Iranian Journal of Fuzzy Systems

Volume 23, Number 4, (2026), pp. 35-54

Original paper

Passivity and dissipativity criteria of discrete-time fractional-order fuzzy genetic regulatory networks

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Abstract

Genetic Regulatory Networks (GRNs) constitute a key framework for understanding the development and evolutionary dynamics of biological systems. With the rapid progress of DNA microarray technologies, large-scale genome-wide analysis of GRNs has become feasible. In this work, we investigate the passivity and dissipativity of Fractional-Order Discrete-Time Fuzzy Genetic Regulatory Networks (FODTFGRNs). Embedding fractional-order operators in the discrete-time formulation allows the model to capture memory-dependent and hereditary features of gene regulatory dynamics. Meanwhile, fuzzy logic techniques are introduced to handle parameter ambiguities and nonlinear gene interactions. This integrated modeling strategy leads to a more accurate and practical representation of genetic regulation phenomena encountered in real biological and medical applications. Moreover, a novel passivity lemma tailored to the considered systems is developed through the construction of a suitable Lyapunov functional. Several sufficient criteria guaranteeing passivity and dissipativity are established by combining the Linear Matrix Inequalities (LMIs) framework with Lyapunov functional analysis, the comparison principle, contradiction arguments, various inequality techniques, and the newly developed passivity lemma. Finally, two simulation examples are presented to validate and illustrate the effectiveness of the proposed theoretical results.

Keywords: Discrete-time, fractional-order, fuzzy genetic regulatory networks, passivity, dissipativity.

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1 Introduction

Genetic regulatory networks (GRNs) represent intricate biological systems in which DNA, RNA, proteins, and other biomolecules interact to control gene expression. These mechanisms are fundamental to numerous cellular processes, including development, differentiation, and adaptation to environmental stimuli. The proper functioning of GRNs is essential for cellular homeostasis, and disruptions in their dynamics are often associated with severe pathologies, such as cancer and genetic disorders. To better understand these complex regulatory interactions, mathematical modeling has become an indispensable tool. Different approaches have been proposed, including ordinary differential equations [1], difference equations [31], and Boolean logic frameworks [29].

A key challenge in GRN modeling lies in accounting for biological delays caused by transcription, translation, and regulatory feedback mechanisms. These time delays are inherent to gene expression and must be considered to accurately capture the temporal dynamics of the system. Over the past decades, several models have been designed to investigate the dynamic behavior of mRNA and protein concentrations [4], providing valuable insights for both theoretical biology and medical applications. As research in this area progresses, it becomes increasingly necessary to adopt modeling strategies capable of incorporating such delays while reflecting the inherent complexity of gene regulation.

Corresponding Author: C. Aouiti

Received: October 2025; Revised: February 2026; Accepted: June 2026.
https://doi.org/10.22111/ijfs.2026.53695.9507

In this context, fractional-order systems have recently gained attention as powerful tools for modeling dynamical processes with memory and hereditary effects. In contrast to traditional integer-order models, they incorporate nonlocal effects, where the current behavior is influenced by past states. This property is particularly relevant in biology [24], where memory effects and delays are intrinsic to processes such as gene regulation. Although initially underexplored due to limited theoretical foundations, fractional calculus has since been widely adopted as an effective framework for representing natural phenomena with fractional-dimensional characteristics. Consequently, the fractional-order modeling has been successfully applied to areas such as neural networks [7, 18] and biological regulatory systems [8, 13], thereby offering a promising avenue for advancing GRN studies.

Complementary to this, fuzzy logic has been introduced to address the uncertainty and imprecision that are inherent to biological systems. Traditional mathematical models often require precise parameters and initial conditions, which are difficult to obtain due to biological variability and experimental limitations. Fuzzy systems overcome this limitation by describing regulatory interactions using fuzzy sets and linguistic rules [19]. This approach allows for the representation of graded responses and partial activations frequently observed in genetic interactions, offering a more realistic depiction than binary logic models [32]. Furthermore, integrating fuzzy logic with a fractional-order models enables researchers to simultaneously account for uncertainties and memory effects, resulting in models with improved accuracy and robustness.

Beyond uncertainty and memory, another crucial aspect of GRN analysis is the system's energy-based behavior, often studied through the concepts of passivity and dissipativity. These notions have been extensively investigated in nonlinear systems with memory and delays, primarily in continuous-time frameworks [15]. However, their extension to discrete-time fractional-order systems, especially fuzzy GRNs, remains relatively recent and still under development [25]. Passivity provides a rigorous framework for ensuring internal stability by constraining how the system exchanges energy with its environment [23]. Dissipativity generalizes this idea by introducing supply functions that describe whether the system stores or dissipates energy, depending on its input–output behavior [28].

Nonlinear control theory provides a broad range of methodologies for regulating complex dynamical systems, among which fuzzy control has proven highly effective in handling nonlinearities and uncertainties across various engineering applications. A recent comprehensive survey emphasizes the expanding practical relevance of fuzzy control in modern systems, particularly in mechatronics, where rigorous stability, robustness, and performance guarantees are systematically developed [20]. Beyond controller synthesis, structural properties such as stability and sensitivity analysis of fuzzy systems have been investigated using advanced tools including Popov's hyperstability theory, with experimental validation on mechatronic servo systems [21]. In autonomous robotics, fuzzy logic has enabled efficient shortest-path planning and obstacle avoidance in static and dynamic indoor environments [12], while adaptive fuzzy-enhanced model-free global sliding mode control schemes have been proposed to address input saturation and mismatched disturbances in constrained mechatronic systems [11]. These studies demonstrate the strong practical impact and robustness of nonlinear and fuzzy control methodologies in engineering domains. However, their application to genetic regulatory networks (GRNs) remains relatively limited, especially when discrete-time fractional-order dynamics, intrinsic biological delays, and energy-based robustness properties must be considered simultaneously. Motivated by these challenges, the present work proposes a discrete-time fractional-order fuzzy GRN framework in which fuzzy MIN/MAX operators are employed solely to model regulatory uncertainty, while the controller itself is designed as a linear delayed state-feedback law synthesized via LMI conditions derived from a fractional Lyapunov functional, thereby ensuring passivity and global controller dissipativity. These energy-based guarantees provide bounded internal energy evolution and robustness against external perturbations, highlighting the potential practical relevance of the proposed theoretical results for the robust regulation and analysis of uncertain, memory-driven biological networks.

Motivated by the preceding discussion, in this manuscript, we bridge this gap by establishing new passivity and dissipativity criteria for a class of FODTFGRNs. First, we formulate a discrete-time fuzzy model that incorporates both the hereditary nature of fractional-order operators and the linguistic uncertainty of gene regulatory mechanisms. Then, by constructing suitable discrete fractional Lyapunov functions and applying LMIs techniques, we derive novel requirements ensuring the passivity and dissipativity of the developed framework. Compared to existing works, our approach handles more realistic features: it considers time delays, captures long-term memory effects, and integrates fuzzy logic to reflect biological ambiguity. The primary contributions are outlined below:

1. The framework presented in this study combines simultaneously discrete fractional-order dynamics, fuzzy logic rules, genetic regulatory networks, and discrete-time delays, thereby significantly broadening the scope of previous models in the literature.

2. This work introduces an original passive lemma tailored to fractional-order discrete-time fuzzy genetic regulatory networks, thereby bridging the current gap in passivity analysis for such systems.

3. Using fractional Lyapunov functionals, LMI-based techniques, Laplace transform arguments, and inequalities from fractional calculus, sufficient requirements are provided to ensure the passivity and dissipativity of the proposed framework, which are achieved through a delayed state-feedback controller.

4. Numerical examples to validate the theoretical findings.

This manuscript is organized in the following manner: Section 2 reviews the fundamentals of fractional calculus and presents a detailed formulation of the proposed model. Section 3 establishes its passivity and dissipativity. Section 4 reports numerical simulations for validation. Section 5 provides concluding observations.

2 Preliminaries

Notations: Let $\mathbb{Z}^+$ denote the set of positive integers. The notation $\mathbb{N}_x$ refers to the sequence beginning at x with unit increments.
For any $x \in \mathbb{R}$, define $\urcorner(x) = x - 1$, and

$$\mathbb{N}_x = \{x,\ x+1,\ x+2, \cdots\}, \quad \mathbb{N}_x^{\epsilon} = \{x,\ x+1,\ x+2, \cdots,\ \epsilon\},$$

where $\epsilon - x = k$ with $k \in \mathbb{N}_1$. For $t \in \mathbb{N}_{x+1}$, the nabla difference of a function $\mathfrak{f}$ is given by

$$\nabla\mathfrak{f}(t) = \mathfrak{f}(t) - \mathfrak{f}(t-1).$$

In matrix formulations, the symbol $\star$ indicates the transposed block ensuring symmetry.
We begin by recalling key definitions and lemmas concerning fractional-order differences.

Definition 2.1. [10] *The κ-th order rising function is defined as*

$$t^{\overline{\kappa}} = \frac{\Gamma(t+\kappa)}{\Gamma(t)},\ 0^{\overline{\kappa}} = 0,$$

where $\kappa \in \mathbb{R}\backslash\{\cdots, -2, -1\}$ and $t \in \mathbb{N}_0$.

Definition 2.2. [10] *Let $\kappa > 0$ and $\phi : \mathbb{N}_x \to \mathbb{R}$, the nabla κ-th order sum of $\phi(t)$ is defined as*

$$\nabla_x^{-\kappa}\phi(t) = \frac{1}{\Gamma(\kappa)} \sum_{\pi=x+1}^{t} (t - \urcorner(\pi))^{\overline{\kappa-1}}\phi(\pi),$$

where $t \in \mathbb{N}_{x+1}$.

Definition 2.3. [10] *For $\kappa > 0$ and $\phi : \mathbb{N}_x \to \mathbb{R}$, the nabla κ-th order Caputo difference of $\phi(t)$ is defined as*

$${}^{C}\nabla_x^{\kappa}\phi(t) = \nabla_x^{-(\delta-\kappa)}\nabla^{\delta}\phi(t) = \frac{1}{\Gamma(\delta-\kappa)} \sum_{\pi=x+1}^{t} (t - \urcorner(\pi))^{\overline{\delta-\kappa-1}}\nabla^{\delta}\phi(\pi),$$

where $x \in \mathbb{R},\ t \in \mathbb{N}_{x+1}$, $\delta = [\kappa] + 1$ and $[\kappa]$ is the largest integer less than κ.

According to definitions 2.2 and 2.3, we have the following properties:

1. ${}^{C}\nabla_x^{\kappa}\hbar = 0,\ \forall\hbar \in \mathbb{R}$,

2. $\nabla_x^{-\kappa_1}\nabla_x^{-\kappa_2}\phi(\hbar) = \nabla_x^{-(\kappa_1+\kappa_2)}\phi(\hbar)$,

3. $\nabla_x^{-\kappa}\ {}^{C}\nabla_0^{\kappa}\phi(\hbar) = \phi(\hbar) - \phi(0),\ \kappa \in (0,\ 1)$.

