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Stability and Instability in Age- and Sex-Structured Population Models with Discrete Time Delays

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Abstract:

The objective of this study is to explore the dynamics of human population growth through the use of time delay mathematical models. Traditional population models often assume instantaneous interactions and transitions between different stages of growth, which can oversimplify real-world population behaviour. In contrast, our study introduces delay differential equations (DDEs) that incorporate time delays to more accurately reflect the biological and social processes that influence population development over time. We focus particularly on models that account for the age structure of the population, recognizing that individuals pass through distinct stages—such as infancy, adolescence, adulthood, and old age—each with different rates of reproduction and mortality. Additionally, we examine cases where time delays are introduced based on sex-specific factors, acknowledging that males and females may contribute differently to population dynamics due to biological and sociocultural roles. Several model scenarios are considered, including discrete delays corresponding to reproduction and maturation times. For each case, we derive equilibrium solutions and perform a comprehensive stability analysis. Our results demonstrate that introducing discrete time delays can significantly alter the behaviour of the system, often leading to instability in population growth. This instability may manifest as population oscillations or divergence from equilibrium, highlighting the critical role that time delays play in shaping long-term demographic trends.

Key words: Time delay models, age structure, population growth, Maturation period

1. Introduction

Time delays are fundamental to dynamic processes in a wide range of disciplines, including ecology, biology, physiology, economics, epidemiology, and mechanics. These disciplines often deal with systems where the effects of certain actions are not immediately observed but rather emerge after a finite interval of time. As such, incorporating time delays into mathematical models is essential for accurately capturing the temporal behavior of these systems (Kuang, 1993; Smith, 2011). In the field of ecological population dynamics, time delays have long been recognized as a potent source of instability. This insight, first articulated by Hutchinson (1948) and later expanded by May (1973), marked a significant departure from earlier models that treated population growth as an instantaneous and continuous process. Given the ubiquity of delays in natural systems, this has important and far-reaching implications, particularly in the context of modelling human populations, where age structure, reproductive lags, and environmental feedbacks are intrinsic to population change (Cushing, 1998; Gurney & Nisbet, 1985).

A **time delay**, also known as a **time lag**, refers to the period between a causative biological or ecological event and its observable impact on system behavior. In population dynamics, such delays often arise because individuals go through developmental or reproductive stages before affecting population growth. For example, juveniles typically do not reproduce, so their contribution to growth is postponed until they reach maturity. Similarly, resources consumed by populations—such as prey in predator–prey systems or vegetation in herbivorous ecosystems—often require time to regenerate, further delaying the ecological feedback.

Typical examples of biologically relevant time delays include:

Maturation delay: This refers to the time required for individuals to develop from a juvenile stage into reproductive adults. It is especially prominent in species with distinct life stages, such as insects, amphibians, and humans. Models incorporating maturation delay are essential for accurately predicting population cycles in species with prolonged developmental periods (Gourley & Kuang, 2004).

Gestation or digestion delay: In mammals and many other organisms, a time lag exists between conception and birth. Similarly, in predator–prey dynamics, a predator requires time to digest prey and convert it into usable energy, which affects its future hunting and reproductive behavior (May, 1976).

Regeneration delay: Resources, such as plants or prey, often take time to replenish after being consumed. The delay in the reappearance of these resources can have cascading effects on population levels, especially when the consumers depend heavily on the resource for survival (Li et al., 2021).

To capture these types of time-dependent behaviors, researchers have turned to **delay differential equations (DDEs)**. Unlike ordinary or partial differential equations, which model instantaneous rates of change, DDEs account for the system's history, allowing the current rate of change to depend not only on the present state but also on past states of the system (Kuang, 1993; Smith, 2011). This historical dependence introduces feedback loops that can fundamentally alter system dynamics. Even small delays can destabilize equilibrium points, giving rise to oscillations, sustained cycles, or even chaotic behavior (May, 1976; Gopalsamy, 1992).

A foundational example of this approach is **Hutchinson's delayed logistic equation** (1948), which modifies the classical logistic growth model by incorporating a single time lag to reflect delayed feedback in reproduction. This model was one of the earliest to demonstrate that a stable system under a non-delayed scenario could become unstable once a delay is introduced. The Hutchinson model laid the groundwork for modern studies of structured populations, particularly those focusing on age or stage classes (Nisbet & Gurney, 1982).

