S107

Analysis of the Genetic Network of Calcium Signalling Components in Myoblasts

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Intracellular Ca^{2+} ion is an important secondary messenger of signalling that controls and executes diverse cellular functions. Therefore, maintaining the Ca^{2+} homeostasis is absolutely required for normal physiology, and for that matter, cells constantly remodel the calcium metabolism system by supplementing defects through the regulation of gene expression. Our study aimed to formulate the genetic network that regulates the expression of Ca^{2+} metabolism genes through a gene expression analysis using DNA chips and in-silico promoter analysis of the genes of calcium signalsomes. Cells were treated with drugs modifying intracellular calcium metabolism and then analyzed for the changes in the expression of calcium signalsome components. We found that functionally related genes of calcium signalling tend to be co-expressed in response to the perturbation of calcium homeostasis. We formulated a genetic network for calcium signalsome genes by correlating their expression pattern with that of transcription factors, and found that members of the same group of co-expressed calcium signalsome genes share many transcription factors in the genetic network, suggesting that they might be indeedco-regulated by a shared genetic circuit. The co-regulation pattern also appeared to be spatially separated inside the cell, reflecting the necessity for the functionally bound genes residing in the same compartment to be coordinately regulated in order to cope with the perturbation more effectively. Moreover, gene groups having opposite functions in calcium metabolism showed reciprocal expression patterns as the result of a set of shared transcription factors controlling the regulatory circuit in an opposed manner for different gene groups. Our results demonstrate the presence and operation of an efficient compensatory regulation system for the calcium signalsome and provide experimental evidence supporting the calcium-induced calcium-signalling remodeling hypothesis.