In this work, we concentrate on analyzing the passivity and global dissipativity of the FODTFGRNs described below:

$$\left\{\begin{array}{lll}
{}^{C}\nabla_0^{\kappa}\chi_x(t) & = & -b_x\chi_x(t) + \sum\limits_{y=1}^{n}\beta_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \bigwedge\limits_{y=1}^{n}\varrho_{xy}\mathfrak{f}_y(\eta_y((t-\alpha_1)) \\
 & + & \bigvee\limits_{y=1}^{n}\varphi_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \sigma_{1x}(t) + I_x(t), \\
{}^{C}\nabla_0^{\kappa}\eta_x(t) & = & -a_x\eta_x(t) + \psi_x\chi_x(t-\alpha_2) + \bigwedge\limits_{y=1}^{n}\bar{\varrho}_{xy}\chi_y(t-\alpha_2) \\
 & + & \bigvee\limits_{y=1}^{n}\bar{\varphi}_{xy}\chi_y(t-\alpha_2) + \sigma_{2x}(t) + J_x(t), \\
y_{1x}(t) & = & \hbar_{1x}\chi_x(t) + \wp_{1x}\sigma_{1x}(t), \\
y_{2x}(t) & = & \hbar_{2x}\eta_x(t) + \wp_{2x}\sigma_{2x}(t),
\end{array}\right. \tag{1}$$

where

- $*$ $x \in \aleph = \{1,\ 2, \cdots, n\}$, ${}^{C}\nabla^{\kappa}{}_0$ is the Caputo fractional nabla operator of order κ $(0 < \kappa < 1)$;
- $*$ $\chi_x(\cdot)$ specifies the mRNA concentration, while $\eta_x(\cdot)$ specifies the protein concentration for the x^{th} node.
- $*$ a_x characterizes the rate at wich protein degrades, whereas, b_x corresponds to the degradation of mRNA.
- $*$ The nonlinear monotonic function $\mathfrak{f}(\cdot)$ used to describe protein feedback regulation, is generally expressed as a Hill function:

$$\mathfrak{f}_y(\zeta_y) = \frac{\left(\frac{\zeta_y}{\tau_y}\right)^{H_y}}{\left(1 + (\frac{\zeta_y}{\tau_y})^{H_y}\right)}, \tag{2}$$

  where H_y being the Hill coefficient and $\tau_y > 0$.
- $*$ α_1 and α_2 are the time delays.
- $*$ ψ_x represents the translation rate.
- $*$ $\varrho_{xy}, \bar{\varrho}_{xy}$ represent entries of the fuzzy feedback MIN template. φ_{xy}, $\bar{\varphi}_{xy}$ are associated with the MAX template.
- $*$ $\bigwedge$ is linked to the fuzzy conjunction (AND). $\bigvee$ represents the fuzzy disjunction (OR).
- $*$ β_{xy} is given by:

$$\beta_{ij} = \begin{cases} \bar{\beta}_{xy} & \text{when transcription factor y acts as an activator of gene x,} \\ 0 & \text{when no regulatory interaction exists from y to x,} \\ -\bar{\beta}_{xy}, & \text{when transcription factor y represses gene x.} \end{cases}$$

- $*$ $\sigma_1(\cdot)$ and $\sigma_2(\cdot)$ are the external imputs;
- $*$ $I(\cdot)$ and $J(\cdot)$ represent the control input;
- $*$ $y_1(\cdot)$ and $y_2(\cdot)$ represent the outputs of the system, while $\hbar_1$, $\hbar_2$, $\wp_1$, and $\wp_2$ denote constant matrices.

Throughout this work, it will be assume that:

Assumption 1. *There exist two nonnegative numbers* $\tilde{\sigma}_1$ *and* $\tilde{\sigma}_2$ *such that* $|\sigma_1(t)| \leq \tilde{\sigma}_1$ *and* $|\sigma_2(t)| \leq \tilde{\sigma}_2$.

Assumption 2. *Assume that for each* $y \in \aleph$, *one can find a constant* $k_y^+ > 0$ *with the property that* $\mathfrak{f}_y(\cdot)$ *fulfills*

$$0 \leq \frac{\mathfrak{f}_y(r_1) - \mathfrak{f}_y(r_2)}{r_1 - r_2} \leq k_y^+,\ y \in \aleph,$$

for all $r_1, r_2 \in \mathbb{R},\ r_1 \neq r_2$. *Denote,* $K^+ = diag\{k_1^+, k_2^+, \cdots, k_n^+\}$.

The initial conditions for model (1) are given by:

$$\begin{cases} \chi_x(\vartheta) &= \beth_x(\vartheta),\ x \in \aleph,\ \vartheta \in [-\nu,\ -\nu+1,\cdots,0], \\ \eta_x(\vartheta) &= \daleth_x(\vartheta),\ x \in \aleph,\ \vartheta \in [-\nu,\ -\nu+1,\cdots,0], \end{cases} \tag{3}$$

where $\beth_x = (\beth_1,\ \beth_2,\cdots,\beth_n)^T \in \mathbb{R}^n$, $\daleth_x = (\daleth_1,\ \daleth_2,\cdots,\daleth_n)^T \in \mathbb{R}^n$, $\nu = \max\{\alpha_1,\ \alpha_2\}$.

Remark 2.4. *In system (1), the fuzzy operators AND ($\bigwedge$) and OR ($\bigvee$) are specified as the minimum and maximum of the weighted functions, respectively:*

$$\begin{aligned} \bigwedge_{y=1}^{n} \varrho_{xy}\mathfrak{f}_y(\eta_y((t-\alpha_1)) &= \min\{\varrho_{xy}\mathfrak{f}_y(\eta_y((t-\alpha_1)))\}, \\ \bigvee_{y=1}^{n} \varphi_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) &= \max\{\varphi_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1))\}. \end{aligned}$$

It should be noted that this formulation does not involve a classical fuzzy inference system with membership functions, rule bases, or defuzzification. Instead, the MIN/MAX operators directly model the uncertain aggregation of multiple regulatory signals, which is a standard approach in fuzzy genetic regulatory network modeling [4, 19]*. This allows the system to capture graded regulatory interactions while remaining analytically tractable for Lyapunov-based analysis.*

Definition 2.5. [16] *System (1) is passive provided that there exists $\theta > 0$ satisfying*

$$2\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)y_1(\varsigma) + \sigma_2^T(\varsigma)y_2(\varsigma)] \geq -\theta\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma)], \tag{4}$$

for all $\iota \geq 0$.

Lemma 2.6. *For a positive function $W(\varsigma)$, if the condition*

$${}_0\nabla_\varsigma^\kappa W(\varsigma) \leq \sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma), \tag{5}$$

is satisfied, then model (1) is regarded as passive.

Proof. It follows from (5) that

$$-2\left[\sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma)\right] \leq -2\,{}_0\nabla_\varsigma^\kappa W(\varsigma). \tag{6}$$

Taking ${}_0\nabla_\varsigma^{-1}$ on both sides of (6), one has

$$\begin{aligned} -2\,{}_0\nabla_\varsigma^{-1}\Big[\sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma)\Big] &\leq -2\,{}_0\nabla_\varsigma^{-1}{}_0\nabla_\varsigma^\kappa W(\varsigma) \\ &= -2\,{}_0\nabla_\varsigma^{\kappa-1}{}_0\nabla_\varsigma^{-\kappa}\Big({}_0\nabla_\varsigma^\kappa W(\varsigma)\Big) \\ &= -2\,{}_0\nabla_\varsigma^{\kappa-1}\Big(W(\varsigma) - W(0)\Big) \\ &= -2\,{}_0\nabla_\varsigma^{\kappa-1}W(\varsigma) \\ &\leq 0. \end{aligned}$$

Since

$$\nabla_\varsigma^{-1}\Big[\sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma)\Big] = \sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)y_1(\varsigma) + \sigma_2^T(\varsigma)y_2(\varsigma)],$$

and

$$\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma)] \geq 0 \text{ for any } \varsigma \geq 0,$$

thus

$$2\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)y_1(\varsigma)+\sigma_2^T(\varsigma)y_2(\varsigma)]\geq 0\geq -\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)\sigma_1(\varsigma)+\sigma_2^T(\varsigma)\sigma_2(\varsigma)],$$

namely

$$2\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)y_1(\varsigma)+\sigma_2^T(\varsigma)y_2(\varsigma)]\geq -\sum_{\varsigma=0}^{\iota}[\sigma_1^T(\varsigma)\sigma_1(\varsigma)+\sigma_2^T(\varsigma)\sigma_2(\varsigma)].$$

Therefore, according to Definition 2.5, model (1) is passive when $\theta=1$. □

Definition 2.7. [4] *System (1) is globally dissipative if there is a compact set $\Omega\subset\mathbb{R}^{2n}$, and for every initial state $(\beth^T(s),\ \daleth^T(s))^T\in\mathbb{R}^{2n}\backslash\Omega,\ s\in[-\nu,\ -\nu+1,\cdots,0]$, there exists a time $\mathfrak{T}(\beth,\daleth)>t_0$ with the property that, for all $t\geq\mathfrak{T}(\beth,\daleth)$, the solution $(\chi^T(t_0,\ t,\ \beth),\ \eta^T(t_0,\ t,\ \daleth))^T$ of model (1) starting from $(\beth(s),\daleth(s))^T$ at time t_0 satisfies $(\chi^T(t_0,\ t,\ \beth),\ \eta^T(t_0,\ t,\ \daleth))^T\subseteq\Omega$. The set Ω is referred to as a globally attractive set.*

Definition 2.8. [10] *For $y:\mathbb{N}_{x+1}\to\mathbb{R}$, the nabla Laplace transform of $z(t)$ is defined by*

$$\mathcal{N}_x\{z(t)\}(s)=\sum_{y=1}^{\infty}(1-s)^{y-1}\ z(x+y).$$

In what follows, the nabla Laplace transform is applied to both the Caputo fractional difference and the classical integer-order difference operators to obtain the stated results.

Lemma 2.9. [10] *For $0<\kappa<1$ and $z:\mathbb{N}_x\to\mathbb{R}$, the nabla Laplace transform of ${}^C\nabla_x^\kappa z(t)$ is described by*

$$\mathcal{N}_x\left\{{}^C\nabla_x^\kappa z(t)\right\}(s)=s^\kappa\mathcal{N}_x\left\{z(t)\right\}(s)-s^{\kappa-1}\ z(x),$$

for $|1-s|<1$.

