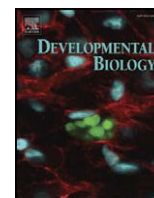


Contents lists available at [ScienceDirect](http://ScienceDirect)

# Developmental Biology

journal homepage: [www.elsevier.com/developmentalbiology](http://www.elsevier.com/developmentalbiology)

## Abstracts

### Cell proliferation

#### Program/Abstract # 261

Withdrawn

doi:10.1016/j.ydbio.2008.05.278

#### Program/Abstract # 262

##### Maternal-effect brambleberry functions during cleavage stage to maintain nuclear integrity

Elliott W. Abrams, Florence Marlow, Lee Kapp, Tripti Gupta, Mary Mullins

University of Pennsylvania, Department of Cell and Developmental Biology, Philadelphia, PA, USA

Embryonic development in most animals initiates with a series of rapid and synchronous cell cleavages. Cleavage cycles are mediated by maternal factors present in the egg and are missing cell cycle checkpoints. Understanding how chromosome integrity is maintained during this very critical period of metazoan development is currently lacking. By performing a forward genetic screen in zebrafish, we recently identified a maternal-effect mutant, *brambleberry* (*bmb*), which exhibits profound chromatin defects during the cleavage stage of development. Interphase *bmb* nuclei appear as individual clusters of chromatin bodies and are sometimes associated with a few DAPI-stained microfragments. Examination of the mitotic spindle indicates that chromosomes are not properly aligned on the metaphase plate during mitosis. Interestingly, inspection of the nuclear envelope reveals that during interphase chromatin bodies appear individually encased in separate nuclear envelopes. Surprisingly, *bmb* nuclear morphology is greatly improved just after the mid-blastula transition (MBT), despite *bmb* mutants arresting development at this point. This effect is not diminished when both maternal and paternal functions of *bmb* are eliminated, suggesting that *bmb* functions specifically during the cleavage stage of embryonic development. Further characterization of *bmb* mutants involves examining other mitotic components and aspects of nuclear/chromatin structure. Finally, significant effort is being put forth into the positional cloning of the *bmb* gene to help elucidate its molecular function.

doi:10.1016/j.ydbio.2008.05.279

#### Program/Abstract # 263

##### Developmental regulation of cell division mechanisms in a vertebrate embryo

Esther Kieserman<sup>a</sup>, Michael Glotzer<sup>b</sup>, John B. Wallingford<sup>a</sup><sup>a</sup> Department of Molecular Cell and Developmental Biology, ICMB, University of Texas at Austin, Austin, TX, USA<sup>b</sup> Department of Molecular Genetics and Cell Biology, University of Chicago, Chicago, IL, USA

Proper completion of cell division in organisms is critical for morphogenesis and embryogenesis. Comparatively little analysis has been made of core cytokinesis mechanisms in developing vertebrate embryos. We have examined cell divisions in two ectodermally-derived tissues of the developing *Xenopus* embryo using 4D confocal microscopy. This analysis has identified striking differences in fundamental mechanism of cell division in the neural plate as compared to tail epidermal cells. The division mechanisms in later epidermal cells reflect those described for differentiated cells in culture. By comparison, cells in the closing neural tube display an exaggerated anaphase B, delayed cytokinesis onset and a pronounced reduction of microtubule density in the spindle midzone. The mechanism of cell division in normal neural plate cells is reminiscent of that observed in cultured cells experimentally depleted of PRC1. We find that PRC1 protein expression is reduced in neural tube cells. Forced expression of PRC1 in neural cells abrogates the extensive spindle elongation and the absence of midzone microtubules. Finally we find that changes in midzone microtubules reflect differences in the midbody. We therefore suggest that the divergent cell division mechanism we observed in neural plate cells may be related to known specializations of the midbody in neural cells. These data demonstrate that the central spindle and midbody are developmentally-regulated structures, and reveal unexpected plasticity to fundamental mechanisms of cell division.

doi:10.1016/j.ydbio.2008.05.280

#### Program/Abstract # 264

##### Differentiation of trophoblast stem cells into giant cells is triggered by p57 inhibition of CDK1 activity

Matthew J. Kohn<sup>a</sup>, Zakir Ullah<sup>a</sup>, Rieko Yagi<sup>a</sup>, Lyubomir Vassilev<sup>b</sup>, Melvin DePamphilis<sup>a</sup><sup>a</sup> Program in Genomics of Development, NICHD, NIH, Bethesda, MD, USA<sup>b</sup> Department of Discovery Oncology, Roche Research Center, Nutley, NJ, USA