the species in a typical territory exist together in amicability. A few of these models called the Lotka-Volterra models have been widely concentrated by numerous mathematicians.

The models of Malthus, Verhulst, Lotka and Volterra are instances of consistent or deterministic population models. Scientific demographers and population researcher have widely built up the theory of ceaseless population dynamics. One of the most significant hypotheses in this improvement has been for models that take into consideration the impacts old enough structure. For some populations, thought of the age dissemination inside the population prompts a progressively practical and valuable scientific model.

Why Age-Structured Models?

In the least complex models for a solitary population all individuals are thought to be tradable. Nonetheless, even the least complex models for illness transmission incorporate organizing the population by sickness state (vulnerable, uncovered, infective, or evacuated)[4,9].

Further developed population models add some structure to the population, for example, determination of spatial area or age. Age is one of the most significant qualities in the modeling of populations and irresistible ailments. People with various ages may have distinctive proliferation and endurance limits. Ailments may have diverse disease rates and death rates for various age gatherings.

People of various ages may likewise have various practices, and social changes are vital in charge and avoidance of numerous irresistible sicknesses. Youthful people will in general be progressively dynamic in associations with or among populations, and in illness transmissions.

Explicitly transmitted ailments (STDs) are spread through accomplice cooperation's with pair-developments, and the pair-arrangements are plainly age-subordinate as a rule. For instance, most AIDS cases happen in the gathering of youthful grown-ups[13,15].

Youth ailments, for example, measles, chicken pox, and rubella, are spread chiefly by contacts between offspring of comparable ages. The greater part of the passing's ascribed to jungle fever are in youngsters under five years old because of their more fragile invulnerable frameworks. This recommends in models for sickness transmission during a time structured population it is important to permit the contact rates between two individuals from the population to rely upon the ages of the two individuals

So as to portray age-structured models for infection transmission, we should initially build up the theory of age-structured populations. Truth be told, the main models for age-structured populations were intended for the investigation of ailment transmission in such populations.

3. Mathematical Preliminaries

Stability theory

The mathematical models or equations that depict physical marvels are by and large standard differential equations of the form

$$x\prime = F(x,t)$$

with the underlying information $x(t_0) = x_0$. Since, the underlying information, which regularly results from a wide range of estimations, may have blunders, it is essential to know the degree to which little unsettling influences in the underlying information impact the ideal conduct of the arrangements of equation. On the off chance that, by making an adequately little change in the underlying information, a considerable deviation is seen in the comparing arrangement, at that point the arrangement acquired from the given starting information is unsatisfactory in light of the fact that it doesn't portray the necessary wonder even around. The issue of researching the conditions that won't permit the answers for surprisingly veer off from the ideal conduct is in this way indispensable.

Stable: The arrangement x(t) is supposed to be steady if, for each $\varepsilon > 0$, there exists a $\delta = \delta(\varepsilon) > 0$ with the end goal that, for any arrangement $x(t) = x(t, t_0, x_0)$ of (1), the disparity $k\, x_0 - x_0\, k \leq \delta$ infers $k\, x(t) - x(t)\, k < \varepsilon$ for all $t \geq t_0$.

Asymptotically Stable: The arrangement x(t) is supposed to be asymptotically steady on the off chance that it is steady and if there exists a $\delta 0 > 0$, with the end goal that $k\, x_0 - x_0\, k \leq \delta_0$ suggests $k\, x(t) - x(t)\, k \to 0$ as $t \to \infty$.

Precarious: The arrangement x(t) is supposed to be shaky on the off chance that it isn't steady.

Stage plane investigation: Consider the general self-governing second request differential equations of the form

$$\frac{dx}{dt} = f(x,y)$$
$$\frac{dy}{dt} = g(x,y)$$

Phase curves or phase trajectories of equations are solutions of

$$\frac{dx}{dy} = \frac{f(x,y)}{g(x,y)}$$

4. Prion Expansion in the Presence of a Chaperone

Prions are irresistible agents and are polymers called PrPSc - Prion protein scrapies, of an ordinary protein, a monomer called P_rP^c - Prion protein cell. These P_rP^{Scs} cause TSEs - Transmissible Spongiform Encephalopathies, for example, cow-like spongiform encephalopathy (BSE) in dairy cattle, scrapies in sheep, Kuru and CreutzfeldJacob

sicknesses in people. Prions are microorganisms answerable for an assortment of creature and human neurodegenerative sicknesses, for example, cow-like spongiform encephalopathy (BSE), scrapie of sheep, Creutzfeldt-Jacob and Gerstmann-Straussler-Scheinker maladies of people. Bewilderingly, every one of these sicknesses can be irregular, hereditary and irresistible, in this way making the ID of the infection instrument a difficult undertaking. For a long time, the prion illnesses were believed to be brought about by moderate acting infections. These infections were regularly alluded to as moderate infection illnesses,transmissible spongiform encephalopathies, or offbeat viral maladies. Impressive exertion was consumed looking for the scrapie infection; yet none was found either concerning the revelation of an infection like molecule or a genome made out of RNA or DNA.