Lemma 2.10. [10] *Let $\hbar(\cdot)$ be a function and W a positive definite matrix. Then, one obtains*

$${}^C\nabla_x^\kappa\big[\hbar^T(s)W\hbar(s)\big]\leq\hbar^T(s)W\,{}^C\nabla_x^\kappa\hbar(s).$$

Lemma 2.11. [4] *For continuous functions $\vartheta_y:\mathbb{R}\to\mathbb{R}$ and real numbers $\zeta_y,\xi_y,\mu_{xy},\nu_{xy}$ with $x,\ y=1,\ldots,n$, the following bounds hold:*

$$\Big|\bigwedge_{y=1}^{n}\mu_{xy}\vartheta_y(\zeta_y)-\bigwedge_{y=1}^{n}\mu_{xy}\vartheta_y(\xi_y)\Big|\leq\sum_{y=1}^{n}|\mu_{xy}||\vartheta_y(\zeta_y)-\vartheta_y(\xi_y)|,$$
$$\Big|\bigvee_{y=1}^{n}\nu_{xy}\vartheta_y(\zeta_y)-\bigvee_{y=1}^{n}\nu_{xy}\vartheta_y(\xi_y)\Big|\leq\sum_{y=1}^{n}|\nu_{pq}||\vartheta_y(\zeta_y)-\vartheta_y(\xi_y)|.$$

Lemma 2.12. [4] *Let $\phi,\hat{\phi}\in\mathbb{R}^n$ and let $\Re$ be a positive definite matrix of appropriate dimension. Then, the following inequality holds:*

$$2\phi^T\hat{\phi}\leq\phi^T\Re^{-1}\phi+\hat{\phi}^T\Re\hat{\phi}.$$

Lemma 2.13. [10] *Assume that $\digamma(t)$ and $g(t)$ satisfy*

$$\begin{cases}{}^C\nabla_x^\kappa\digamma(t)=\varsigma\digamma(t),\\ {}^C\nabla_x^\kappa\chi(t)\leq\varsigma\chi(t).\end{cases}$$

Then, under the condition $\kappa\in(0,\ 1)$, $\varsigma<1$ and $\chi(x)=\digamma(x)>0$, it follows that $\chi(t)\leq\digamma(t)$ for all $t\in\mathbb{N}_{x+1}$.

3 Main results

The current section addresses the investigation of passivity and dissipativity in system (1).
The control input is constructed as

$$I(t) = -R_1\chi(t) - R_2\eta(t-\alpha_1),\ J(t) = -R_3\eta(t) - R_4\chi(t-\alpha_2). \tag{7}$$

We begin by analyzing the following theorem, which establishes the criterion for passivity.

Theorem 3.1. *Under Assumption 2, let P, Q, S_1, and S_2 be positive definite diagonal matrices satisfying the following LMIs:*

$$\Gamma = \begin{bmatrix} \Omega_{11} & \Omega_{12} & 0 & 0 & -\hbar_1^T & 0 \\ \star & -S_1 & 0 & 0 & 0 & 0 \\ \star & \star & \Omega_{33} & \Omega_{34} & 0 & -\hbar_2^T \\ \star & \star & \star & -S_2 & 0 & 0 \\ \star & \star & \star & \star & P-2\wp_1 & 0 \\ \star & \star & \star & \star & \star & Q-2\wp_2 \end{bmatrix} < 0, \tag{8}$$

where,

$$\begin{aligned} \Omega_{11} &= -2PB - 2PR_1 + P + (\alpha_2+1)S_2,\ \Omega_{12} = P\Big([\beta+\varrho+\varphi]K^+ - R_2\Big), \\ \Omega_{33} &= -2QA - 2R_3 + Q + (\alpha_1+1)S_1,\ \Omega_{34} = Q\Big([\psi+\bar{\varrho}+\bar{\varphi}] - R_4\Big). \end{aligned}$$

As a result, system (1) is passive when subjected to the control input (7).

Proof. The Lyapunov functional associated with the model is given as follows:

$$\begin{aligned} W(t) &= \sum_{x=1}^{n}\chi_x(t)p_x\chi_x(t) + \sum_{x=1}^{n}\eta_x(t)q_x\eta_x(t) \\ &+ \sum_{x=1}^{n}\Big[\sum_{s=t-\alpha_1}^{t}\eta_x(s)S_{1x}\eta_x(s) + \sum_{s=t-\alpha_2}^{t}\chi_x(s)S_{2x}\chi_x(s)\Big]. \end{aligned}$$

According to Lemma 2.10 and Assumption 2, it follows that

$$\begin{aligned} {}^{C}\nabla_0^{\kappa}W(t) &\le 2\sum_{x=1}^{n}\chi_x(t)p_x\,{}^{C}\nabla_0^{\kappa}\chi(t) + 2\sum_{x=1}^{n}\eta_x(t)q_x\,{}^{C}\nabla_0^{\kappa}\eta(t) \\ &+ (\alpha_1+1)\sum_{x=1}^{n}\eta_x(t)S_{1x}\eta_x(t) - \sum_{x=1}^{n}\eta_x(t-\alpha_1)S_{1x}\eta_x(t-\alpha_1) \\ &+ (\alpha_2+1)\sum_{x=1}^{n}\chi_x(t)S_{2x}\chi_x(t) - \sum_{x=1}^{n}\chi_x(t-\alpha_2)S_{2x}\chi_x(t-\alpha_2) \\ &= 2\sum_{x=1}^{n}\chi_x(t)p_x\Big\{-b_x\chi_x(t) + \sum_{y=1}^{n}\beta_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \bigwedge_{y=1}^{n}\varrho_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) \\ &+ \bigvee_{y=1}^{n}\varphi_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \sigma_{1x}(t) + I_x(t)\Big\} + 2\sum_{x=1}^{n}\eta_x(t)q_x\Big\{-a_x\eta_x(t) \\ &+ \psi_x\chi_x(t-\alpha_2) + \bigwedge_{y=1}^{n}\bar{\varrho}_{xy}\chi_y(t-\alpha_2) + \bigvee_{y=1}^{n}\bar{\varphi}_{xy}\chi_y(t-\alpha_2) + \sigma_{2x}(t) + J_x(t)\Big\} \\ &+ (\alpha_1+1)\sum_{x=1}^{n}\eta_x(t)S_{1x}\eta_x(t) - \sum_{x=1}^{n}\eta_x(t-\alpha_1)S_{1x}\eta_x(t-\alpha_1) \\ &+ (\alpha_2+1)\sum_{x=1}^{n}\chi_x(t)S_{2x}\chi_x(t) - \sum_{x=1}^{n}\chi_i(t-\alpha_2)S_{2x}\chi_x(t-\alpha_2) \end{aligned}$$

$$
\begin{aligned}
&\leq && -2\chi^T(t)PB\chi(t) + 2\chi^T(t)P[\beta+\varrho+\varphi]\mathfrak{f}(\eta(t-\alpha_1)) + 2\chi^T(t)P\sigma_1(t) + 2\chi^T(t)PI(t)\\
&- && 2\eta^T(t)QA\eta(t) + 2\eta^T(t)Q[\psi+\bar{\varrho}+\bar{\varphi}]\chi(t-\alpha_2) + 2\eta^T(t)Q\sigma_2(t) + 2\eta^T(t)QJ(t)\\
&+ && (\alpha_1+1)\eta^T(t)S_1\eta(t) - \eta^T(t-\alpha_1)S_1\eta(t-\alpha_1)\\
&+ && (\alpha_2+1)\chi^T(t)S_2\chi(t) - \chi^T(t-\alpha_2)S_2\chi(t-\alpha_2)\\
&\leq && -2\chi^T(t)[-2PB-2PR_1+(\alpha_2+1)S_2]\chi(t) + 2\chi^T(t)P\Big([\beta+\varrho+\varphi]K^+ - R_2\Big)\eta(t-\alpha_1)\\
&+ && 2\chi^T(t)P\sigma_1(t) + \eta^T(t)[-2QA-2R_3+(\alpha_1+1)S_1]\eta(t) + 2\eta^T(t)Q\Big([\psi+\bar{\varrho}+\bar{\varphi}] - R_4\Big)\chi(t-\alpha_2)\\
&+ && 2\eta^T(t)Q\sigma_2(t) - \eta^T(t-\alpha_1)S_1\eta(t-\alpha_1) - \chi^T(t-\alpha_2)S_2\chi(t-\alpha_2).
\end{aligned}
$$

Invoking Lemma 2.12, it can be shown that

$$
\begin{gathered}
2\chi^T(t)P\sigma_1(t) \leq \chi^T(t)P\chi(t) + \sigma_1^T(t)P\sigma_1(t),\\
2\eta^T(t)Q\sigma_2(t) \leq \eta^T(t)Q\eta(t) + \sigma_2^T(t)Q\sigma_2(t).
\end{gathered}
$$

from which it follows that

$$
\begin{aligned}
{}^C\nabla_0^\kappa W(t) &\leq && \chi^T(t)[-2PB-2PR_1+P+(\alpha_2+1)S_2]\chi(t) + 2\chi^T(t)P\Big([\beta+\varrho+\varphi]K^+ - R_2\Big)\eta(t-\alpha_1)\\
&+ && \eta^T(t)[-2QA-2R_3+Q+(\alpha_1+1)S_1]\eta(t) + \sigma_1^T(t)P\sigma_1(t)\\
&+ && 2\eta^T(t)Q\Big([\psi+\bar{\varrho}+\bar{\varphi}] - R_4\Big)\chi(t-\alpha_2)\\
&+ && \sigma_2^T(t)Q\sigma_2(t) - \eta^T(t-\alpha_1)S_1\eta(t-\alpha_1) - \chi^T(t-\alpha_2)S_2\chi(t-\alpha_2).
\end{aligned}
$$

So

$$
\begin{aligned}
{}^C\nabla_0^\kappa W(t) &- && 2u_1^T(t)y_1(t) - 2u_2^T(t)y_2(t) \leq \chi^T(t)[-2PB-2PR_1+P+(\alpha_2+1)S_2]\chi(t)\\
&+ && 2\chi^T(t)P\Big([\beta+\varrho+\varphi]K^+ - R_2\Big)\eta(t-\alpha_1) + \eta^T(t)[-2QA-2R_3+Q+(\alpha_1+1)S_1]\eta(t)\\
&+ && 2\eta^T(t)Q\Big([\psi+\bar{\varrho}+\bar{\varphi}] - R_4\Big)\chi(t-\alpha_2) + \sigma_1^T(t)P\sigma_1(t) + \sigma_2^T(t)Q\sigma_2(t)\\
&- && \eta^T(t-\alpha_1)S_1\eta(t-\alpha_1) - \chi^T(t-\alpha_2)S_2\chi(t-\alpha_2) - 2\sigma_1^T(t)\hbar_1\chi(t) - 2\sigma_1^T(t)\wp_1\sigma_1(t)\\
&- && 2\sigma_2^T(t)\hbar_2\eta(t) - 2\sigma_2^T(t)\wp_2\sigma_2(t)\\
&\leq && \Xi^T(t)\Gamma\Xi(t),
\end{aligned}
$$

where $\Xi(t) = \begin{bmatrix} \chi(t) & \eta(t-\alpha_1) & \eta(t) & \chi(t-\alpha_2) & \sigma_1(t) & \sigma_2(t) \end{bmatrix}^T$. From the condition $\Gamma < 0$, we obtain

$$
{}^C\nabla_0^\kappa W(t) \leq 2\sigma_1^T(t)y_1(t) + 2\sigma_2^T(t)y_2(t).
$$

Consequently, according to Lemma 2.6, system (1) satisfies the passivity property. □

Next, we examine the following theorem, which provides the criterion for dissipativity.

