

Stanford

Julie Baker

Professor of Genetics

Bio

ACADEMIC APPOINTMENTS

- Professor, Genetics
- Member, Bio-X
- Member, Child Health Research Institute

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

With the complete sequence of the human and mouse genomes on the horizon, the critical next step in genomic analysis is to provide functional criteria for the newly discovered proteins. Understanding the diverse protein functions present within complex genomes must encompass many different and unique approaches. These functional approaches currently include two-hybrid screens for interacting proteins, microarray techniques for visualizing expression complexity, and other assays designed to elucidate specialized functions. We have developed one such functional-based assay that we are utilizing to identify potentially hundreds of molecules that can alter specific cell-fate responses.

Our main focus is to understand the signals necessary for patterning and specifying diverse cellular fates during gastrulation in the mouse. Mouse gastrulation, even more so than amphibian and teleost gastrulation, is a period of vast differentiation and growth. During this stage, the mouse embryo transitions from having only two cell types, to having hundreds. Although an incredibly rich source of cell signaling, the mouse gastrula has not been used by molecular biologists to mine for molecules. This is mainly due to the size (100mm) and inaccessibility of the mouse gastrula, which therefore precludes the effective use of biochemistry, embryology and molecular assays in general.

We have devised a screen that taps the identity of molecules involved in cell-fate specification during mouse gastrulation. This approach delivers random combinations of cDNAs from mouse gastrula libraries into the more tractable *Xenopus* embryo. We then observe these embryos for changes in specific marker gene expression, indicating changes # positive or negative # in cell-fate. We are particularly interested in the alteration of mesodermal, endodermal, neural, endothelial and somitic cell-fate decisions. By proceeding with a trial run of the screen, we have already identified 17 molecules, 8 of which have no previously understood function.

Of the 8 unexplored molecules identified, we are currently characterizing 4 in-depth. One of these inhibits vasculogenesis and causes the ectopic formation of neurons. Another is an endogenous inhibitor of MAP Kinase signaling and is required for the formation of mesodermal cells. Two others can induce the formation of endoderm. Studies on these proteins, and others like them, are on going in our laboratory.

Teaching

COURSES

2017-18

- Living with Viruses: THINK 61 (Aut)

2016-17

- Advanced Genetics: GENE 205 (Win)

2015-16

- Advanced Genetics: GENE 205 (Win)

2014-15

- Advanced Genetics: GENE 205 (Win)

STANFORD ADVISEES

Postdoctoral Faculty Sponsor

Guillaume Cornelis, Elisa Zhang

Doctoral Dissertation Advisor (AC)

Michael Guernsey

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Genetics (Phd Program)

Publications

PUBLICATIONS

- **The E3 ubiquitin ligase GREUL1 anteriorizes ectoderm during *Xenopus* development** *DEVELOPMENTAL BIOLOGY*
Borchers, A. G., Hufton, A. L., Eldridge, A. G., Jackson, P. K., Harland, R. M., Baker, J. C.
2002; 251 (2): 395-408
- **Wnt signaling in *Xenopus* embryos inhibits Bmp4 expression and activates neural development** *GENES & DEVELOPMENT*
Baker, J. C., Beddington, R. S., Harland, R. M.
1999; 13 (23): 3149-3159
- **From receptor to nucleus: the Smad pathway** *CURRENT OPINION IN GENETICS & DEVELOPMENT*
Baker, J. C., Harland, R. M.
1997; 7 (4): 467-473
- **A novel mesoderm inducer, Madr2 functions in the activin signal transduction pathway** *GENES & DEVELOPMENT*
Baker, J. C., Harland, R. M.
1996; 10 (15): 1880-1889
- **A human Mad protein acting as a BMP-regulated transcriptional activator** *Nature*
J. C. Baker, Liu, F., A. Hata, J. Doody, J. Carcamo, R. M. Harland, J. Massague
1996; 381