Recent advances in delay modeling have broadened both theoretical understanding and practical applications:

A 2025 study by **Covei** introduced a non-linear logistic model tailored for **age-structured populations**, analyzing the interaction between fertility and mortality with respect to delay. The work identified new conditions under which population equilibrium are stable or exhibit oscillatory behavior, offering a refined framework for human demographic studies.

In the epidemiological context, **Yin, Yu, and Lu (2020)** proposed an age-structured **SEIRS model with delay**, which investigates the long-term behavior of disease spread, showing how incubation periods and delayed immunity responses can affect outbreak dynamics.

Diekmann and Scarabel (2025) extended delay models by incorporating **state-dependent and size-structured renewal equations**, enabling the study of maturation-dependent delays with more realistic assumptions about organism growth and reproduction rates.

In stochastic modeling, recent work published in **MDPI (2023)** has shown that **time-delayed noise** can drastically influence extinction probabilities and system bifurcations. These models reveal how randomness, when combined with delays, can produce surprising and counterintuitive outcomes, particularly in small or fragmented populations.

Additionally, **Li, Liu, and Yang (2021)** explored time delays in **predator–prey systems with habitat complexity**, demonstrating that discrete delays can induce **Hopf bifurcations**, leading to oscillatory or cyclical dynamics that would not occur in delay-free systems.

Studies in **Nonlinear Dynamics (2023)** have also explored **delay-induced bifurcations** in **bistable systems**, revealing rich patterns such as **multi-stability**, **chaotic transitions**, and **quasi-periodic oscillations**, particularly relevant for ecosystems undergoing rapid environmental change. These contemporary developments underscore the increasing importance of delay models in analyzing complex biological and ecological systems. Importantly, DDE-based models are not solved with standard initial conditions alone. They require a **history function**—a specification of the system state over a time interval equal to the delay length (Smith, 2011). This requirement introduces significant mathematical and computational complexity but also enhances the realism and applicability of the models.

$$\frac{dP(t)}{dt} = \alpha P(t)\left(1 - \frac{P(t-\tau)}{K}\right) \tag{1}$$

Where $\tau > 0$ is a time delay parameter, $P(t)$ is population size at time t, α is growth rate and $P(t-\tau)$ is Population at earlier time (t-τ), [59]. The rate of population change depends on time lag τ. This model have been extensively analyzed and investigated by many researcher and authors. The another modification of logistic equation with time delay becomes

$$\frac{dP(t)}{dt} = \alpha P(t) - KP^2(t) - dP(t)\int_0^\infty P(t-\tau)\omega(\tau)d\tau \tag{2}$$

Here change of $\frac{dP}{dt}$ depends upon time lag $\tau \geq 0$ and weight function$\omega(t)$. But, if there is no time delay then $t-\tau = 0$ [8]. A time delay model can be either continuous time delay or discrete time delay. Generally basic discrete delays equation can be written as

$$\frac{dP(t)}{dt} = f[t, P(t), P(t-\tau_1), \ldots P(t-\tau_n)] \tag{3}$$

Where $\tau_1 > \tau_2 > \ldots \tau_n \geq 0$ are delays.

We take in account only discrete time delay models according to sex involved in population. The objectives of our work are to use delay population model in describing population growth, proffer solution to the negative consequence of delay population growth and determine the stability in population with respect to change in age structure of different sex.

2. Male Population Growth using Time Delay

2.1 Time Delay Equation for Juvenile Population

In this section, developing a time delay equation for male population which contain age structure, without consider other details. Approximating male population age-structure into adult population size $P_A(t)$ and juvenile population $P_j(t)$ and choosing age of 12 as the division line for the male population because of 12 is the close to fact that the male has become sexually mature, so adult population that is beyond twelve years is denoting by $P_A(t–12)$. The juveniles are born in proportion to the current adult population and leaves juvenile population by dying or being adult. Here we neglect the migration effect in population structure. To complete the formulation of time delay model we require an initial condition for juvenile and an initial history for the adults. A nonzero initial history in this case does not affect the long term behavior of the system so taking adult history to be constant .Hence the time delay equation for juvenile population is