Theorem 3.2. *Under Assumption 2, let $P,\ Q,\ M_1$ and M_2 be positive definite diagonal matrices satisfying the following LMIs:*

$$
\Xi = \begin{bmatrix} \Xi_{11} & \Xi_{12} & 0 & 0\\ \star & -M_1 & 0 & 0\\ \star & \star & \Xi_{33} & \Xi_{34}\\ \star & \star & \star & -M_2 \end{bmatrix} < 0, \tag{9}
$$

where,

$$
\begin{aligned}
\Xi_{11} &= -2PB-2PR_1+(1+\xi)P+(\alpha_2+1)M_2,\ \Xi_{12} = P\Big([\beta+\varrho+\varphi]K^+ - R_2\Big),\\
\Xi_{33} &= -2QA-2R_3+(1+\xi)Q+(\alpha_1+1)M_1,\ \Xi_{34} = Q\Big([\psi+\bar{\varrho}+\bar{\varphi}] - R_4\Big).
\end{aligned}
$$

As a result, system (1) exhibits global dissipativity, with

$$\Omega \;=\; \left\{(\chi^T,\ \eta^T)^T \in \mathbb{R}^{2n} : \|\chi\|_1 + \|\eta\|_1 \leq \frac{\zeta}{\xi}\right\},$$

acting both as a positively invariant and globally attracting region, where $\xi > 0$ and $\zeta \geq 0$.

Proof. The Lyapunov functional associated with the model is given as follows:

$$\begin{aligned} W(t) \;&=\; \sum_{x=1}^{n} \chi_x(t) p_x \chi_x(t) + \sum_{x=1}^{n} \eta_x(t) q_x \eta_x(t) \\ &+\; \sum_{x=1}^{n} \Bigg[\sum_{s=t-\alpha_1}^{t} \eta_x(s) M_{1x} \eta_x(s) + \sum_{s=t-\alpha_2}^{t} \chi_x(s) M_{2x} \chi_x(s) \Bigg]. \end{aligned}$$

According to Lemma 2.10 and Assumption 2, it follows that

$$\begin{aligned} {}^{C}\nabla_0^{\kappa} W(t) \;&\leq\; 2\sum_{x=1}^{n} \chi_x(t) p_x \, {}^{C}\nabla_0^{\kappa} \chi(t) + 2\sum_{x=1}^{n} \eta_x(t) q_x \, {}^{C}\nabla_0^{\kappa} \eta(t) \\ &+\; (\alpha_1+1) \sum_{x=1}^{n} \eta_x(t) M_{1x} \eta_x(t) - \sum_{x=1}^{n} \eta_x(t-\alpha_1) M_{1x} \eta_x(t-\alpha_1) \\ &+\; (\alpha_2+1) \sum_{x=1}^{n} \chi_x(t) M_{2x} \chi_x(t) - \sum_{x=1}^{n} \chi_x(t-\alpha_2) M_{2x} \chi_x(t-\alpha_2) \\ &=\; 2\sum_{x=1}^{n} \chi_x(t) p_x \Bigg\{ - b_x \chi_x(t) + \sum_{y=1}^{n} \beta_{xy} \mathfrak{f}_y(\eta_y(t-\alpha_1)) + \bigwedge_{y=1}^{n} \varrho_{xy} \mathfrak{f}_y(\eta_y(t-\alpha_1)) \\ &+\; \bigvee_{y=1}^{n} \varphi_{xy} \mathfrak{f}_y(\eta_y(t-\alpha_1)) + \sigma_{1x}(t) + I_x(t) \Bigg\} + 2\sum_{x=1}^{n} \eta_x(t) q_x \Bigg\{ - a_x \eta_x(t) \\ &+\; \psi_x \chi_x(t-\alpha_2) + \bigwedge_{y=1}^{n} \bar{\varrho}_{xy} \chi_y(t-\alpha_2) + \bigvee_{y=1}^{n} \bar{\varphi}_{xy} \chi_y(t-\alpha_2) \sigma_{2x}(t) + J_x(t) \Bigg\} \\ &+\; (\alpha_1+1) \sum_{x=1}^{n} \eta_x(t) M_{1x} \eta_x(t) - \sum_{x=1}^{n} \eta_x(t-\alpha_1) M_{1x} \eta_x(t-\alpha_1) \\ &+\; (\alpha_2+1) \sum_{x=1}^{n} \chi_x(t) M_{2x} \chi_x(t) - \sum_{x=1}^{n} \chi_x(t-\alpha_2) M_{2x} \chi_x(t-\alpha_2) \\ &\leq\; \chi^T(t)[-2PB - 2PR_1 + (\alpha_2+1)M_2]\chi(t) + 2\chi^T(t) P \Big([\beta + \varrho + \varphi] K^+ - R_2 \Big) \eta(t-\alpha_1) \\ &+\; 2\chi^T(t) P \sigma_1(t) + \eta^T(t)[-2QA - 2R_3 + (\alpha_1+1)M_1]\eta(t) + 2\eta^T(t) Q \Big([\psi + \bar{\varrho} + \bar{\varphi}] - R_4 \Big) \chi(t-\alpha_2) \\ &+\; 2\eta^T(t) Q \sigma_2(t) - \eta^T(t-\alpha_1) M_1 \eta(t-\alpha_1) - \chi^T(t-\alpha_2) M_2 \chi(t-\alpha_2). \end{aligned}$$

By employing the Lemma 2.12, we get

$$\begin{gathered} 2\chi^T(t) P \sigma_1(t) \leq \chi^T(t) P \chi(t) + \sigma_1^T(t) P \sigma_1(t), \\ 2\eta^T(t) Q \sigma_2(t) \leq \eta^T(t) Q \eta(t) + \sigma_2^T(t) Q \sigma_2(t), \end{gathered}$$

which imply,

$$\begin{aligned}
{}^{C}\nabla_{0}^{\kappa}W(t) &\leq \chi^{T}(t)[-2PB-2PR_{1}+P+(\alpha_{2}+1)M_{2}]\chi(t)+2\chi^{T}(t)P\bigg([\beta+\varrho+\varphi]K^{+}-R_{2}\bigg)\eta(t-\alpha_{1})\\
&+ \eta^{T}(t)[-2QA-2R_{3}+Q+(\alpha_{1}+1)M_{1}]\eta(t)+\sigma_{1}^{T}(t)P\sigma_{1}(t)\\
&+ 2\eta^{T}(t)Q\bigg([\psi+\bar{\varrho}+\bar{\varphi}]-R_{4}\bigg)\chi(t-\alpha_{2})\\
&- \eta^{T}(t-\alpha_{1})M_{1}\eta(t-\alpha_{1})-\chi^{T}(t-\alpha_{2})M_{2}\chi(t-\alpha_{2})+\sigma_{2}^{T}(t)Q\sigma_{2}(t)\\
&\leq \chi^{T}(t)[-2PB-2PR_{1}+(1+\xi)P+(\alpha_{2}+1)M_{2}]\chi(t)+2\chi^{T}(t)P\bigg([\beta+\varrho+\varphi]K^{+}-R_{2}\bigg)\eta(t-\alpha_{1})\\
&+ \eta^{T}(t)[-2QA-2R_{3}+(1+\xi)Q+(\alpha_{1}+1)M_{1}]\eta(t)+2\eta^{T}(t)Q\bigg([\psi+\bar{\varrho}+\bar{\varphi}]-R_{4}\bigg)\chi(t-\alpha_{2})\\
&- \eta^{T}(t-\alpha_{1})M_{1}\eta(t-\alpha_{1})-\chi^{T}(t-\alpha_{2})M_{2}\chi(t-\alpha_{2})+\sigma_{1}^{T}(t)P\sigma_{1}(t)+\sigma_{2}^{T}(t)Q\sigma_{2}(t)\\
&- \xi\chi^{T}(t)P\chi(t)-\xi\eta^{T}(t)Q\eta(t)\\
&\leq \Sigma^{T}(t)\Xi\Sigma(t)-\xi\chi^{T}(t)P\chi(t)-\xi\eta^{T}(t)Q\eta(t)+\tilde{\sigma}_{1}^{T}P\tilde{\sigma}_{1}+\tilde{\sigma}_{2}^{T}Q\tilde{\sigma}_{2},
\end{aligned}$$

where $\Sigma(t)=\begin{bmatrix}\chi(t) & \eta(t-\alpha_{1}) & \eta(t) & \chi(t-\alpha_{2})\end{bmatrix}^{T}$. From the condition $\Xi<0$, we get

$$\begin{aligned}
{}^{C}\nabla_{0}^{\kappa}W(t) &\leq -\xi\chi^{T}(t)P\chi(t)-\xi\eta^{T}(t)Q\eta(t)+\tilde{\sigma}_{1}^{T}P\tilde{\sigma}_{1}+\tilde{\sigma}_{2}^{T}Q\tilde{\sigma}_{2}\\
&\leq -\xi W(t)+\tilde{\sigma}_{1}^{T}P\tilde{\sigma}_{1}+\tilde{\sigma}_{2}^{T}Q\tilde{\sigma}_{2}.
\end{aligned}$$

Let us analyze the system below

$$ {}^{C}\nabla_{0}^{\kappa}\digamma(t) = -\xi\digamma(t)+\zeta, \tag{10}$$

where $\zeta=\tilde{\sigma}_{1}^{T}P\tilde{\sigma}_{1}+\tilde{\sigma}_{2}^{T}Q\tilde{\sigma}_{2}$ and $\digamma(t)$ has same initial condition with $W(t)$ and assume $\digamma(t)\geq 0$. Then, by using Lemma 2.13, one has $0\leq W(t)\leq\digamma(t)$. Based on Caputo fractional-order derivative properties, equation (10) can be written as

$$ {}^{C}\nabla_{0}^{\kappa}\digamma^{*}(t) = -\xi\digamma^{*}(t), \tag{11}$$

where $(\digamma^{*}(t)=\digamma(t)-\zeta^{*})$ and $\zeta^{*}=\frac{\zeta}{\xi}$.
Applying the discrete Laplace transform to equation (11) yields:

$$\mathcal{L}\{{}^{C}\nabla_{0}^{\kappa}\digamma^{*}(t)\}(s)=-\mathcal{L}\{\xi\digamma^{*}(t)\}(s),$$

more exactly

$$\begin{gathered}
s^{\kappa}\digamma^{*}(s)-s^{\kappa-1}\digamma^{*}(0)=-\xi\digamma^{*}(s),\\
\Delta(s)\digamma^{*}(s)=\ell(s),
\end{gathered}$$

where $\Delta(s)=s^{\kappa}+\xi$, $\ell(s)=s^{\kappa-1}\digamma^{*}(0)$.
We now aim to prove that the characteristic equation $\det(\Delta(s))=0$ admits no purely imaginary roots. Suppose, for the sake of contradiction, that $\det(\Delta(s))=0$ does indeed possess purely imaginary roots; in other words,

$$s=i\varpi=|\varpi|\left(\cos\left(\frac{\eta}{2}\right)+i\sin\left(\pm\frac{\eta}{2}\right)\right),\quad \text{for } \varpi\in\mathbb{R}.$$

