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Prof. Eva Nogales

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Eva Nogales received her bachelor's degree in physics from the Universidad Autónoma de Madrid and her doctorate in biophysics from the University of Keele, UK. For her postdoctoral work at the Lawrence Berkeley National Laboratory (LBNL), she worked with Ken Downing on the structure determination of tubulin by electron crystallography. She joined the University of California, Berkeley in 1998, where she is now a Distinguished Professor of Molecular and Cell Biology. She has been a Howard Hughes Medical Institute Investigator since 2000 and she is also a Senior Faculty Scientist at Lawrence Berkeley National Laboratory (LBNL). In 2020 she served as President of ASCB.

Eva has received the Burton medal of the Microscopy Society of America, the Grimwade medal from the University of Melbourne, the Biophysical Lectureship from the Biophysical Society, the Dorothy Crowfoot Hodgkin Award from the Protein Society, the Mildred Cohn Award from the American Society of Biochemistry and Molecular Biology, the Keith R Porter Lecture Award from the American Society for Cell Biology (ASCB), and the LBNL Director's Award for Exceptional Science Achievement. She is a Fellow of ASCB and the Biophysical Society, a member of the National Academy of Sciences of the USA and the American Academy of Arts & Sciences, and foreign associate member of EMBO and the Real Academia de Ciencias de España.

The work in Eva's lab is dedicated to the mechanistic understanding of large macromolecular assemblies through the visualization, using cryo-electron microscopy, of their structure, dynamics and regulatory interactions.

Structural insights into the regulation of the gene silencer PRC2

Polycomb repressive complex 2 (PRC2) is an epigenetic regulator that modifies histone H3 by trimethylation of lysine 27 (H3K27me3). Spreading of the H3K27me3 mark ultimately results in chromatin compaction and leads to the repression of the underlying genes. PRC2 activity is essential during embryonic development and to maintain cell identity in adulthood, with mutations and/or misregulation of PRC2 leading to cancer. We have studied the molecular principles underlying regulation of the core PRC2 by cofactors and by histone modifications. Recently, it has been shown that PRC2 undergoes auto-methylation within its catalytic SET domain, which results in stimulation of its function by an unknown mechanism. Using cryo-electron microscopy, optimized for fragile complexes, we have been able to obtain structural information that leads to a novel mechanism of autoregulation by an epigenetic factor.