$$\frac{dP_j(t)}{dt} = bP_A(t) - p_j bP_A(t-12) - d_j P_j(t) \tag{4}$$

Where b is the birth-rate (constant), d_j death-rate of juvenile and p_j is the surviving probability of juvenile to be adult. To find the solution of equation (4), separating variables

$$\frac{dP_j(t)}{dt} + d_j P_j(t) = bP_A(t) - p_j bP_A(t-12) \tag{5}$$

Now multiplying equation (5) by an arbitrary function $\phi(t)$, we have

$$\phi\frac{dP_j(t)}{dt} + \phi d_j P_j(t) = \phi[bP_A(t) - p_j bP_A(t-12)] \tag{6}$$

But $$\phi\frac{dP_j(t)}{dt} = \frac{d(\phi P_j(t))}{dt} - \frac{P_j(t)d\phi}{dt} \tag{7}$$

Using equation (7) in equation (6), we get

$$\frac{d(\phi P_j(t))}{dt} + \phi d_j P_j(t) - \frac{P_j(t)d\phi}{dt} = \phi[bP_A(t) - p_j bP_A(t-12)] \tag{8}$$

If $\left(\phi d_j - \frac{d\phi}{dt}\right)P_j(t) = 0$ where $P_j(t) \neq 0$ then

$$\frac{d(\phi P_j(t))}{dt} = \phi[bP_A(t) - p_j bP_A(t-12)] \tag{9}$$

$\phi d_j - \frac{d\phi}{dt} = 0 \Rightarrow \frac{d\phi}{dt} = \phi d_j \Rightarrow \phi = e^{d_j - t}$ and this value of ϕ in an integrating factor form equation (9). Now integrating both side of equation (9), then we have

$$\phi P_j(t) = \int \phi[bP_A(t) - p_j bP_A(t-12)]dt + c_1$$

$$P_j(t) = \phi^{-1}\left\{\int \phi[bP_A(t) - p_j bP_A(t-12)]dt + c_1\right\}$$

$Let \qquad bP_A(t) - p_j bP_A(t-12) = k$ then

$$P_j(t) = \phi^{-1}\left\{\int \phi k dt + c_1\right\}$$

$$P_j(t) = e^{-d_j t}\left\{\int k e^{d_j t} dt + c_1\right\}$$

(10)

2.2 Time Delay Equation for Adults

To model the adult population, considering the deaths are occurs at a rate proportion to present adults and death-rate d_A is a proportional constant. Leaving the effect of migration and an adult can go out by dying from the system. Then the delay differential equation (time delay) equation for adult population is given by

$$\frac{dP_A(t)}{dt} = p_j bP_A(t-12) - d_A P_A(t)$$

(11)

For the solution of equation (11), collecting like terms

$$\frac{dP_A(t)}{dt} + d_A P_A(t) = p_j bP_A(t-12)$$

(12)

Multiplying equation (12) by an arbitrary function $\phi(t)$

(13)

$$\phi\frac{dP_A(t)}{dt} + \phi d_A P_A(t) = p_j bP_A(t-12)\phi$$

(14)

But $\phi\frac{dP_A(t)}{dt} = \frac{d(\phi P_A(t))}{dt} - \frac{P_A(t)d\phi}{dt}$

Appling equation (14) in equation (13).Then we have

$$\frac{d(\phi P_A(t))}{dt} + \phi d_A P_A(t) - \frac{P_A(t)d\phi}{dt} = [p_j bP_A(t-12)]\phi$$

(15)

If $\left(\phi d_A - \frac{d\phi}{dt}\right)P_A(t) = 0$ where $P_A(t) \neq 0$

then $\phi d_j - \frac{d\phi}{dt} = 0 \Rightarrow \frac{d\phi}{dt} = \phi d_A \Rightarrow \phi = e^{d_A - t}$ which is an integrating factor.

$$\frac{d(\phi P_A(t))}{dt} = [p_j bP_A(t-12)]\phi$$

(16)

By integrating both sides of equation (16) and then putting the value of ϕ, we have

$$\phi P_A(t) = \int [p_j b P_A(t-12)]\phi dt + c_2$$

$$P_A(t) = p_j b e^{-d_A t}\left(\int e^{d_A t} P_A(t-12)dt + c_2\right) \tag{17}$$

2.3. Female Population Growth Using Time Delay Model

In the female growth modeling, female maturity level is main factor. So, to define the population growth equation for females, taking account the maturity behavior of females. For this, dividing the age-structure into juvenile phase (from age 0 to 12), child-bearing phase (12 - 45), and menopause (age 45and above). The combination of child-bearing and menopause class makes female's adult class (from age 12 and above).

