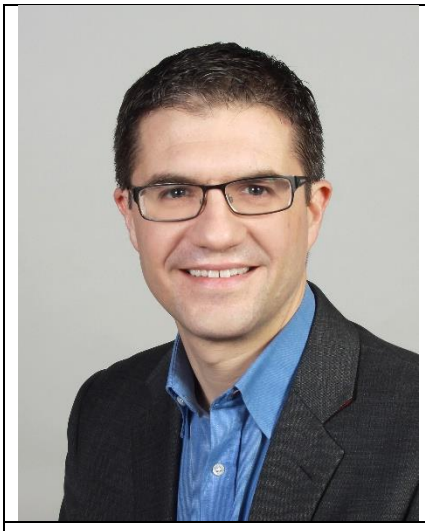

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Biography

Beat Fierz studied at the University of Basel and obtained a Diploma in Biophysical Chemistry in 2002. Subsequently, he performed his PhD studies on the dynamics of synthetic peptides in the laboratory of Prof. Thomas Kiefhaber at the Biozentrum (Univ. Basel). In 2007 he joined the laboratory of Prof. Tom W Muir at the Rockefeller University and Princeton University, using chemical biology to investigate chromatin regulation. In 2012, he set up his independent group as a tenure-track assistant professor at the institute of Chemistry and Chemical Enigneering (ISIC) at EPFL in Switzerland, where he established a program in chemical biology and biophysics to investigate chromatin function on the single-molecule scale. In 2019, he was promoted to associate Professor at EPFL.

Abstract title: Synthetic and single-molecule exploration of the dynamic chromatin landscape

The organization of the eukaryotic genome into chromatin is integral to genome regulation. Chromatin structure and dynamics, modulated by histone post-translational modifications (PTMs) as well as architectural proteins, dictate DNA access for transcription factors and the gene expression machinery. Due to the fundamental role of chromatin, a molecular and dynamic understanding of this nucleoprotein complex is required.
We dissect chromatin signaling on the single-molecule scale, combining chemical biology approaches and mechanistic biophysics. In particular, we have developed single-molecule fluorescence approaches to directly observe chromatin dynamics as well as to monitor protein interaction dynamics with modified chromatin fibers in real-time. Together with chemical approaches to reconstitute differentially modified chromatin, these methods allowed us to reveal fundamental mechanisms in gene repression by the polycomb machinery, and gene activation by pioneer transcription factors. Together, our results provide a mechanistic view of how chromatin structure and dynamics are regulated by chromatin PTMs and protein effectors to establish repressive or active architectures, thereby controlling gene expression.