
SANDRA B. HAKE

CV

PARTICIPANT AT:

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

**October, 29th-30th, 2015, Barcelona**

Sandra B. Hake, Independent Group Leader, BioMedical Center, Department of Molecular Biology at the LMU Munich, Germany

Background in Biology and PhD in Molecular Immunology from the Max-Planck-Institute in Freiburg, Germany. Postdoctoral studies at the Memorial Sloan-Kettering Cancer Center (Prof. Lisa Denzin) and the Rockefeller University (Prof. C. David Allis). Her main research focuses on chromatin functions with particular emphasis on the role of mammalian histone variants in gene regulation and cell cycle control. She currently applies a system-wide approach to functionally characterize the interactomes of human histone variants by integrating quantitative proteome, transcriptome and genome data. She is vice-speaker of the collaborative research center 1064 and member of the international Max-Planck graduate school IMPRS-LS and an associated member of CIPSM.

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SANDRA B. HAKE

ABSTRACT

PARTICIPANT AT:

**CODING AND NON-CODING
FUNCTIONS OF THE GENOME**
BARCELONA CONFERENCE ON
EPIGENETICS AND CANCER**October, 6th-7th, 2015, Barcelona**

Sandra B. Hake, Independent Group Leader, BioMedical Center, Department of Molecular Biology at the LMU Munich, Germany

Multivalent binding of PWWP2A to H2A.Z chromatin regulates chromosome segregation and organ development

The essential histone variant H2A.Z affects various DNA-based biological processes by so far not well understood mechanisms. Using a comprehensive label-free quantitative mass spectrometry-based approach we identified the human H2A.Z nucleosome interactome providing further insights into H2A.Z's regulatory functions. Besides histone writer, eraser and reader complexes we identified PWWP2A as a novel H2A.Z-nucleosome binder. PWWP2A binds unprecedented strong to chromatin preferable at the -1 H2A.Z-nucleosome of transcribed genes through a multivalent chromatin-binding mode. Two internal protein regions separate H2A.Z-specificity and nucleosome interaction, whereas the PWWP domain mediates DNA and H3 tail binding. Cellular depletion of PWWP2A results in impaired cell proliferation caused by a block in metaphase-anaphase progression. Accordingly, knockdown of frog PWWP2A leads to severe developmental defects. Together, this study identifies PWWP2A as an H2A.Z-specific multivalent chromatin binder and provides a novel link between H2A.Z, chromosome segregation and organ development.

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