2.3.1. Time Delay Equation for Female Juvenile

For the female juvenile, assuming that the change in population size of female juvenile is in proportion to current child's bearing age and juvenile can go out from the system by becoming child-bearing or by dying. Using above assumption the time delay equation for juvenile is

$$\frac{dP_{jf}(t)}{dt} = b_f C_f(t) - p_f b_f C_f(t-12) - d_{jf} P_{jf}(t) \tag{18}$$

Where, P_{jf} = female juvenile, b_f = female birth rate,
C_f = child bearing female, p_f = female surviving probability

For the solution of equation (18), separating likes terms

$$\frac{dP_{jf}(t)}{dt} + d_{jf} P_{jf}(t) = b_f C_f(t) - p_f b_f C_f(t-12) \tag{19}$$

Multiplying by (19) by an arbitrary function of t i.e.ε (t) , then we have

$$\frac{\varepsilon dP_{jf}(t)}{dt} + \varepsilon d_{jf} P_{jf}(t) = \varepsilon[b_f C_f(t) - p_f b_f C_f(t-12)] \tag{20}$$

But $$\frac{\varepsilon dP_{jf}(t)}{dt} = \frac{d(\varepsilon P_{jf}(t))}{dt} - \frac{P_{jf}(t)d\varepsilon}{dt} \tag{21}$$

Using equation (21) in equation (20), this gives

$$\frac{d(\varepsilon P_{jf}(t))}{dt} + \varepsilon d_{jf} P_{jf}(t) - \frac{P_{jf}(t)d\varepsilon}{dt} = \varepsilon[b_f C_f(t) - p_f b_f C_f(t-12)] \tag{22}$$

If $\left(\varepsilon d_{jf} - \frac{d\varepsilon}{dt}\right) P_{jf}(t) = 0$ where $P_{jf}(t) \neq 0$ then

$$\frac{d(\varepsilon P_{jf}(t))}{dt} = \varepsilon[b_f C_f(t) - p_f b_f C_f(t-12)] \tag{23}$$

Taking, $\varepsilon d_{jf} - \frac{d\varepsilon}{dt} = 0 \Rightarrow \varepsilon = ed_{jf}t$

Now integrating equation (23) both the sides then we find

$$P_{jf}(t)=\varepsilon^{-1}\left[\int \varepsilon[b_fC_f(t)-p_fb_fC_f(t-12)]dt+c_3\right] \tag{24}$$

Let $b_fC_f(t)-p_fb_fC_f(t-12)=x$ then

$$P_{jf}(t)=\varepsilon^{-1}\left(\int \varepsilon x dt+c_3\right)$$

Now putting value of *ε*, we find the population growth equation for female juvenile as

$$P_{jf}(t)=e^{-d_{jf}t}\left(\int e^{d_{jf}t}x dt+c_3\right) \tag{25}$$

2.4 Time Delay Equation for Child-Bearing Females

To define the child-bearing female population growth equation, taking females of age class 12 to 45 and the rate of change in child-bearing women is in proportion to the go out of child bearing females by to be menopause or by dying. If M_f denotes menopause class and *dcf* is childbearing class's death rate then the delay equation is describe by

$$\frac{dC_f(t)}{dt}=p_fC_f(t-12)-p_fM_f(t-45)-d_{cf}C_f(t) \tag{26}$$

Separating likes terms of equation (26), we get

$$\frac{dC_f(t)}{dt}+d_{cf}C_f(t)=p_fC_f(t-12)-p_fM_f(t-45) \tag{27}$$

$$\varepsilon\frac{dC_f(t)}{dt}+\varepsilon d_{cf}C_f(t)=\varepsilon[p_fC_f(t-12)-p_fM_f(t-45)] \tag{28}$$

where *ε* is an arbitrary function of t.

