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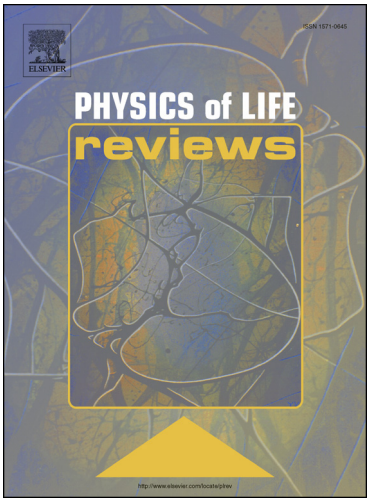

Dissecting genomic imprinting and genetic conflict from a game theory prospective

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Comment

Dissecting genomic imprinting and genetic conflict from a game theory prospective: Comment on: "Epigenetic game theory: How to compute the epigenetic control of maternal-to-zygotic transition" by Qian Wang et al.

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Game theory as a tool to model genomic imprinting

Epigenetics typically refers to changes in the structure of a chromosome that affect gene activity and expression. Genomic imprinting is a special type of epigenetic phenomenon in which the expression of an allele depends on its parental origin. When an allele inherited from the mother (or father) is imprinted (i.e., silent), it is termed as maternal (or paternal) imprinting. Imprinting is often resulted from DNA methylation and tends to cluster together in the genome [1]. It has been shown to play a key role in many genetic disorders in humans [2]. Imprinting is heritable and undergoes a reprogramming process in gametes before and after fertilization [1]. Sometimes the reprogramming process is not reversible, leading to the loss of imprinting [3]. Although efforts have been made to experimentally or computationally infer imprinting genes, the underlying molecular mechanism that leads to unbalanced allelic expression is still largely unknown.

The game theory proposed by Wang et al. [4] provides a promising tool to understand the epigenetic control of maternal-to-zygotic transition that leads to genomic imprinting. Game theory was originally proposed to solve economic and financial problems, but it has been extended to biological fields with applications such as for gene mapping [5] and for understanding the evolution of ATP-producing pathways [6]. As the major contributing factor for genomic imprinting, DNA methylation is a dynamic reprogramming process where methylation signals are removed during the gamete stage and are subsequently reprogrammed after fertilization [7]. Recent studies show that many epigenetic events are spontaneous, and meiotically stable epialleles can be produced due to changes in DNA methylation [8]. Such transgenerational epigenetic variation in DNA methylation can lead to new epialleles that alter gene expression and morphology, providing new insights for phenotypic diversity [9]. At the molecular level, the degree of epigenetic changes largely relies on the cooperation and competition between the two sets of gametes, the so called epiallelic interactions. Such interactions have consequences on individual fitness. As pointed out in the paper, "each sex tends to maximize the expression of its genes in the zygote toward the maximum fitness of the progeny" [4]. Thus, the maternally and paternally derived alleles tend to compete with each other to gain the advantage of natural selection [4]. In the meanwhile, they both

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