

Symmetry and stochastic gene regulation

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Short Abstract — A binary stochastic model to gene expression, containing Lorentz-like Lie symmetry $SO(2,1)$ is presented. The invariant of the algebra characterizes the dynamical evolution of the gene switch. The azimuthal eigenvalue describes the affinity between the regulatory protein and the gene operator site. Raising and lowering operators are made and act enlarging or reducing the affinity parameter. The noise state of the gene is given from the group theoretical numbers.

Keywords — Stochastic gene regulation, Lie symmetry, auto-regulatory system

I. PURPOSE

Understand the dynamical functioning of the genetic networks is an important open problem in the post genomic era. The basic mechanisms of gene regulation are enlightened from works on lambda-phage [1] and lac operon [2] furnishing the fundamental tools to the description of intricate gene webs. These networks control the biological processes occurring inside the cell and present a high level of complexity, even in a prokaryotic organism. A way to approach this complexity is to consider the elementary constituents of the gene circuits corresponding to specific functions [3]. The current status of experimental techniques permits the investigation of those building blocks [4].

Randomness is a inherent feature of intracellular chemical reactions, since that these systems are low copy number consisted [5]. That aspect can be confirmed experimentally by observing that the quantity of a given fluorescent protein in a single cell deviates in relation to its mean number in the cellular culture [6]. Modeling of those phenomena is achieved in two schemes. The former consider non-linear phenomenological equations for proteins concentrations, here noise is introduced by the Langevin mechanism. The latter employs stochastic master equations for probability distributions of proteins. Mean values, square root deviation are then calculated from probabilities. In this way a binary (spin boson) model have been proposed [7], and solved analytically for a self interacting gene [8] and a externally induced gene [9]. Both analytical solutions are given in terms of the confluent hypergeometric functions [10].

Here we show that the equations of the model present Lorentz-like Lie symmetry $SO(2,1)$. In this way, the differential form of the operators of the algebra is given. The eigenstates are defined in terms of the analytical solutions of the model [11].

The Casimir operator's eigenvalue have been calculated and has a precise biological meaning. In order to obtain it the time-dependent master equation is considered and we analyze the decaying rate of the system to the steady state. We observe that the Casimir operator's eigenvalue is proportional to the mean life of the non-stationary state of the gene.

The azimuthal operator's eigenvalues give the affinity between the gene operator site and the regulatory protein. Their high values imply in the adiabatic regime as defined at [hornos], in contrary the gene assumes a non-adiabatic state.

The noise of the system is characterized by summing the algebraic eigenvalues. If the sum results positive, null or negative the associated fluctuations are characterized as super-Fano, Fano, and sub-Fano, respectively.

We have been observed that the decaying rate of the gene switch to the steady state is obtained from the calculation of the Casimir operator's eigenvalues. The adiabaticity parameter arises from the azimuthal operator's eigenvalues. By summing those algebraic numbers we classify the noise state of the gene products.

The success of the model in description of biological modules is a guess to application to systems composed by two or more systems. Group theoretical tools can be used in the search of a composition rule between individual genes. A second possibility is the description of genes containing more than two levels of regulation by considering the matrix representation of the algebra.

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