B·DEBATE International Center for Scientific Debate BARCELONA

CRG Centre for Genomic Regulation

ROBERT SCHNEIDER

CV

PARTICIPANT AT:

CODING AND NON-CODING FUNCTIONS OF THE GENOME
BARCELONA CONFERENCE ON EPIGENETICS AND CANCER

October, 29th-30th, 2015, Barcelona

Robert Schneider, Director of Recherche, Institute of Genetics and Molecular and Cellular Biology (IGBMC), Strasbourg France

Robert Schneider did his PhD at the University of Munich in close collaboration with the LMB in Cambridge, UK. For a PostDoc he decided to move to Cambridge to join the group of Tony Kouzarides at the Gurdon Institute where he focused on methylation of lysines in the histone H3 tail. From there he went to the Max Planck Institute for Immunobiology and Epigenetics in Freiburg to start his own independent group there and extended his research towards novel sites and types of histone modifica tions. Since 2012 he is senior group leader at the Institute of Genetics and Molecular and Cellular Biology (IGBMC) in Strasbourg, France and apart from continuing his work on new histone modifications, he became excited about single cell epigenetics. Robert was holder of an ERC starting grant and the Chaire Gutenberg. Amongst other awards he has received an HFSP Career Development Award and is member of Epigenesys. He has authored more than 60 publications. Furthermore Robert is founder of the TriRhena Chromatin Club and organizer of the Abcam “Chromatin and Epigenetics” meetings in 2012 and 2014.

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ABSTRACT

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Novel Players in Chromatin

One of the major goals of post-genomic biological research is to understand the molecular basis and physiological role of covalent protein modifications. These post-translational modifications (PTMs) can regulate protein interactions and thus trigger particular downstream responses. It has been suggested that PTMs of histones constitute a so-called "histone code" defining distinct chromatin or “epigenetic” states. Nonetheless the set of characterised histone modifications is far from complete and many modifications are awaiting identification. How mechanistically chromatin and “epigenetic” states are inherited through cellular divisions is currently only poorly understood. This inheritance of epigenetic states offers an important memory mechanism. However to define the heritability of epigenetic states within a population of cells is difficult due to cell heterogeneity, combined with varying levels of stability of these states. We are just beginning to understand how chromatin states (and chromatin modulators) can mediate “epigenetic” memory and the inheritance of these states on individual cell level. One of the key questions in the field is if histone PTMs can be causative for processes like transcription or are just by-products, with limited functional relevance. We recently demonstrated a causative function for a novel lysine acetylation on the lateral surface of the histone octamer. We found that acetylations within the core of the nucleosome at positions that are in contact with the DNA are sufficient to stimulate transcription by modulating histone-DNA binding. Our model is that nucleosome function and signaling to the epigenome is directly regulated by specific lateral surface modifications. Furthermore, we identified additional novel PTMs that act as guardian of genome stability by regulating the activity of transposable elements.

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