If $\varpi>0$,

$$s=i\varpi=|\varpi|\left(\cos\left(\frac{\eta}{2}\right)+i\sin\left(\frac{\eta}{2}\right)\right),$$

or if $\varpi<0$,

$$s=i\varpi=|\varpi|\left(\cos\left(\frac{\eta}{2}\right)-i\sin\left(\frac{\eta}{2}\right)\right),$$

then substituting $s=i\varpi=|\varpi|\left(\cos\left(\frac{\eta}{2}\right)+i\sin\left(\pm\frac{\eta}{2}\right)\right)$ into $\det(\Delta(s))=0$, it follows

$$|\varpi|^{\kappa}\left(\cos\left(\frac{\kappa\eta}{2}\right)+i\sin\left(\pm\frac{\kappa\eta}{2}\right)\right)+\xi=0. \tag{12}$$

Decomposing into real and imaginary components, we get

$$|\varpi|^{\kappa}\cos\left(\tfrac{\kappa\eta}{2}\right)+\xi=0,$$
$$|\varpi|^{\kappa}\sin\left(\pm\tfrac{\kappa\eta}{2}\right)=0.$$

Thus

$$|\varpi|^{2\kappa}+2\xi|\varpi|^{\kappa}\cos\left(\frac{\kappa\eta}{2}\right)+\xi^{2}=0. \tag{13}$$

Based on the previous equation, one finds that

$$\Delta=\left[2\xi\cos\left(\frac{\kappa\eta}{2}\right)\right]^{2}-4\xi^{2}=4\xi^{2}\left[\cos^{2}\left(\frac{\kappa\eta}{2}\right)-1\right]<0. \tag{14}$$

Equation (13) evidently does not admit any purely real solutions, thereby invalidating the hypothesis.
Consequently, the matrix $\theta=-\xi$ possesses eigenvalues negative, that is to say that $|\arg(\lambda(\theta))|>\frac{\eta}{2}$. As a result, the trivial solution of (11) is globally asymptotically stable in the Lyapunov sense. Hence, $\digamma(t)-\zeta^{*}\to 0$ for $t\to\infty$. That is, for every $\varepsilon>0$, there exists a time $\mathfrak{T}>0$ with the property that $\digamma(t)<\zeta^{*}+\varepsilon$, with $\digamma(t)>0$.
By invoking Lemma 2.13, we get $0\le W(t)\le\digamma(t)$. Therefore, we observe that $W(t)<\zeta^{*}+\varepsilon$ for $t>\mathfrak{T}$, which implies $W(t)\le\zeta^{*}=\zeta/\varsigma$ for $t>\mathfrak{T}$, that is, $\|\chi(t)\|_{1}+\|\eta(t)\|_{1}\le\zeta^{*}$ for $t>\mathfrak{T}$. Accordingly, there exists a time $\mathfrak{T}>0$ with the property that the trajectory of $(\chi^{T}(t_{0},\ t,\ \beth),\ \eta^{T}(t_{0},\ t,\ \daleth))^{T}$ remains within the set Ω for any initial condition $(\beth(s),\ \daleth(s))^{T}\in\mathbb{R}^{2n}$. In conclusion, the system defined in (1) is demonstrated to be dissipative. □

Remark 3.3. *The present study establishes a conceptual framework for enhancing the architecture of fractional-order fuzzy genetic regulatory networks, particularly through the incorporation of template input/output structures and sum-of-product operations. As a result, the findings not only enrich the analysis of dynamical properties in discrete-time fractional-order fuzzy GRNs but also extend and unify prior research in this area.*

Remark 3.4. *Unlike continuous-time formulations* [4, 22] *or integer-order discrete models* [27]*, the proposed discrete-time fractional-order GRN framework simultaneously captures the sampled nature of biological data and the long-term memory effects inherent in genetic regulation, making it more consistent with realistic scenarios. Furthermore, the derived passivity and dissipativity criteria are established in this fractional discrete setting, which allows our results to cover a wider class of genetic regulatory networks than previous approaches, many of which are restricted to either continuous-time or integer-order formulations.*

Remark 3.5. *Classical nonlinear control strategies, such as sliding mode control* [3]*, adaptive control* [17]*, intermittent control* [2]*, event-triggered control* [9]*, and quantized control [6], have been extensively developed to ensure asymptotic or practical stability of nonlinear systems, and have demonstrated effectiveness in various engineering contexts. However, these approaches primarily focus on trajectory stabilization and generally do not provide explicit energy-based performance characterizations of the closed-loop dynamics. In this regard, the proposed framework complements existing methods by guaranteeing passivity and global dissipativity, which offer a quantitative description of the energy exchange between the system and its environment, thereby ensuring bounded internal energy evolution and enhanced robustness against external disturbances properties that are particularly meaningful in biological regulatory networks subject to environmental fluctuations. Moreover, unlike Takagi–Sugeno fuzzy control schemes* [5]*, which depend on heuristic rule bases, membership function construction, and defuzzification procedures, the proposed method employs fuzzy MIN/MAX operators solely to model regulatory uncertainties, while the controller itself is constructed as a linear state-feedback law with delayed terms and synthesized systematically via linear matrix inequalities. This design avoids the structural complexity of full fuzzy inference systems while still capturing nonlinear regulatory behavior, and provides rigorous, computationally tractable passivity and dissipativity certificates within a fractional-order discrete-time framework, making the proposed approach a theoretically sound and practically implementable complementary tool for the analysis and control of uncertain, memory-dependent biological networks.*

Remark 3.6. *Recent studies have extensively investigated the dynamic behavior of genetic regulatory networks (GRNs) under different modeling paradigms. For example, global dissipativity of continuous-time fuzzy GRNs with mixed delays was analyzed in* [4] *using Lyapunov functional and LMI techniques, while passivity conditions for discrete-time GRNs with reaction-diffusion coupling and delays were established in* [30]*. In addition, robust passivity analysis for stochastic GRNs with time-varying delays was addressed in* [14]*, and improved stability criteria for discrete-time GRNs subject to stochastic perturbations were developed in* [26]*. Although these contributions provide valuable insights, they are* *typically formulated either in continuous-time settings, integer-order discrete-time frameworks, or stochastic environments without simultaneously accounting for memory effects and fuzzy regulatory uncertainty. In contrast, the present paper introduces a discrete-time fractional-order fuzzy genetic regulatory network framework that integrates multiple realistic biological features within a unified structure, namely: (i) memory and hereditary properties modeled through fractional-order Caputo differences, (ii) regulatory uncertainty captured via fuzzy MIN/MAX operators, (iii) intrinsic transcription–translation delays, and (iv) robustness characterized from an energy-based perspective through newly derived passivity and global dissipativity criteria. By jointly incorporating these aspects, the proposed framework extends existing results and provides a more comprehensive and flexible analytical tool for modeling, analysis, and robust control of complex genetic regulatory mechanisms.*

4 Illustrative examples

To illustrate the practical relevance of the theoretical findings, two examples are included.

Example 4.1. *Consider the following FODTFGRNs with* $n = 4$

$$\left\{\begin{array}{rcl} {}^{C}\nabla_0^{0.93}\chi_x(t) & = & -b_x\chi_x(t) + \sum\limits_{y=1}^{4}\beta_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \bigwedge\limits_{y=1}^{2}\varrho_{xy}\mathfrak{f}_y(\eta_y((t-\alpha_1)) \\ & + & \bigvee\limits_{y=1}^{4}\varphi_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \sigma_{1x}(t) + I_x(t), \\ {}^{C}\nabla_0^{0.93}\eta_x(t) & = & -a_x\eta_x(t) + \psi_x\chi_x(t-\alpha_2) + \bigwedge\limits_{y=1}^{4}\bar{\varrho}_{xy}\chi_y(t-\alpha_2) \\ & + & \bigvee\limits_{y=1}^{4}\bar{\varphi}_{xy}\chi_y(t-\alpha_2) + \sigma_{2x}(t) + J_x(t), \end{array}\right. \tag{15}$$

where

$$B = \begin{bmatrix} 0.9 & 0 & 0 & 0 \\ 0 & 0.7 & 0 & 0 \\ 0 & 0 & 0.6 & 0 \\ 0 & 0 & 0 & 0.68 \end{bmatrix},\ \beta = \begin{bmatrix} 1.3 & -2.3 & 0.9 & -0.8 \\ 1.29 & -1.5 & 1.3 & 0.7 \\ 1.19 & -1.2 & 0.98 & -0.5 \\ -1.2 & 0.9 & -0.7 & 1.1 \end{bmatrix},\ \varrho = \begin{bmatrix} -0.57 & 0.32 & 0.4 & 0.11 \\ 0.3 & 0.13 & -0.11 & 0.23 \\ -0.1 & 0.21 & 0.13 & -0.4 \\ 0.11 & -0.15 & 0.2 & 0.6 \end{bmatrix},$$

$$\varphi = \begin{bmatrix} 0.65 & -0.43 & 0.1 & 0.2 \\ 0.1 & 0.6 & 0.4 & 0.4 \\ -0.1 & 0.1 & 0.2 & -0.2 \\ 0.5 & -0.5 & 0.1 & 0.3 \end{bmatrix},\ A = \begin{bmatrix} 0.61 & 0 & 0 & 0 \\ 0 & 0.41 & 0 & 0 \\ 0 & 0 & 0.6 & 0 \\ 0 & 0 & 0 & 0.7 \end{bmatrix},\ \psi = \begin{bmatrix} 0.6 & 0 & 0 & 0 \\ 0 & 1.3 & 0 & 0 \\ 0 & 0 & 0.9 & 0 \\ 0 & 0 & 0 & 0.7 \end{bmatrix},$$

$$\bar{\varrho} = \begin{bmatrix} -0.19 & 0.06 & -0.09 & -0.16 \\ 0.03 & -0.05 & 0.05 & -0.04 \\ -0.07 & 0.013 & 0.08 & -0.06 \\ -0.012 & 0.01 & 0.1 & -0.1 \end{bmatrix},\ \bar{\varphi} = \begin{bmatrix} 0.16 & -0.09 & 0.19 & -0.02 \\ 0.05 & 0.02 & -0.1 & 0.04 \\ 0.01 & 0.01 & -0.02 & 0.031 \\ 0.06 & 0.08 & -0.01 & -0.02 \end{bmatrix},\ H_1 = \begin{bmatrix} 0.86 & 0 & 0 & 0 \\ 0 & 0.42 & 0 & 0 \\ 0 & 0 & 0.4 & 0 \\ 0 & 0 & 0 & 0.61 \end{bmatrix},$$

$$H_2 = \begin{bmatrix} 1.1 & 0 & 0 & 0 \\ 0 & 0.75 & 0 & 0 \\ 0 & 0 & 0.4 & 0 \\ 0 & 0 & 0 & 0.7 \end{bmatrix},\ \wp_1 = \begin{bmatrix} 0.87 & 0 & 0 & 0 \\ 0 & 0.79 & 0 & 0 \\ 0 & 0 & 0.5 & 0 \\ 0 & 0 & 0 & 0.6 \end{bmatrix},\ \wp_2 = \begin{bmatrix} 1.1 & 0 & 0 & 0 \\ 0 & 1.21 & 0 & 0 \\ 0 & 0 & 0.9 & 0 \\ 0 & 0 & 0 & 0.95 \end{bmatrix},$$

$\mathfrak{f}_y(\varsigma_y(t)) = \dfrac{\varsigma_y^2(t)}{1+\varsigma_y^2(t)}$, $\alpha_1 = 2$, $\alpha_2 = 1$, $\sigma_{1x}(t) = \sin(t)$, $\sigma_{2x}(t) = \cos(t)$, *The function* $\mathfrak{f}_y$ *is constructed as* (2) *with* $H_y = 2$ *and* $\tau_y = 1$, *commonly used in genetic regulatory network modeling, and satisfies Assumption 2 with* $K^+ = \mathrm{diag}\{0.65,\ 0.65, 0.65,\ 0.65\}$. *Moreover, since* $|\sin(t)| \le 1$ *and* $|\cos(t)| \le 1$, *Assumption 1 holds with* $\tilde{\sigma}_1 = \tilde{\sigma}_2 = 1$.

