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M-matrix-based globally asymptotic stability criteria for genetic regulatory networks with time-varying discrete and unbounded distributed delays

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ABSTRACT

The problem of globally asymptotic stability for nonnegative equilibrium points of genetic regulatory networks (GRNs) with time-varying discrete delays and unbounded distributed delays is considered. So far, there are very few results concerning the problem; and in which the nonnegativity of equilibrium points is neglected. In this paper, the existence of nonnegative equilibrium points is firstly presented. Then, by using the nonsingular M-matrix theory and the functional differential equation theory, M-matrix-based sufficient conditions are proposed to guarantee that the kind of GRNs under consideration here has a unique nonnegative equilibrium point which is globally asymptotically stable. The M-matrix-based stability criteria derived here can be easily verified, since they are to check whether a constant matrix is a nonsingular M-matrix. Several numerical examples are offered to illustrate the effectiveness of the approach proposed in this paper.

1. Introduction

In order to understand interactions among messenger Ribonucleic Acids (mRNAs) and proteins, the concept of genetic regulatory networks (GRNs) was first proposed in 1960s. Since then, GRNs have received wide attention from many experts and scholars [1–7]. Based on the theoretical analysis and simulation experiments, several simple GRN models have been successfully established in the past two decades. Basically, there are two types of models for GRNs, that is, the differential equation model [1–3,7,8] and the Boolean model [5,6]. In GRNs, mRNAs and proteins may be synthesized at different locations; thus, the transcription or the diffusion of mRNAs and proteins among these locations results in sizable delays [1]. Therefore, a GRN model without consideration of delay is generally inaccurate, and even provides wrong predictions [1,7,8]. A functional differential equation is a differential equation including delayed states. So, it is more accurate to model GRNs by functional differential equations, which can better show the nature of life. Moreover, the functional differential equations have been used to describe various practical systems, including systems of infectious diseases and epidemics [9,10], population dynamics [11], neural networks [12,13], vehicle active suspension [14], and biological and chemical kinetics [15,16].

As we all know, stability is one of the most important properties for any dynamic system. Since the time delays often lead to poor performance of systems, and even make system instable [12,17–20], a great number of sufficient conditions' testing stability of equilibrium points of GRNs, modeled by functional differential equations only with discrete delays, have been proposed (see [1,7,21–25] and the references therein). In addition, for individual molecules, movement of mRNA from a transcription site to translation sites is an active process with a significant range of transport times, so it is significant and necessary to model GRNs by using functional differential equations with mixed (i.e., discrete and distributed) delays [8]. And stability criteria for GRNs only with discrete (distributed) delays are generally unavailable for GRNs with mixed delays. For this reason, the stability analysis for equilibrium points of GRNs with mixed delays has received more and more attention from experts and scholars (see [8,26–30] and the references therein). All of these stability criteria established in these papers except [8] are in the form of linear matrix inequalities (LMIs). In order to reduce conservativeness of LMI-based stability criteria, some useful approaches have been introduced, e.g., delay decomposition approach [28], reciprocally convex combination approach [26], augmented Lyapunov functional approach [29], free-weighting matrix approach [27,29], convex combination approach [27] and delay-probability-distribution-based model

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transformation approach [27,30]. These approaches are generally available for reducing conservativeness, but they will also increase the number of LMIs or variables in LMI(s), which results in the computational complexity. For this reason, the so-called M-matrix-based approach has been proposed by Zhang, Wu and Zou in [8] to infer the stability for equilibrium points of GRNs with time-varying discrete delays and constant distributed delays. Compared with the LMI-based stability criteria, an M-matrix-based one possesses less computational complexity because it needs only to verify whether a constant matrix is a nonsingular M-matrix. It is worth emphasizing that, for GRNs with discrete and *unbounded* distributed delays, Zhang et al.'s approach in [8] is available to present existence conditions of nonnegative equilibrium point; however, it does not apply to establish M-matrix-based globally asymptotic stability criteria, since the Lyapunov function employed in [8] is invalid to the *unbounded* distributed delays. Therefore, it is interesting and important to give M-matrix-based stability criteria for equilibrium points of the GRNs with discrete and *unbounded* distributed delays.

In this paper we will develop an M-matrix-based approach to establish globally asymptotic stability criteria for nonnegative equilibrium points of the GRNs with time-varying discrete delays and *unbounded* distributed delays. Firstly, the problem formulation and some preliminary results are presented (see Section 2 below). Then, based on the nonsingular M-matrix theory and the functional differential equation theory, M-matrix-based sufficient conditions are given to guarantee that the kind of GRNs under consideration here has a unique nonnegative equilibrium point which is globally asymptotically stable (see Section 3 below). Finally, the effectiveness of the theoretical results obtained in this paper is illustrated by the simulation results of several numerical examples (see Section 4 below).

In addition, the globally asymptotic stability criteria obtained in this paper are also available for GRNs only with discrete delays by setting $V=0$. In this case, the results of this paper can be viewed as a extensive and supplementary version of the corresponding results in [7,8,21,24,25].