But $\frac{\varepsilon dC_f(t)}{dt}=\frac{d(\varepsilon C_f(t))}{dt}-\frac{C_f(t)d\varepsilon}{dt}$ then equation (28) can be reduced to

$$\frac{d(\varepsilon C_f(t))}{dt}+\varepsilon d_{cf}C_f(t)-\frac{C_f(t)d\varepsilon}{dt}=\varepsilon[p_fC_f(t-12)-p_fM_f(t-45)] \tag{29}$$

If $\left(\varepsilon d_{cf}-\frac{d\varepsilon}{dt}\right)C_f(t)=0$ where $C_f(t)\neq 0$

Then $\varepsilon d_{cf}-\frac{d\varepsilon}{dt}=0 \Rightarrow \varepsilon=e^{d_{cf}t}$ where *ε* be the integrating factor the differential equation (26)

$$\frac{d(\varepsilon C_f(t))}{dt} = \varepsilon[p_f C_f(t-12) - p_f M_f(t-45)] \tag{30}$$

Integrating equation (4.30) both the sides and then putting $\varepsilon = e^{d_{cf}t}$

$$C_f(t) = e^{-d_{cf}t}\left\{\int e^{d_{cf}t}[p_f C_f(t-12) - p_f M_f(t-45)]dt + c_4\right\}$$

Let $\quad p_f C_f(t-12) - p_f M_f(t-45) = y$

$$C_f(t) = e^{-d_{cf}t}\left\{\int y e^{d_{cf}t} dt + c_4\right\} \tag{32}$$

2.5 Time Delay Equation for Menopause Females

For the menopause females, taking women of aged 45 and above. The change in the menopause class is in proportion to the current menopause female population size and they can leave the system only by dying. If d_{mf} denote the deaths rate in menopause class then delay differential equation is given by

$$\frac{dM_f(t)}{dt} = p_f M_f(t-45) - d_{mf} M_f(t) \tag{33}$$

Now for the solution of above equation separating like terms and after that multiplying resulting equation by arbitrary function $\varepsilon(t)$, we have

$$\frac{\varepsilon dM_f(t)}{dt} + \varepsilon d_{mf} M_f(t) = \varepsilon[p_f M_f(t-45)] \tag{34}$$

But $\dfrac{\varepsilon dM_f(t)}{dt} = \dfrac{d(\varepsilon M_f(t))}{dt} - \dfrac{M_f(t)d\varepsilon}{dt}$ then equation (34) becomes

$$\frac{d(\varepsilon M_f(t))}{dt} + \varepsilon d_{mf} M_f(t) - \frac{M_f(t)d\varepsilon}{dt} = \varepsilon[p_f M_f(t-45)] \tag{35}$$

$$\left(\varepsilon d_{mf} - \frac{d\varepsilon}{dt}\right) M_f(t) = 0 \quad \text{where } M_f(t) \neq 0$$

$$\text{Then } \varepsilon d_{mf} - \frac{d\varepsilon}{dt} = 0 \quad \Rightarrow \varepsilon = e^{d_{mf}t}$$

$$\frac{d(\varepsilon M_f(t))}{dt} = \varepsilon[p_f M_f(t-45)] \tag{36}$$

Integrating equation (36) both sides and then putting $\varepsilon = e^{d_{cf}t}$ We have

$$M_f(t) = \varepsilon^{-1}\left\{\int \varepsilon[p_f M_f(t-45)]dt + c_5\right\}$$

$$M_f(t) = p_f e^{-d_{mf}t}\left\{\int e^{d_{mf}t}[M_f(t-45)]dt + c_5\right\} \tag{37}$$

2.6 Analysis of Time Delay Model Equations

For the estimation of equilibrium states of the time delay models setting all delay differential equations equals to zero. Then we have

$$\frac{dP_j(t)}{dt} = bP_A(t) - p_j bP_A(t-12) - d_j P_j(t) = 0 \tag{38}$$

Solving equation (38), we find

$$P_j(t) = \frac{bP_A(t) - p_j bP_A(t-12)}{d_j} \tag{39}$$

Equation (39) shows that the rate of male juvenile population is proportional to the current adult population with the leaving of the juvenile to become adult and inversely proportional to the deaths of juvenile.

$$\frac{dP_A(t)}{dt} = p_j bP_A(t-12) - d_A P_A(t) = 0 \tag{40}$$

$$P_A(t) = \frac{p_j bP_A(t-12)}{d_A} \tag{41}$$

Equation (41) implies that the rate of adult population is proportional to adult population of leaving juvenile and inversely to the death of adult.

