On January 4, 2012 the Laboratory of Genetics lost a beloved colleague, friend, and inspiration in Professor Jim Crow, who passed away peacefully just shy of his 96th birthday. Jim was a revered researcher, teacher, and public servant whose career spanned over 70 years. His groundbreaking research in population genetics spanned a wide variety of topics, including molecular evolution, statistical genetics, natural variation, plant and animal breeding, the genetic effects of radiation, and much more.

Jim joined the Genetics faculty in 1948, after beginning his career at Dartmouth College. His early work in Drosophila fostered his future investigations of human and animal genetics. Although he retired in 1986, Jim remained an active member of the faculty until his death, authoring over 250 publications.

Jim was also a renowned teacher. He penned his famous ‘Crow’s Notes’ for his General Genetics course and the classic textbook Introduction to Population Genetics Theory with his former student Kimura. During his time at UW-Madison, Jim also served as Chair of the Laboratory of Genetics, Dean of the School of Medicine and Public Health, president of several professional organizations, and a member of over a dozen national advisory and review committees.

Jim was known to the world as an outstanding geneticist, teacher, and statesmen – but to many of us in the Laboratory of Genetics, he was simply one of the best colleagues one could ask for. He was in his office most days – up until the end of his life – reading papers, writing manuscripts, and keeping up on the latest news in and out of science. He seemed perpetually in a good mood, and could talk to anyone about any subject matter. He always asked the most interesting and insightful questions at seminars, yet was so down to earth that he managed to include everyone in the discussion. He is sorely missed.

To honor Professor Crow, a fund has been established to create an endowed Professorship that bears his name. The Professorship will be used to attract and support the research of a world-class scientist to join the faculty in Genetics. Please join us in celebrating Jim by contributing to the James F. Crow Professorship Fund (http://www.genetics.wisc.edu).
The 6th Annual Smithies Symposium will be held on May 29, 2013, and will host three nationally renowned scientists with a focus on developmental biology.

Nobel laureate (1995) Eric Wieschaus from Princeton will highlight the patterning that occurs in the early Drosophila embryo. He has focused on “zygotically” active genes because he believes the temporal and spatial pattern of their transcription may provide the triggers controlling the normal sequence of embryonic development. Kathryn Anderson from Sloan-Kettering Institute will focus her talk on the Sonic hedgehog (Shh) signal transduction pathway and how it is essential for the development and patterning of numerous organ systems, and has important roles in a variety of human cancers. Jonathan Epstein from the University of Pennsylvania will present “Molecular Mechanisms of Cardiac Development”. Dr. Epstein’s research has focused on the molecular mechanisms of cardiovascular development and implications for understanding and treating human disease.

Each year the Smithies Symposium hosts world-class researchers on topics relevant to modern genetics and biology. The symposium is possible due to a generous donation from Dr. Oliver Smithies, who won the 2007 Nobel Prize for his work on gene knockouts in mice, which was conducted while he was a UW Genetics Professor. Read more about it below. Also in this issue, we will highlight awards won by students and faculty and have included a feature article on the research of Professor Barry Ganetzky and Qiang Chang.

Our student programs are thriving as we continue to train the next generation of scientists. Currently we have 277 undergraduate genetics majors and 60 graduate students. Both the undergraduate and graduate curricula have been revised and upgraded with new course offerings. In the pages that follow you find more details on these and other items of interest.
Qiang Chang: Understanding Rett syndrome

Rett syndrome (RTT) is an autism spectrum developmental disorder with an estimated prevalence of 1 in 10,000-15,000 girls. Although mutations in the X-linked MECP2 (methyl-CpG binding protein 2) gene have been identified as the cause of classic RTT cases, the underlying molecular mechanism of RTT pathogenesis remains elusive. Consequently, there is no cure or effective treatment for RTT. MeCP2 is capable of binding to methylated DNA to repress or activate expression of neighboring genes. Our long-term goal is to understand the central role MeCP2 in DNA methylation-dependent epigenetic regulation of the brain function during mammalian development and disease. Current projects in my laboratory include: 1) using unique mouse genetic tools for genome-wide location analysis to identify MeCP2 target genes in an unbiased fashion; 2) generating and characterizing phospho-mutant MeCP2 mice to study the critical role of MeCP2 phosphorylation in dynamically regulating activity-dependent gene expression, neuronal development, microcircuit maturation and animal behavior; 3) administering novel small molecules that have been shown to specifically activate TrkB (receptor of BDNF [brain-derived neurotrophic factor]) in MeCP2 mutant mice to evaluate the therapeutic potential of BDNF or activation of BDNF signaling through TrkB; 4) establishing a cell culture based human model of RTT by generating patient-specific induced pluripotent stem cells (iPSCs) and differentiating the iPSCs into neurons and astrocytes for phenotypic characterization, drug screening and toxicology testing.

Together, these projects will identify MeCP2 target genes and help us to understand how MeCP2 directly modulates the expression of its target. At the same time, this work not only demonstrates that basic knowledge of the disease mechanism (based on one identified target gene) can be translated into therapeutic strategies, but also provides a general framework of translational research in the specific context of Rett syndrome. Once more target genes are identified and the mechanism by which MeCP2 modulates the expression of these genes is understood, more potential therapies can be designed and tested. Our work using patient-specific iPSCs brings us one step closer to translate our bench-side findings to potential bedside treatments for RTT. As both the natural extension and the ultimate test of the mouse studies, it provides human relevance to our work.
Many thanks to our corporate partners for providing funds for this newsletter.

Donations make a difference: Support UW Genetics Today

Every contribution, no matter the amount, supports our mission to foster Research, Education, and Public Outreach related to genetics.

Research: UW-Genetics supports world-class research in agricultural and plant genetics, human and animal development, and basic biology. Funds support our research infrastructure, supplies, and discretionary funds related to research.

Education: UW is a leader in training the next generation of geneticists. With close to 300 undergraduate majors and 60 graduate students, financial gifts support student stipends, travel awards, and student-run organizations.

Outreach: Genetics faculty and students are active in community outreach, participating in UW Science Festivals, our own Darwin Day, ‘Wednesday Nights at the Lab’, and local elementary outreach. Donations cover supplies and handout materials for hands-on science activities. Your donation matters. Visit our website www.genetics.wisc.edu or contact us at fund@genetics.wisc.edu to make your contribution today.

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