For the control inputs (7), let $R_1 = \begin{bmatrix} 1.8 & 0 & 0 & 0 \\ 0 & 1.6 & 0 & 0 \\ 0 & 0 & 1.5 & 0 \\ 0 & 0 & 0 & 1.7 \end{bmatrix}$, $R_2 = \begin{bmatrix} 1.41 & 0 & 0 & 0 \\ 0 & 1.28 & 0 & 0 \\ 0 & 0 & 1.1 & 0 \\ 0 & 0 & 0 & 1.7 \end{bmatrix}$, $R_3 = \begin{bmatrix} 1.5 & 0 & 0 & 0 \\ 0 & 1.42 & 0 & 0 \\ 0 & 0 & 1.6 & 0 \\ 0 & 0 & 0 & 1.4 \end{bmatrix}$

and $R_4 = \begin{bmatrix} 1.52 & 0 & 0 & 0 \\ 0 & 1.34 & 0 & 0 \\ 0 & 0 & 1.4 & 0 \\ 0 & 0 & 0 & 1.7 \end{bmatrix}$. *Solving the LMI (8) via YALMIP yields the valid matrices:*

$$P = \begin{bmatrix} 0.3485 & 0 & 0 & 0 \\ 0 & 0.1377 & 0 & 0 \\ 0 & 0 & 0.5143 & 0 \\ 0 & 0 & 0 & 0.7523 \end{bmatrix}, \quad Q = \begin{bmatrix} 0.1677 & 0 & 0 & 0 \\ 0 & 0.4102 & 0 & 0 \\ 0 & 0 & 0.5120 & 0 \\ 0 & 0 & 0 & 0.2864 \end{bmatrix},$$

$$S_1 = \begin{bmatrix} 0.5932 & -0.0257 & -0.0215 & 0.0184 \\ -0.0257 & 0.7338 & -0.0881 & 0.0271 \\ -0.0215 & -0.0881 & 0.7280 & 0.0432 \\ 0.0184 & 0.0271 & 0.0432 & 0.6141 \end{bmatrix} \text{ and } S_2 = \begin{bmatrix} 0.1545 & -0.0518 & -0.0395 & 0.0064 \\ -0.0518 & 0.0762 & -0.0299 & 0.0626 \\ -0.0395 & -0.0299 & 0.2674 & -0.0003 \\ 0.0064 & 0.0626 & -0.0003 & 0.5732 \end{bmatrix}.$$

With all conditions of Theorem 3.1 satisfied, system (15) is confirmed to be passive under the control input (7). Figures 1 and 2 display the trajectories of system (15) under control protocol (7) with starting conditions $\chi(\vartheta) = (-2.4, -1.5, 1.4, 1.85)^T$ *and* $\eta(\vartheta) = (1.6, 2.75, 0.4, 1.75)^T$, *for* $\vartheta \in [-2, -1, 0]$. *Figures 3 and 4 refer to the trajectories of outputs* $y_{1x}(t)$ *and* $y_{2x}(t)$, *respectively. Figures 5 and 6 refer to the trajectories of control input* $I_x(t)$ *and* $J_x(t)$.

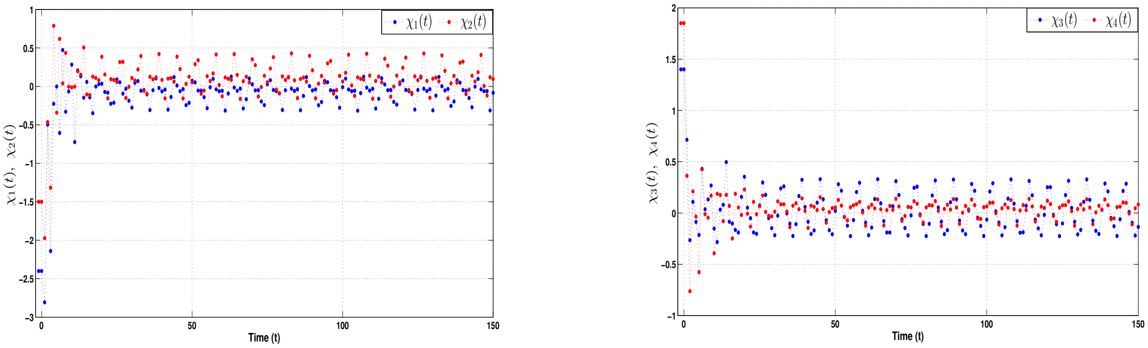

Figure 1: Time evolution of $\chi(t)$ in system (15).

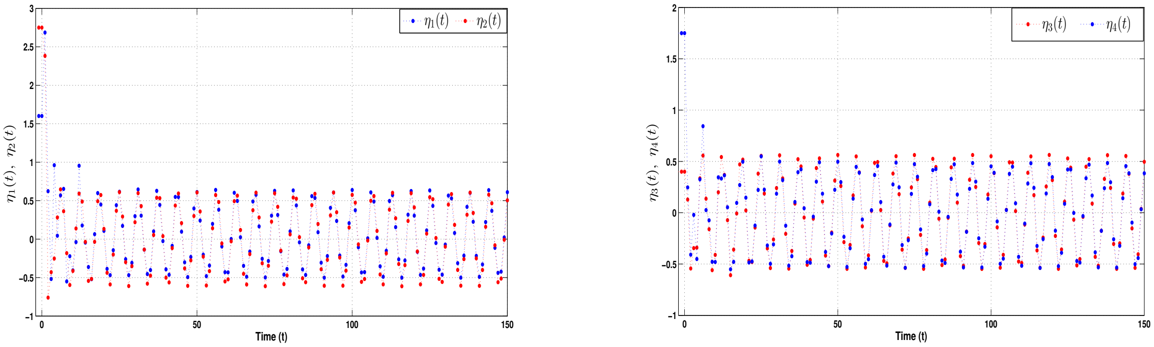

Figure 2: Time evolution of $\eta(t)$ in system (15).

Figure 7 refers to the trajectories of $2(\sigma_1^T(t)y_1(t) + \sigma_2^T(t)y_2(t))$. *It can be observed that this quantity remains strictly positive for all* $t \in \mathbb{N}_0$. *On the other hand, we have* $\sigma_1^T(t)\sigma_1(t) + \sigma_2^T(t)\sigma_2(t) = 4(\sin^2(t) + \cos^2(t)) = 4$, *which is always positive. Consequently, for* $\theta = 1$, *the term* $-\theta(\sigma_1^T(t)\sigma_1(t) + \sigma_2^T(t)\sigma_2(t))$ *is always negative. Therefore, the inequality*

$$2\sum_{\varsigma=0}^{t}(\sigma_1^T(\varsigma)y_1(\varsigma) + \sigma_2^T(\varsigma)y_2(\varsigma)) \geq -\sum_{\varsigma=0}^{t}(\sigma_1^T(\varsigma)\sigma_1(\varsigma) + \sigma_2^T(\varsigma)\sigma_2(\varsigma)),$$

holds for every $t \in \mathbb{N}_0$, *which means that system* (15) *is passive. This simulation result is consistent with Theorem 3.1.*

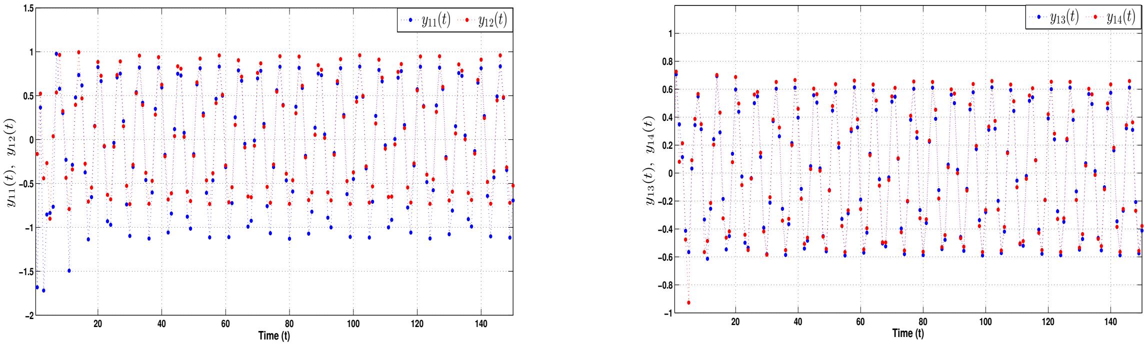

Figure 3: Output trajectories $y_{1x}(t)$ for system (15).

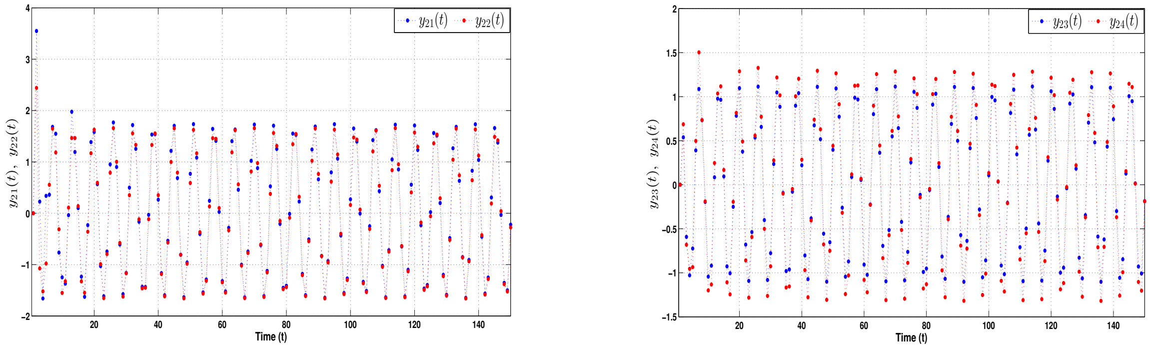

Figure 4: Output trajectories $y_{2x}(t)$ for system (15).