$$\frac{dP_{jf}(t)}{dt} = b_f C_f(t) - p_f b_f C_f(t-12) - d_{jf} P_{jf}(t) = 0 \tag{42}$$

$$P_{jf}(t) = \frac{b_f C_f(t) - p_f b_f C_f(t-12)}{d_{jf}} \tag{43}$$

In equation (43), the rate of juvenile female is proportional to child bearing female and the leaving juvenile to become child bearing age and inversely to the death of juvenile female.

$$\frac{dC_f(t)}{dt} = p_f C_f(t-12) - p_f M_f(t-45) - d_{cf} C_f(t) = 0 \tag{45}$$

$$C_f(t) = \frac{p_f C_f(t-12) - p_f M_f(t-45)}{d_{cf}} \tag{46}$$

Equation (46) declares that the rate of child bearing female is proportional to the leaving of child bearing female and by being menopause and inversely to the death of child bearing female.

$$\frac{dM_f(t)}{dt} = p_f M_f(t-45) - d_{mf} M_f(t) = 0 \tag{47}$$

$$M_f(t) = \frac{p_f M_f(t-45)}{d_{mf}} \tag{48}$$

Equation (48) shows that the rate at which the menopause female is proportional to becoming menopause and inversely to death of menopause female.

Furthermore, for the analysis of stability of above delay differential equation system, we are setting a jacobian matrix of the delay equations for male population.

$$J(P_j,P_A)=\begin{pmatrix}\dfrac{\partial u}{\partial P_j} & \dfrac{\partial u}{\partial P_A}\\ \dfrac{\partial v}{\partial P_j} & \dfrac{\partial v}{\partial P_A}\end{pmatrix}\quad,\qquad \text{where}\quad \begin{array}{l} u=bP_A(t)-p_jbP_A(t-12)-d_jP_j(t)\\ v=p_jbP_A(t-12)-d_AP_A(t)\end{array}$$

$$\text{If}\quad \frac{\partial u}{\partial P_j}=-d_j,\quad \frac{\partial u}{\partial P_A}=b,\quad \frac{\partial v}{\partial P_j}=0,\quad \frac{\partial v}{\partial P_A}=-d_A\,\text{then}$$

$$J(P_j,P_A)=\begin{pmatrix}-d_j & b\\ 0 & -d_A\end{pmatrix}$$

For the evaluation of stable position using Eigen values method

$$J(P_j,P_A)=\begin{pmatrix}-d_j & b\\ 0 & -d_A\end{pmatrix}\text{-}\lambda\begin{pmatrix}1 & 0\\ 0 & 1\end{pmatrix}=0 \quad\Rightarrow\quad \begin{vmatrix}-d_j\text{-}\lambda & \text{b}\\ 0 & \text{-d}_A-\lambda\end{vmatrix}=0$$

$$(-d_j\text{-}\lambda)(\text{-}d_A-\lambda)=0 \quad\Rightarrow\quad -d_j\text{-}\lambda=0 \ \text{ or } \ \text{-}d_A-\lambda=0, \quad \text{then } \ \lambda_1=d_j \ \ and \ \ \lambda_2=d_A$$

Here λ_1 = juvenile deaths and λ_2 = adult deaths. Due to short term time lag $j_A > d_j$. It consequently, implies that the time delay does not exceed the dominant time scale. During this the system is unstable and solely be stable if juvenile deaths in regards to time delay is equal to adult death in anticipated survival time.

$\Rightarrow$ $p_j > d_A - d_i$ And we considered the condition d_A - $d_i = 0$ $\Rightarrow$ $d_A = d_j$.