The irregular properties of the irresistible agent turned into the focal point of consideration starting during the 1960s, and in the mid 1980s Stanley Prusiner, expanding upon prior recommendations proposed the prion speculation. This expressed the irresistible agent in human and creature spongiform encephalopathies was made solely out of a solitary sort of protein particle assigned $P_rP^{Sc}$with no encoding nucleic corrosive. In light of prior discoveries, the term prion was acquainted with recognize the proteinaceous irresistible particles that cause scrapie from the two viroids and infections. Maybe, the best current working meaning of a prion is a proteinaceous irresistible molecule that needs nucleic corrosive.

This protein can show up in two structures that contrast just in their adaptation. One structure is the principally α-helical structure, called cell prion protein (or P_rP^{c}). This is the local type of the protein which normally shows up in numerous tissues, anyway with a prominent wealth in the cerebrum, where it is mostly situated at synaptic regions. It is normally accepted that the agent causing prion sicknesses is made out of the subsequent structure, called scrapie prion protein (or P_rP^{Sc}). It contrasts from P_rP^{c} just by its optional structure which is commanded by beta-sheet. The auxiliary contrasts cause contrasts in physical and concoction properties, for example a high obstruction of $P_rP^{Sc}$to proteases and an inclination of P_rP^{Sc} to total and structure polymers and even enormous amyloid plaques. Moreover, collaboration of the two structures prompts a change of PrP^{c} into P_rP^{Sc}. Along these lines, P_rP^{Sc} duplicates and goes about as an infective agent.

Prions multiply by a procedure called nucleated polymerization. The infective agent, P_rP^{Sc} is certifiably not a solitary protein, however a polymer or short oligomer. The P_rP^{Sc} expands its length by joining units of P_rP^{c} in a string like style. At that point, the P_rP^{c} which is joined to the P_rP^{Sc} is changed over to the irresistible structure. When the P_rP^{Sc} is sufficiently long to wrap into a helical shape called the core, it structures settling bonds and consequently gets steady. P_rP^{Scs} can comprise of thousands of monomer units. P_rP^{Sc} polymers may part into two littler irresistible polymers which can protract further. On the off chance that the split PrPSc falls under a basic length, it debases promptly into typical P_rP^{c} monomers. Consequently, the instability of short polymers is a hindrance to the arrangement of P_rP^{Sc} polymers. Since, the development of the cores is accepted to be a moderate procedure, this model records for the long brooding times of the TSEs**.**

5. The model

In this segment, we depict the model of prion multiplication under the inhibitory impacts of a chaperone. The supposition that will be that the prions duplicate by nucleated polymerization [10,16]. As indicated by this model, the natural procedures of coagulation, parting and the inhibitory impacts of the chaperone can be portrayed by a coupled framework comprising of common differential equations and a halfway differential equation. Let V (t) signify the population of P_rP^{c} monomers at time t, u (x, t) be the population of P_rP^{Sc} polymers of length x at time t and C(t) means the measure of chaperone in the framework at time t. Let λ signify the steady pace of creation of P_rP^{c} in the framework and γ be the consistent pace of corruption of the P_rP^{c}because of metabolic procedures. τ is the transformation pace of monomers P_rP^{c} to polymers P_rP^{Sc} and they are changed over at a rate corresponding to the population of the absolute number of polymers $R \propto x_0 u (x, t) dx$. β(x) is the twofold parting pace of the P_rP^{Sc} polymers of length x and κ (x, y) is the likelihood thickness work that a polymer of length y parts into one of length x and another of length y − x.

x0 is the basic length of the polymer underneath which the polymer corrupts into typical PrPc monomers. Therefore, the pace of progress of the monomer population is givenby

$$\frac{dV(t)}{dt} = \lambda - \gamma V(t) - \tau V(t) \int_{x_0}^{\infty} u(x,t)dx + 2\int_0^{x_0} x \int_{x_0}^{\infty} \beta(y)\kappa(x,y)u(y,t)dydx$$

where the keep going term on the right-hand size speaks to the monomers picked up when a PrPSc polymer parts with at any rate one polymer shorter than the base length x0. We expect that such a polymer piece debases promptly into PrPc monomers.

The 2 in the articulation represents the way that a polymer of length x more prominent than x0 parts into two P_rP^{Sc} polymers.

The polymer lengths have been appeared to go more than a great many monomer units. In, polymer lengths x was thought to be whole number qualities, however we accept constant qualities for mathematical tractability. μ(x) is the pace of debasement of the P_rP^{Scs} because of digestion. δ2 means the rate at which the PrPSc population gets decreased because of the nearness of the chaperone. −τV (t) ∂u (x, t) ∂x represents the loss of polymers of length x because of extending. $2 R \propto x \beta(y)\kappa (x, y) u (y, t) dy$ indicates the quantity of PrPScs which are added to the population when longer polymers split into polymers of length x.

