

Novel epigenetic and epitranscriptomic in cancer and adventures in personalized medicine

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New developments in DNA sequencing have shown that dozens of DNA modifications exist to define the epigenome. Moreover, recent work has shown RNA modifications like m6A, collectively called the “epitranscriptome,” are a pervasive feature of mammalian cells and likely play a role in development and disease. An enrichment of m6A near the last CDS of thousands of genes has implicated m6A in transcript processing, translational regulation, and potentially a mechanism for regulating miRNA maturation. Also, because the sites of m6A show strong evolutionary conservation and have been replicated in nearly identical sites between mouse and human, strong evolutionary pressures are likely being maintained for this mark. We leverage these and other tools to study cancer genomes, providing comprehensive profiles of somatic and epigenomic mutations in cancer, since somatic mutations alone do not fully explain cancers. We find that additional epigenetic and epitranscriptomic alterations can play a central role in cancer pathogenesis, and that somatic mutations contribute through systematic alterations in DNA methylation and chromatin state. We also describe work on personalized medicine in cancer therapies.

Biography

Christopher E. Mason received his dual B.S. in Genetics and Biochemistry from University of Wisconsin-Madison in 2001, his Ph.D. in Genetics from Yale University in 2006, and spent three years as post-doc working at Yale Medical School. He is now an Assistant Professor of Computational Genomics at Weill Cornell Medical College of Cornell University in New York City, in the Department of Physiology and Biophysics and at the Institute for Computational Biomedicine. He also holds an appointment in the Tri-Institutional Program on Computational Biology and Medicine (Cornell, Memorial Sloan-Kettering and Rockefeller) and the Weill Cornell Cancer Center.

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