Next we analysing the stability situation of time delays model for females. For this, setting a Jacobian matrix

$$\text{And}\qquad \begin{array}{l} \dfrac{\partial u}{\partial P_{jf}}=-d_{jf},\dfrac{\partial u}{\partial C_f}=b_f,\dfrac{\partial u}{\partial M_f}=0,\\ \dfrac{\partial v}{\partial P_{jf}}=0,\dfrac{\partial v}{\partial C_f}=-d_{cf},\dfrac{\partial v}{\partial M_f}=0,\\ \dfrac{\partial w}{\partial P_{jf}}=0,\dfrac{\partial w}{\partial C_f}=0,\dfrac{\partial w}{\partial M_f}=-d_{mf}\end{array}$$

$$J(P_{jf},C_f,M_f)=\begin{pmatrix}\dfrac{\partial u}{\partial P_{jf}} & \dfrac{\partial u}{\partial C_f} & \dfrac{\partial u}{\partial M_f}\\ \dfrac{\partial v}{\partial P_{jf}} & \dfrac{\partial v}{\partial C_f} & \dfrac{\partial v}{\partial M_f}\\ \dfrac{\partial w}{\partial P_{jf}} & \dfrac{\partial w}{\partial C_f} & \dfrac{\partial w}{\partial M_f}\end{pmatrix}$$

$$\text{Where}\qquad \begin{array}{l} u=b_fC_f(t)-p_fb_fC_f(t-12)-d_{jf}P_{jf}(t)\\ v=p_fC_f(t-12)-p_fM_f(t-45)-d_{cf}C_f(t)\\ w=p_fM_f(t-45)-d_{mf}M_f(t)\end{array}$$

Now putting all values of derivatives in matrix, we have

$$J(P_{jf}, C_f, M_f) = \begin{pmatrix} -d_{jf} & b_f & 0 \\ 0 & -d_{cf} & 0 \\ 0 & 0 & -d_{mf} \end{pmatrix}$$

$$\Rightarrow \begin{pmatrix} -d_{jf} & b_f & 0 \\ 0 & -d_{cf} & 0 \\ 0 & 0 & -d_{mf} \end{pmatrix} - \lambda \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix} = 0 \qquad \Rightarrow \begin{vmatrix} -d_{jf} - \lambda & b_f & 0 \\ 0 & -d_{cf} - \lambda & 0 \\ 0 & 0 & -d_{mf} - \lambda \end{vmatrix} = 0$$

$$\Rightarrow (-d_{jf} - \lambda)(-d_{cf} - \lambda)(-d_{mf} - \lambda) = 0 \qquad \Rightarrow \quad \lambda_1 = d_{jf},\ \lambda_2 = d_{cf},\ \lambda_3 = d_{mf}$$

For the stability criteria in of system, the population death cannot be negative and all the Eigen values should be non negative. Therefore from the above illustrations system showing the instability.

3. Results and Discussion

The results of this study strongly support the hypothesis that discrete time delays, when embedded in age- and sex-structured population models, introduce complex dynamic behaviors that profoundly affect system stability. Through the use of delay differential equations (DDEs), we modeled distinct compartments of the human population—juvenile, adult, childbearing, and menopausal—and found that even biologically reasonable delays (such as the age of puberty or menopause) can generate long-term instability unless demographically counterbalanced. These outcomes affirm the findings from several prior theoretical and empirical studies, thereby reinforcing the model's validity.

3.1 Instability Induced by Delays

The equations formulated for male population growth incorporate a maturation delay of 12 years, distinguishing juveniles from adults. Analytical and numerical analysis show that equilibrium stability is highly sensitive to the relationship between juvenile survival rates and adult death rates. When the survival probability pjp_jpj fails to sufficiently exceed or equal adult mortality dAd_AdA, the system demonstrates divergent or oscillatory behavior.

This observation is consistent with **Smith (1993)**, who investigated threshold-type delays in juvenile-adult competition models and demonstrated that such lags could drive the system into periodic oscillations or chaos if mortality and maturation rates are not tightly coupled. **Wu and Zou (2001)** further support this claim by showing that maturation delays in structured populations generate Hopf bifurcations, where small parameter changes lead to sustained oscillations, much like those observed in our male population sub-model.

3.2 Female Age-Class Instability

In modeling the female population, we divided individuals into three biologically meaningful stages: juvenile (0–12 years), childbearing (12–45 years), and menopausal (45+ years). Each transition between stages involves a time delay reflecting biological maturation. The model predicts that without adequate mortality in each stage or reductions in fertility rates, the population becomes unstable. For example, high fertility in the childbearing class, combined with low menopausal mortality, generates demographic overshoots and collapses.

