Abstracts

Functional genomics

Program/Abstract # 270

Computational analysis of gene expression patterns in the embryo using shape characteristics

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Images of structurally complex biological specimens (such as embryos), stained to visualise biological processes (such as gene expression) are routinely used in the study of developmental biology. Over the past several years we have pioneered the use of computational-based approaches to allow spatially complex patterns of gene expression to be analysed based on their shape characteristics. We have established a public resource (EMAGE), which houses 500,000+ images of in situ gene expression assays (for 10,000+ genes) that have been sourced from the community and the literature, and provides a suite of spatial analysis tools to explore similarities between the patterns (e.g. to find synexpression groups), or to find examples of patterns that have local spatial similarity to a query domain (e.g. "What genes are expressed in a stripe in a particular location?). The data can also be interrogated using text-based methods (e.g. "What genes are expressed in a named anatomical part (e.g. eye?)"). New data types to be incorporated in EMAGE this year include miRNA and putative enhancer element driven reporter data. The data and tools are freely available from http://emouseatlas.org/emage/. Visit the EMAGE trade stand at this meeting for more information and hands-on tutorials.

doi:10.1016/j.ydbio.2009.05.295

Program/Abstract # 271

Abnormal retinal development in the Btrc null mouse

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Btrc is required in the ubiquitin-dependent degradation of particular intracellular proteins. Previous micro array analysis revealed Btrc down-regulation in the retina of mouse embryos specifically lacking cholinergic amacrine cells (CACs) as a result of the absence of skeletal musculature and fetal ocular movements. An investigation of retinal morphology in Btrc−/− mouse fetuses showed a normal number of cell layers and cells per layer with normal cell proliferation and apoptosis. However, there was a complete absence of CACs and a decrease in tyrosine hydroxylase-expressing amacrine cells. The population size of other amacrine cell subtypes was similar to that in the wild-type retina, whereas the number of the amacrine precursor cells was decreased. There was also a reduction in the number of retinal ganglion cells, whereas their progenitor cell numbers were increased. These findings indicate that, despite having no apparent effect on basic retinal lamination, Btrc is involved in differentiation of CACs and possibly other retinal cell types, subtypes and their precursors. This work is funded by NSERC to B.K.

doi:10.1016/j.ydbio.2009.05.296

Program/Abstract # 272

Role of skeletal muscle in the epigenetic shaping of motor neuron fate choices

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Since July 2000, the members of the Mouse Models of Human Diseases Laboratory have been able to study the role of muscle in the epigenetic (N.B., we use this term with the broader and more integrative meaning) shaping of developing motor neuron fate choices employing an approach based on mouse mutagenesis and pathology. The developmental role of skeletal muscle is studied in the whole mouse embryo by knocking out myogenic regulatory factors Myf5 and MyoD, to obtain either an embryo without any skeletal musculature or embryos with myoblasts at different levels of commitment. By employing micro array analysis and a series of follow-up studies our goal is to perform a large scale functional analysis of the mammalian genome to find muscle-provided triggers of motor neuron death relevant to motor neuron diseases, such as amyotrophic lateral sclerosis. The reason for this kind of thinking is the fact that a complete absence of lower and upper motor neurons, which is the pathological definition of amyotrophic lateral sclerosis, is only achieved in the complete absence of the muscle. We hope that by employing the approach described in the presentation, it will be possible to significantly increase, in a short period of time, the number of factors known to enhance motor neuron survival from the skeletal muscle. This work is funded by CIHR and NSERC to B.K.

doi:10.1016/j.ydbio.2009.05.297