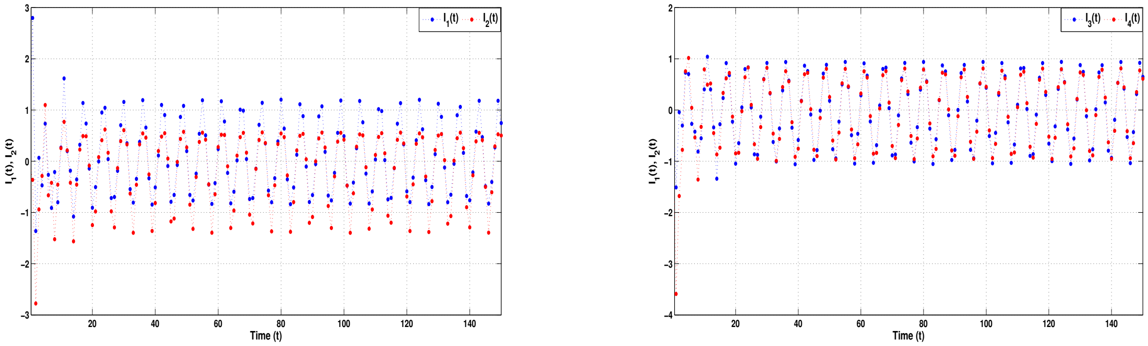

Figure 5: Time evolution of the control inputs $I(t)$.

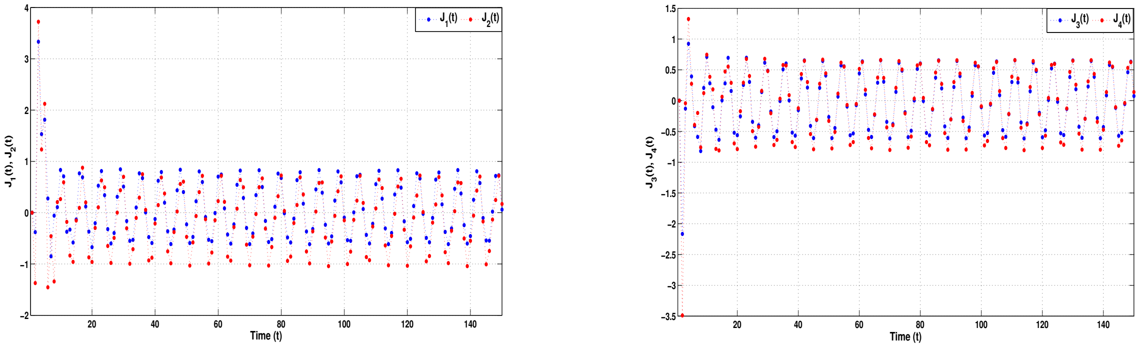

Figure 6: Time evolution of the control inputs $J(t)$.

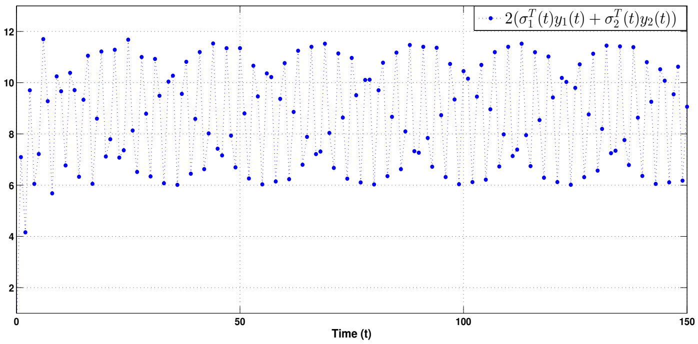

Figure 7: Trajectories of $2(\sigma_1^T(t)y_1(t) + \sigma_2^T(t)y_2(t))$.

Example 4.2. *Consider a FODTFGRNs composed of* $n = 2$ *nodes.*

$$\begin{cases} {}^{C}\nabla_0^{0.95}\chi_x(t) = -b_x\chi_x(t) + \sum_{y=1}^{2}\beta_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \bigwedge_{y=1}^{2}\varrho_{xy}\mathfrak{f}_y(\eta_y((t-\alpha_1)) \\ \qquad + \bigvee_{y=1}^{2}\varphi_{xy}\mathfrak{f}_y(\eta_y(t-\alpha_1)) + \sigma_{1x}(t) + I_x(t), \\ {}^{C}\nabla_0^{0.95}\eta_x(t) = -a_x\eta_x(t) + \psi_x\chi_x(t-\alpha_2) + \bigwedge_{y=1}^{2}\bar{\varrho}_{xy}\chi_y(t-\alpha_2) \\ \qquad + \bigvee_{y=1}^{2}\bar{\varphi}_{xy}\chi_y(t-\alpha_2) + \sigma_{2x}(t) + J_x(t), \end{cases} \tag{16}$$

where $B = \begin{bmatrix} 0.95 & 0 \\ 0 & 0.95 \end{bmatrix}$, $\beta = \begin{bmatrix} 0.02 & -0.01 \\ -0.03 & 0.05 \end{bmatrix}$, $\varrho = \begin{bmatrix} 0.01 & 0.018 \\ 0.01 & -0.05 \end{bmatrix}$, $\varphi = \begin{bmatrix} 0.08 & -0.04 \\ 0.05 & 0.09 \end{bmatrix}$ $A = \begin{bmatrix} 0.98 & 0 \\ 0 & 0.98 \end{bmatrix}$, $\psi = \begin{bmatrix} -0.01 & 0 \\ 0 & 0.03 \end{bmatrix}$, $\bar{\varrho} = \begin{bmatrix} -0.02 & 0.05 \\ -0.015 & 0.065 \end{bmatrix}$, $\bar{\varphi} = \begin{bmatrix} 0.024 & 0.02 \\ 0.04 & -0.05 \end{bmatrix}$, $H_1 = \begin{bmatrix} 0.8 & 0 \\ 0 & 0.4 \end{bmatrix}$, $H_2 = \begin{bmatrix} 1.1 & 0 \\ 0 & 0.6 \end{bmatrix}$, $\wp_1 = \begin{bmatrix} 0.8 & 0 \\ 0 & 0.8 \end{bmatrix}$, $\wp_2 = \begin{bmatrix} 0.67 & 0 \\ 0 & 0.9 \end{bmatrix}$,

and the nonlinear regulation function $\mathfrak{f}_y(\varsigma_y(t)) = \dfrac{\varsigma_y^2(t)}{1+\varsigma_y^2(t)}$, *which is a Hill-type function. The time delays are set to* $\alpha_1 = 3, \alpha_2 = 4$, *and the external inputs are* $\sigma_{1x}(t) = \cos(t)$, $\sigma_{2x}(t) = \sin(t)$. *It can be verified that Assumption 2 holds with* $K^+ = \operatorname{diag}\{0.65, 0.65\}$, *and its clear that* $\tilde{u}_1 = 1$ *and* $\tilde{u}_2 = 1$.

The controller gains in (7) *are chosen as*

$$R_1 = \begin{bmatrix} 0.3 & 0 \\ 0 & 0.4 \end{bmatrix}, \quad R_2 = \begin{bmatrix} 0.5 & 0 \\ 0 & 0.38 \end{bmatrix}, \quad R_3 = \begin{bmatrix} 0.3 & 0 \\ 0 & 0.47 \end{bmatrix}, \quad R_4 = \begin{bmatrix} 0.44 & 0 \\ 0 & 0.324 \end{bmatrix}.$$

With $\xi = 0.6$, *solving the LMI* (9) *using YALMIP yields the following feasible matrices:*

$$P = \begin{bmatrix} 0.3447 & 0 \\ 0 & 1.0138 \end{bmatrix}, \quad Q = \begin{bmatrix} 0.1030 & 0 \\ 0 & 0.3447 \end{bmatrix}, \quad M_1 = \begin{bmatrix} 0.1134 & 0.0024 \\ 0.0024 & 0.1942 \end{bmatrix}, \quad M_2 = \begin{bmatrix} 0.0157 & 0.0014 \\ 0.0014 & 0.0842 \end{bmatrix}.$$

Thus, all conditions of Theorem 3.2 are satisfied, and system (16) *under the controller* (7) *is globally dissipative. Moreover, the set*

$$\Omega = \left\{ (\chi^T, \eta^T)^T \in \mathbb{R}^4 : \|\chi\|_1 + \|\eta\|_1 \leq 3 \right\},$$

is a positively invariant and globally attractive set for system (16)*. Figures 8 and 9 depict the time evolution of the mRNA and protein concentrations and the corresponding chaotic trajectory, respectively. Figure 10 shows the output trajectories* $y_{1x}(t)$ *and* $y_{2x}(t)$*, while Figure 11 displays the control inputs* $I_x(t)$ *and* $J_x(t)$*. Finally, Figure 12 illustrates the time evolution of* $\|\chi\|_1 + \|\eta\|_1$*, confirming that the state remains inside the attractive set* Ω*.*

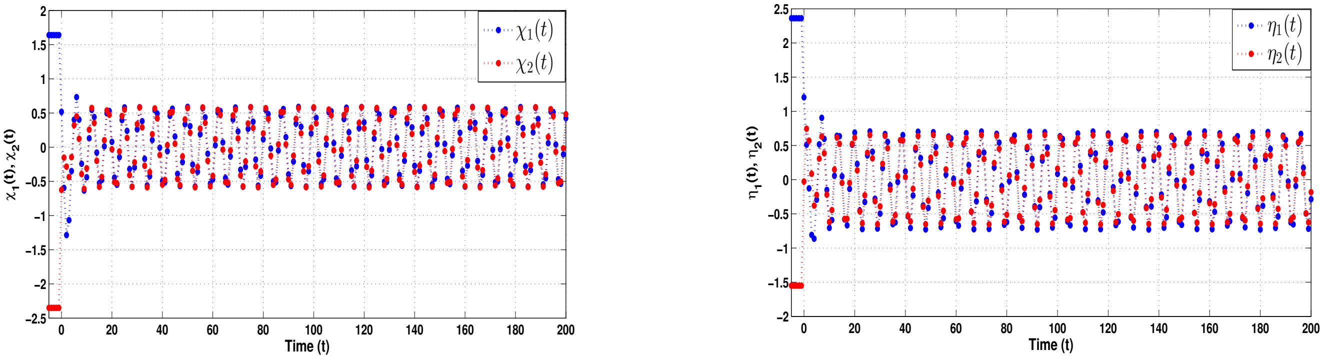

Figure 8: Time evolution of $\chi(t)$ and $\eta(t)$ in system (16).