In this way, the pace of progress of P_rP^{Scs} is given by

$$\frac{\partial u(x,t)}{\partial t} = -\tau V(t)\frac{\partial u(x,t)}{\partial x} - (\mu(x) + \beta(x) + \delta_2 C(t))u(x,t) + 2\int_x^{\infty} \beta(y)\kappa(x,y)u(y,t)dy$$

Let $\delta_0$be the rate at which the chaperone is debased from the framework because of metabolic procedures and δ1, the rate at which the chaperone is getting expanded in the framework.

Table 1: Parameters of the prion proliferation model

Parameters	Description
λ	Steady pace of creation of P rPc in the framework.
γ	Steady pace of corruption of the PrPc because of metabolic procedures.
τ	Change pace of monomers P rPc to polymers PrP Sc.
β(x)	Parallel parting pace of the PrPSc polymers of length x.
κ(x, y)	Likelihood thickness work that a polymer of length y parts into one of length x and another of length y – x.
x_0	Basic length of the polymer underneath which the polymer debases into ordinary P rPc monomers.
μ(x)	Pace of debasement of the PrPScs because of digestion
δ_2	Consistent rate at which the PrPSc population gets diminished because of the nearness of the chaperone.
δ_0	Steady rate at which the chaperone is corrupted from the framework because of metabolic procedures
δ_1	Steady rate at which the chaperone is getting expanded in the system.

In this way, the pace of progress of chaperone in the framework is given by

$$\frac{dC(t)}{dt} = -\delta_0 C(t) + \delta_1 C(t) \int_{x_0}^{\infty} u(x,t)dx$$

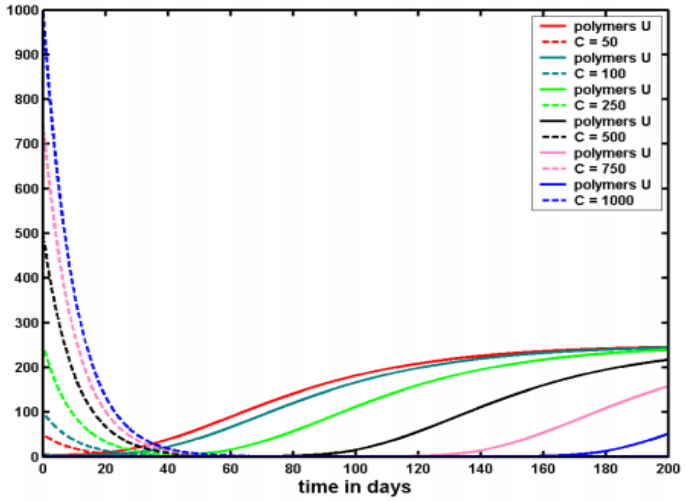
Now, in the above model we make the accompanying presumptions
Let μ(x) = μ, β(x) = βx,
For every y > x0, κ (x, y) = 1/y for x ∈ (0, y) and 0 otherwise.
Substituting the above in our model, the model changes into the accompanying:

$$\begin{aligned}
\frac{dV(t)}{dt} &= \lambda - \gamma V(t) - \tau V(t) \int_{x_0}^{\infty} u(x,t)dx + \beta x_0^2 \int_{x_0}^{\infty} u(x,t)dx \\
\frac{\partial u(x,t)}{\partial t} &= -\tau V(t) \frac{\partial u(x,t)}{\partial x} - (\mu + \beta x + \delta_2 C(t))u(x,t) \\
&\quad +2\beta \int_{x}^{\infty} u(y,t)dy \\
\frac{dC(t)}{dt} &= -\delta_0 C(t) + \delta_1 C(t) \int_{x_0}^{\infty} u(x,t)dx \\
V(0) &= V_0 \\
C(0) &= C_0 \\
u(x,0) &= u_0(x), \quad x_0 < x < \infty \\
u(x_0,t) &= 0, \quad t \geq 0
\end{aligned}$$

where the constants λ, γ, τ, β, δ_0, δ_1, δ_2 are all positive.

6. Conclusion

In this work, we have proposed a model of prion expansion with the impacts of a chaperone. Our model gives an expansion to the model of Webb et. al. We have contemplated the stability of the equilibrium purposes of the model and have



Dose response curve of population of polymers U for varying amounts of chaperone C (50, 100, 250, 500, 750 and 1000 units of chaperone)

demonstrated that the consistent state arrangements of the model are locally asymptotically steady. From the examination of the model and the outcomes demonstrated in segment 3.4, we close the accompanying:

Let $R_0 = \frac{\lambda\beta\tau}{\gamma(\mu+\beta x_0)^2}$ indicate the quantity of optional diseases created on average by one irresistible prion.

In the event that R0 < 1, at that point the infection vanishes and the sickness free equilibrium E1 is locally asymptotically steady. On the off chance that R0 > 1, at that point the infection continues and the malady state equilibrium E2 is locally asymptotically steady. The over two ends are drawn by breaking down the equilibrium focuses, when the measure of chaperone is zero. From the numerical delineation in area 3.5, we locate that an infection free state can be accomplished within the sight of a chaperone. The length of the infection free state is found to increment with the measure of chaperone and this measure of chaperone can be processed from the model.

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