Perspective

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Interaction between Feedbacks and Molecular Memory Affects Gene Expression

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Description

Due to molecular memory between individual reactions, gene expression is a non-Markovian discrete stochastic process that involves many small biochemical reaction steps. As of now, this interaction is effectively explored by summed up substance ace condition models. However, the role of feedback networks in gene expression is not taken into account by these models. It is still unclear how the interaction between molecular memory and feedbacks affects gene expression. Positive and negative feedback-based generalized chemical master equation models of gene expression are developed in this paper. We get the analytical expression for this model in a steady state and the measure of the noise of protein numbers if the process of making proteins follows an Erlang probability distribution. In addition, we discover that molecular memory competes with positive feedback in the suppression of protein number noise. Molecular memory has the potential to intensify the degree to which it reduces this noise in our model with negative feedback. These intriguing findings suggest that molecular memories play an equally significant role in influencing gene expression as feedbacks do.

Neuronal Activation and Other Cellular Processes

Leptin is a 16 kDa satiety hormone whose circulating levels are inversely correlated with body fat mass. It is primarily derived from adipocytes. Additional peripheral tissues, such as the heart, produce leptin. It can have both central and local effects because it activates cells through the leptin transmembrane receptor, which is found in numerous brain regions and peripheral organs like the heart. When leptin binds to ObR, a number of intracellular signaling cascades, such as IRS/PI3K/JAK2/AKT, JAK2/STAT3, SHP2/MAPK, and AMPK/ACC are activated. These cascades can control transcription, translation, and the metabolism of cells. As a result, neuronal activation and other cellular processes like proliferation, growth, survival, apoptosis, and apoptosis can be affected by leptin. Leptin is a hormone that regulates the melanocortine/neuropeptide Y system in the hypothalamus to reduce food intake, increase energy expenditure, and reduce body weight. Numerous cis-elements in the gene promoter that are able to respond to multiple transcription factors, including HIF-1, NF-kB, and AP-1, all of which are responsive to hypoxia indicate that the leptin gene itself is open to a variety of regulatory controls. Findings from ob/ob and db/db mice with spontaneous mutations in the genes encoding leptin or ObR, respectively suggested that leptin has a beneficial effect on the heart. Additionally, it has been reported that leptin protects cultured cardiac-related cells from hypoxia-induced toxicity, apoptosis after H2O2 treatment, serum deprivation and TNF administration or both. It should be noted that leptin in the heart was also reported to have some negative effects. Particularly hypertrophy, most likely caused by leptin's potential growth effects. In contrast to mice that are restricted in calories and experience persistent hunger, MUPA transgenic mice exhibit an unusual combination of low calorie intake and satiated animal metabolism. MUPA mice differ from their wild-type (WT) ancestors, the FVB/N, in that they have a modified circadian rhythm and naturally elevated circulating levels of leptin throughout their entire lives. When a leptin antagonist was given to the mice, it was found that leptin was largely to blame for the MUPA metabolic phenotype. MUPA mice live significantly longer for both sexes. At least the cardiac aging, fractional shortening, infarct size, inflammatory response, and elevated serum leptin levels were all reduced in the female mice. We demonstrated, through the use of leptin neutralizing antibodies and pharmacological inhibitors, that endogenous leptin was responsible for reducing the impairment in fractional shortening and infarct size following a heart attack in the MUPA heart.

Gene Ontology Enrichment Analysis

The development of ketosis in dairy cows that produce a lot of milk highlights the need to learn more about the genetics of metabolic diseases and how it affects animals' health. A meta-analysis of gene expression and genome-wide association studies was carried out so that the pattern of differential gene expression in the livers of cows under negative energy balance (NEB), as well as under subclinical and clinical ketosis, could be evaluated. Based on the key words "cow," "liver," "negative energy balance," "ketosis," "expression," "qPCR," "microarray," "proteomic," "RNA-Seq," and "GWAS," a preliminary systematic review identified 118 articles. Twenty articles were included in the analysis after further screening for only relevant, peer-reviewed articles on gene expression during NEB and clinical and subclinical ketosis considering plasma levels of -hydroxybutyrate. By considering chromosome and base pair positions in the ARS-UCD 1.2 bovine assembly, 430 significant SNPs identified by

genome-wide association studies were assigned to genes reported in gene expression studies from the systematic review. Gene Ontology enrichment analysis using official gene names was used to integrate the data from the systematic review using Venn diagrams. Additionally, a QTL enrichment analysis was carried out in order to locate potential candidate loci for positions. On chromosomes 2, 3, 6, 9, 11, 14, 27, and 29, the coordinates of differentially expressed genes contained 24 significant SNPs. NEB and subclinical and clinical ketosis was linked to three significant metabolic pathways. Furthermore, 2 significant qualities, PPARA (peroxisome proliferator enacted receptor alpha) and ACACA (acetyl-coenzyme A carboxylase α), were distinguished, which were differentially communicated in the 3 metabolic circumstances. The ACACA gene encodes an enzyme that catalyzes the carboxylation of acetyl-coenzyme A to malonyl-coenzyme A, a rate-limiting step in the synthesis of fatty acids, and the PPARA gene is involved in the regulation of lipid metabolism and fatty liver disease. Quality organization examination uncovered co-articulation collaborations among 34 qualities related with capabilities including unsaturated fat vehicle and unsaturated fat digestion. Nine QTL for ketosis were identified among the annotated QTL.QTL previously associated with the trait "ketosis" on chromosome 2 and the trait "milk iron content" on chromosome 14 were enriched for the genes FN1 and PTK2, which are primarily involved in cell adhesion and the formation of extracellular matrix constituents, respectively. The genetics of NEB and subclinical and clinical ketosis in dairy cattle can now be better understood thanks to this integration of gene expression and GWAS data. As a result, it is a useful strategy for locating the biological mechanisms that are responsible for these metabolic conditions in dairy cattle.