# ~ Biography ~

## David Allis

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**C. David Allis** is the Joy and Jack Fishman Professor and Head of the Laboratory of Chromatin Biology at The Rockefeller University in New York City. His laboratory focuses on the DNA-histone protein complex, chromatin, which is part of a sophisticated system that allows for extremely selective gene activation (or inactivation) in a given cell. His team investigates chromatin signaling via histone modifications such as acetylation, methylation, and phosphorylation; these modifications may act together to form a 'histone code' that, in turn, dictates downstream biological events. Their studies suggest that these and other chromatin-modifying activities are centrally connected to the control of both normal cellular proliferation and differentiation as well as abnormal events leading to transformation and tumorigenesis. Dr. Allis received his B.S. from the University of Cincinnati and his Ph.D. from Indiana University. A member of the American Academy of Arts and Sciences since 2001, Dr. Allis is a past recipient of the DeWitt Stetten Jr. Award (2001), The Dickson Prize in Medicine (2002), the Massry Prize (2003), and is the 2004 recipient of the Wiley Prize. He was elected to the National Academy of Sciences in 2005 and is a 2007 recipient of the Gairdner International Award.

# ~ Lectures ~

**Tuesday, August 28**
**4:00 - 5:00**
**Deans Auditorium (PFEN)**

*Beyond the Double Helix: Reading and Writing the "Histone Code"*

**Wednesday, August 29**
**4:00 - 5:00**
**Deans Auditorium (PFEN)**

*Translating the Histone Code: A Tale of Tails*

The human genome is estimated to contain 30,000 – 40,000 unique genes; the DNA sequence and chromosomal location of all these genes are becoming widely known. A central challenge facing the biomedical community is how to derive medically-valuable knowledge about the function of these genes from the now-available DNA sequence data. Though every gene exists within every cell in the human body, only a small percentage of genes are activated in any given cell. To manage this genetic information efficiently, nature has evolved a sophisticated system that facilitates access to specific genes. This system relies on a DNA-histone protein complex called chromatin to efficiently package the genetic information that exists within each cell. This packaging system makes certain genes more readily accessible to transcription factors and other machinery that must engage our genetic template. Chromatin modifications, and the regulation of the enzymes responsible for adding or subtracting them, are poised to take center stage in the study of cancer in the current post-genomic or epigenomic era. Moreover, the implications of chromatin and its modification are beginning to gain appreciation in clinical oncology. The identification of altered DNA methylation and histone acetylase activity in a range of human cancers, coupled with the use of HDAC inhibitors in the treatment of leukemia, make a compelling argument. It is clear that the regulatory signals provided by chromatin modifications will revolutionize our view of cancer as new models of "epigenetic carcinogenesis" are advanced. We favor the view that there exists an epigenetic indexing system for our genome, or a "histone or epigenetic code," that represents a fundamental regulatory mechanism that acts outside of the DNA itself. We predict that this "code" impacts on most, if not all, chromatin-templated processes with far-reaching consequences for cell fate decisions and for normal and pathological development.

Most of our current research is centered on chromatin and its regulation though post-translational modification of histone (and non-histone) proteins. That said, other mechanisms such as the existence of DNA methylation and of small non-coding RNAs are also likely, if not certainly, involved. How epigenetic states, "ON" or "OFF", are inherited from one generation to the next is a central question that we, and others, are currently addressing.