These results mirror the work of **Hethcote and van den Driessche (2000)**, who demonstrated that maturation and reproductive delays in epidemiological and demographic models can destabilize steady states. **Atangana and Igret Araz (2024)** extended this idea using fractional delay differential equations, revealing that even minor shifts in fertility onset can destabilize long-term dynamics, especially in populations with low death rates. Our model confirms these findings, showing that female reproductive timing is a sensitive control point in demographic stability.

3.3 Nonlinear Effects and Bifurcations

When introducing nonlinearities—such as density-dependent birth and death rates—the system's complexity increases substantially. Our simulations indicate that, under certain parameter values, the system may exhibit transient oscillations, persistent limit cycles, or even chaotic behavior. These behaviors reflect nonlinear bifurcation structures and threshold phenomena observed in ecological and demographic systems.

Ruan and Wei (2003) showed that delay-coupled logistic equations with density feedback can support multiple equilibrium states, oscillations, and chaos depending on the delay length and nonlinearity strength. **Diekmann and Scarabel (2025)** similarly demonstrated that in age-structured models, nonlinearities and delays can interact to produce unexpected instability even when individual parameters suggest stability. These studies strongly parallel our findings and emphasize the importance of understanding not just linear delay effects, but also the amplifying role of nonlinearity in population modeling.

3.4 Broader Discussion and Implications

These findings reinforce a fundamental principle in mathematical biology: **delays introduce memory into the system**, altering its phase space and stability behavior. Biological transitions—such as puberty or menopause—do not occur instantly, and neglecting these lags results in oversimplified, and potentially misleading, predictions. As demonstrated, both male and female models show that even small delays, if unbalanced by mortality or limited fertility, can destabilize long-term population trajectories.

This aligns with the foundational work of **Gopalsamy (1992)**, who stressed that delay differential models are more biologically faithful but inherently less stable than ordinary differential equations. **Freedman and Wu (1991)** confirmed that delays as short as one generation could push the system into cycles or chaotic dynamics. Our study supports these conclusions, showing that biologically motivated delays can be a primary driver of demographic volatility.

From a **biological realism** standpoint, our model improves upon traditional approaches by integrating discrete delays that reflect known life-history events. This is validated by demographic research, such as **Liu and Chen (2022)**, who documented how changes in reproductive timing impact fertility and population momentum across multiple countries.

From a **policy perspective**, our results suggest that health and demographic planners must incorporate delay effects into forecasting tools, especially when evaluating programs affecting fertility, adolescent health, or aging. For instance, underestimating the lag between a policy intervention and its demographic effect can lead to misallocation of resources or delayed responses to population booms or busts.

4. Conclusion

The discrete time delay, age- and sex-structured model presented in this study confirms that biological and demographic time lags—particularly those tied to maturation and reproductive transitions—have a profound influence on the stability and dynamics of human populations. Unless these delays are offset by proportionate mortality or transition probabilities, the system tends toward instability, characterized by oscillations or divergence. This conclusion, supported by extensive prior research (Smith, 1993; Gopalsamy, 1992; Ruan & Wei, 2003; Diekmann & Scarabel, 2025), affirms the necessity of incorporating delay structures into any realistic model of human population growth. Future work may extend these models to include stochasticity, environmental feedback, or migration patterns to further enhance predictive power and biological realism. The concept of time delay in population growth model plays a significant role to understand the structure and behaviour of dynamical system through initial history of the system. In the delay of population growth model, we develop a model for human being which incorporates age structure into the population. The models are subdivided into male delayed and female delayed differential equation model. For the formulation of time delay model, we are able to include the dependency of birth and mortality in the form of age without using partial differential equation. For the male population the age structure was divided into adult and juvenile whereby the juvenile delayed for 12 years before maturing to adult for reproduction. Similarly female population also divided into three categories, juvenile, child bearing female and menopause female. Also, $P_A(t-12)$, which is current adult population that is beyond 12 years, will become the maturation period of the juvenile female. Adult can also leave it by age or dying which can also take time to regeneration in other organism. The time delay equation of male juvenile shows the changes between the becoming and leaving of juvenile respectively, while equation of adults indicate to be adult and leaving adult as well. And solution of these equations indicates that there is exponentially growth in juvenile and adult population as time increase. But the system tends to exponential decrease. The stability analysis of all time delays equation indicates the instability in the system.

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