nerve sheath tumor (MPNST) cell line, both of which are NF1−/−, have also been shown to be SC-like in culture. The mESC can also be differentiated into neuron-like cells and the behavior and genetic repertoires of the cells under different developmental conditions can be compared. This system provides an ideal paradigm for studies of the role of NF1 in cell growth and differentiation.

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214
Role of the *Drosophila melanogaster bubblegum* and doublebubble genes in nervous system patterning and embryogenesis

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The *Drosophila melanogaster* homologous genes *bubblegum* (*bgm*) and *doublebubble* (*dbb*) code for very-long-chain fatty-acid (VLCFA) acyl-CoA synthetases required for VLCFA degradation. It has been previously shown that mutations in *bgm* lead to neurodegeneration characterized by a bubbly appearance of the optic lobe, a reduced life span, and visual impairment (Min KT and Benzer S, 1999, *Science* 284, 1985). Our analysis of the *bgm* and *dbb* spatial expression profiles in *Drosophila* embryos reveals both transcripts to be ventrally restricted: The *bgm* and *dbb* mRNAs are evident in presumptive mesoderm in the early embryo and in mesoderm derivatives during later embryonic stages. We have shown that *dbb* and *bgm* are (1) coregulated by the Dorsal and Twist transcription factors essential for specification of ventral cell fates during dorsoventral patterning in *Drosophila* embryos and (2) play redundant roles in *Drosophila* embryogenesis. Bioinformatics analysis reveals a short, highly conserved sequence in the chromosomal region between the *bgm* and *dbb* coding regions. In agreement with our hypothesis that this conserved sequence comprises an essential regulatory element for the *dbb* and/or *bgm* genes, *dbb* transcription is down-regulated in mutants homozygous for the P-element insertion that maps immediately upstream of this sequence and causes lethality at the postembryonic stages of *Drosophila* development. We are currently testing the hypothesis that the *bgm* and *dbb* gene products are biological effectors of ventral fate determination and nervous system development in *Drosophila*.

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215
A microarray for Olig2 targets identifies potential regulators of neural progenitor cell maintenance in the developing spinal cord

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Within the developing spinal cord, a series of instructive environmental signals commit spatially discrete domains of neural progenitor cells to the production of specific types of neurons and glia. Subsequently, each population proliferates to expand their pool size before exiting the cell cycle and terminally differentiating in a stereotypical manner. Within the motor neuron (MN) progenitor domain of the spinal cord, the bHLH transcription repressor Olig2 has been found to be a critical regulator of the switch between proliferating neural progenitors and differentiated MNs. The pathways through which Olig2 acts, however, are largely unknown. To identify target genes regulated by Olig2, we have carried out a microarray-based screen for genes deregulated in *Olig2* mutant versus wild-type spinal cords. Through this approach, we have identified several candidate regulators of neural progenitor maintenance, and we will report our recent progress in determining how Olig2 controls the expression of these genes and defining the function of these genes in neural progenitors.

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216
Characterization of a novel bHLH protein in the developing spinal cord

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The proneural basic helix–loop–helix (bHLH) transcription factors play a central role in the specification of neural cell types. Olig2 is a bHLH transcription factor that is exclusively expressed in the developing spinal cord in those progenitor cells that will form spinal motor neurons. Olig2 is thought to work as a transcriptional repressor, promoting motor neuron formation by suppressing the expression of genes that inhibit differentiation and those genes that direct the formation of spinal interneurons. A microarray screen for Olig2-regulated target genes identified the novel, structurally related bHLH protein BHLHB5 whose expression is associated with developing spinal interneurons. Preliminary analyses of developmental expression patterns show that BHLHB5 is expressed in select subclasses of spinal interneurons. In Olig2 mutant mice, BHLHB5 expands ventrally into the progenitor region that normally expresses Olig2 and this expansion corresponds to an increase in the appearance of ectopic interneurons. The function of BHLHB5 was assessed using in ovo electroporation of BHLHB5 and were analyzed using antibody probes to determine the effect of misexpression. Expression of BHLHB5 was knocked down by in ovo electroporation of siRNAs and analyzed in the same manner. Results from these experiments are contributing to an expanded model of