Remark 4.3. *Although the numerical example consider a two-node genetic regulatory network for clarity of presentation, the proposed LMI conditions are formulated in a general form that is valid for an arbitrary number of genes* n*. While the dimension of the LMIs naturally increases with* n*, their mathematical structure and feasibility conditions remain unchanged. Therefore, the choice* $n = 2$ *is only for illustrative purposes and does not restrict the applicability of the proposed results.*

Remark 4.4. *The system parameters used in the numerical examples are selected to satisfy the sufficient conditions of the proposed LMIs and to prove the passivity and dissipativity analysis. These parameter choices are not unique and are representative of a broader class of admissible configurations for which the theoretical results remain valid.*

Remark 4.5. *The control gains* R_1*,* R_2*,* R_3*, and* R_4 *in* (7) *are selected to satisfy the linear matrix inequalities (LMIs)* (8) *and* (9) *in Theorems 3.1 and 3.2, thereby guaranteeing passivity (or dissipativity). If the LMIs are infeasible, the gains can be iteratively adjusted and the feasibility problem re-solved until admissible solutions are obtained.*

Remark 4.6. *In real biological systems, mRNA and protein concentrations are affected by numerous factors, leading to complex and heterogeneous dynamics. The proposed model adopts a simplified mathematical framework that captures the essential mRNA–protein interactions, incorporates memory effects via fractional-order operators, includes fuzzy MIN/MAX regulatory uncertainty, and considers time delays, enabling the derivation of passivity and dissipativity criteria. However, mutations, cell-to-cell variability, spatial heterogeneity, and other biological influences are not explicitly modeled. Thus, the framework represents a first step toward a rigorous mathematical foundation, with future work aimed at integrating greater biological complexity.*

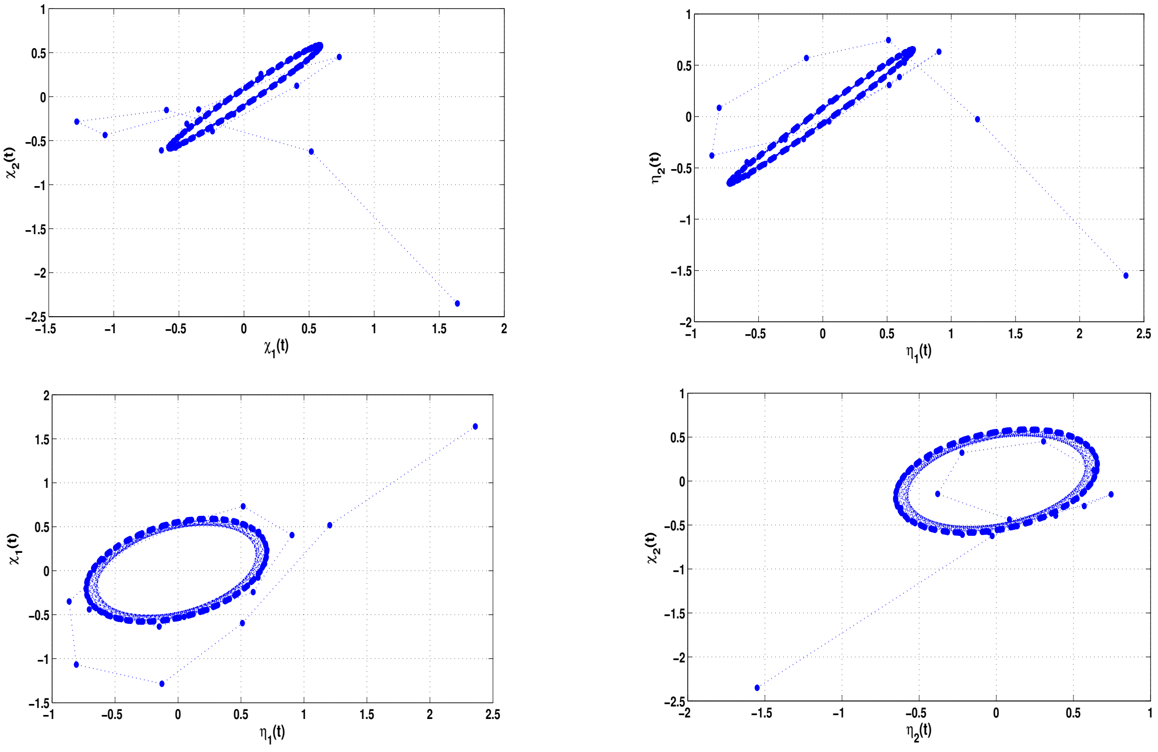

Figure 9: Chaotic trajectory of system (16).

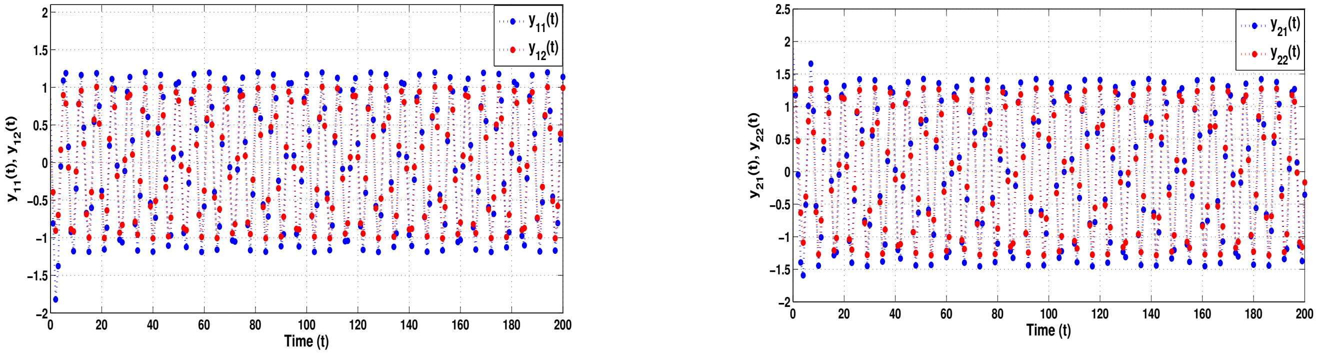

Figure 10: State trajectories of the outputs $y_{1x}(t)$ and $y_{2x}(t)$ of system (16).

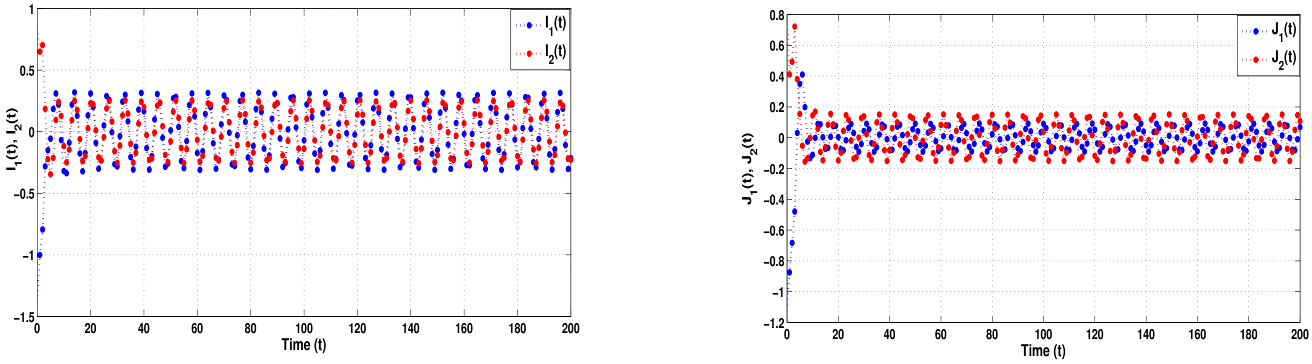

Figure 11: Time evolution of the control inputs $I(t)$ and $J(t)$.

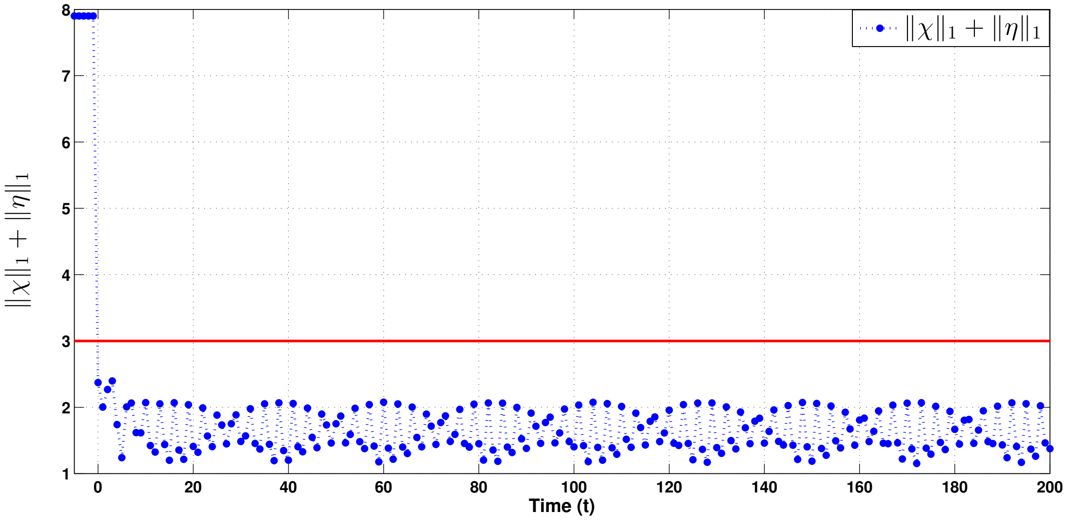

Figure 12: Time evolution of $|\chi|_1 + |\eta|_1$ in model (16) under controller (7).

5 Conclusion and future developments

This study has successfully addressed the challenges related to a class of FODTFGRNs. A new discrete-time fractional passive lemma has been formulated within a Lyapunov functional framework. By combining Lyapunov functionals with the LMI technique, we have provided novel requirements to secure the passivity and dissipativity of the proposed model. To validate the theoretical findings, two numerical examples accompanied by simulations were presented, demonstrating the effectiveness and applicability of the results. As far as we are aware, this work marks the first study tackling the passivity and dissipativity of FODTFGRNs, which highlights the originality of the contribution. Future research may proceed in several promising directions. Extending the present framework to more complex and large-scale network topologies, including multi-layer genetic regulatory structures, would further enhance its applicability. In addition, investigating the effects of stochastic disturbances, impulsive dynamics, environmental factors, and parametric uncertainties would improve the robustness and biological realism of the model. Exploring potential applications in medical diagnosis, biotechnology, and synthetic biology could also strengthen the practical relevance of the proposed methodology. Altogether, these perspectives will not only deepen the theoretical development of passivity and dissipativity analysis for fractional-order discrete-time systems but also broaden their impact in the study and control of complex nonlinear biological networks.

Acknowledgement

The authors wish to express their appreciation for several excellent suggestions for improvements in this paper made by the referees.

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