Notation: Throughout this paper, the set of real numbers will be denoted by $\mathbb{R}$. Let $\mathbb{R}^{n\times m}$ represent the set of all $n\times m$ matrices over $\mathbb{R}$. Set $\mathbb{R}^n=\mathbb{R}^{n\times 1}$. For a matrix $A=[a_{ij}]\in\mathbb{R}^{n\times m}$, we use $|A|$ to denote the $n\times m$ matrix $[|a_{ij}|]$, and use $\chi_j(A)$ to denote the number of nonzero elements in the jth row of A. Set $\chi(A)=\mathrm{diag}(\chi_1(A),\chi_2(A),\ldots,\chi_n(A))$. For two real matrices $A=[a_{ij}]$ and $B=[b_{ij}]$, we say $A\le B$ ($A<B$) if $a_{ij}\le b_{ij}$ ($a_{ij}<b_{ij}$) for all i and j, and we denote by $A\circ B$ the Hadamard product of A and B, that is $A\circ B=[a_{ij}b_{ij}]$. For given positive numbers $a_1,a_2,\ldots,a_n$, let Λ_a denote the diagonal matrix $\mathrm{diag}(a_1,a_2,\ldots,a_m)$. Let $\|\cdot\|_2$ represent the 2-norms of vectors or matrices. A connected subset of $\mathbb{R}$ is called an interval. For given an interval $\mathbb{J}$ and a positive integer n, the set of all continuous functions $h:\mathbb{J}\to\mathbb{R}^n$ is denoted by $C(\mathbb{J},\mathbb{R}^n)$. It is a linear space with respect to the usual operations on functions, and is further a Banach space with respect to the norm $\|\cdot\|$ defined by

$$\|h\|=\sup_{s\in\mathbb{J}}\|h(s)\|_2,\quad \forall h\in C(\mathbb{J},\mathbb{R}^n).$$

Let $\mathcal{C}((-\infty,0],\mathbb{R}^n)$ be the Banach space of functions $\psi\in C((-\infty,0],\mathbb{R}^n)$ such that $\psi$is bounded and uniformly continuous, with norm

$$\|\psi\|_{\mathcal{C}}:=\sup_{-\infty<s\le 0}\|\psi(s)\|_2+\int_{-\infty}^{0}\|\psi(s)\|_2\,\mathrm{d}s<\infty.$$

Superscript T denotes the matrix transposition.

2. Problem formulation

GRNs describe how the interactions among mRNAs and proteins, and are usually modeled as [1,8]:

$$\dot m_i(t)=-k_{mi}m_i(t)+\sum_{j=1}^{n}f_{ij}g_j(p_j(t-\tau_{pj}(t)))+J_i,\quad t\ge 0, \tag{1a}$$

$$\dot p_i(t)=-k_{pi}p_i(t)+r_im_i(t-\tau_{mi}(t)),\quad t\ge 0, \tag{1b}$$

$$m_i(t)=\varphi_i(t),\quad p_i(t)=\psi_i(t),t\in[-d,0], \tag{1c}$$

(see Fig. 1 for an intuitive explanation [31]) where $i\in\langle n\rangle$, $m_i(t)$ and $p_i(t)$ denote the concentrations of mRNA i and protein i at time t, respectively; k_{mi} and k_{pi} are positive real numbers that denote the degradation rates of mRNA i and protein i, respectively; r_i is a positive real number that denotes the rate of translation from mRNA i to protein i; $g_j(x)=(x/b_j)^{h_j}/(1+(x/b_j))^{h_j}$, b_j is a positive scalar, and $h_j\ge 1$ is the Hill coefficient that denotes the degree of cooperativity;

$$f_{ij}=\begin{cases}-a_{ij} & \text{if transcription factor } j \text{ represses gene } i,\\ 0 & \text{if transcription factor } j \text{ does not regulate } i,\\ a_{ij} & \text{if transcription factor } j \text{ activates gene } i,\end{cases}$$

a_{ij} is the dimensionless transcriptional rate of transcription factor j to gene i, which is nonnegative and bounded constant; $J_i=\sum_{j\in S_i}a_{ij}$, $S_i=\{j: j\in\langle n\rangle, f_{ij}<0\}$; $\varphi_i,\psi_i\in C([-d,0],\mathbb{R}^n)$, $d=\max\{\overline{\tau}_m,\overline{\tau}_p\}$ with

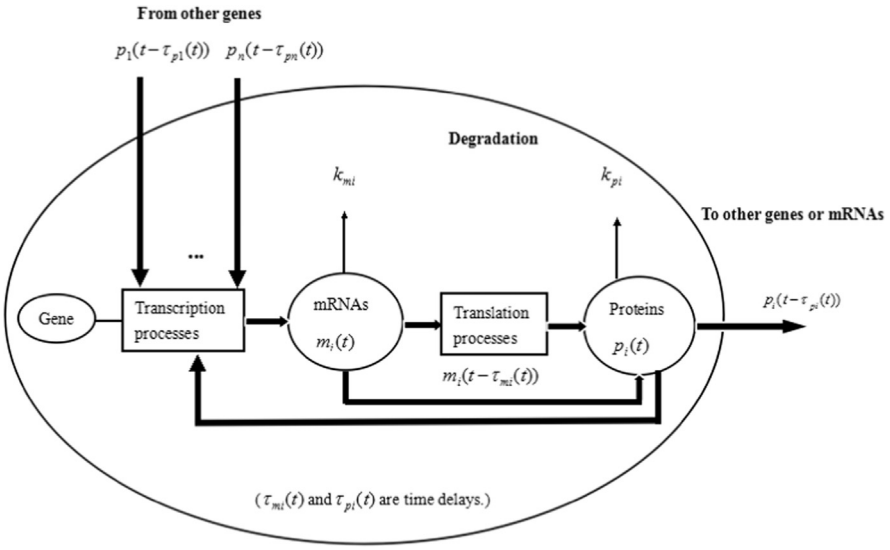

Fig. 1. Genetic regulatory network with a feedback loop for transcription and translation processes.