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In their own words: Interviews with Cell Cycle

Cdc14p regulates condensin binding to rDNA

Dr. Alexander Strunnikov on his highly cited paper published in *Cell Cycle*

Wang BD, Yong-Gonzalez V, Strunnikov AV. Cell Cycle. 2004; 3:960-7.

Dr. Strunnikov was educated in Russia and received his bachelor degree in Biology and Genetics from St. Petersburg University in 1984 and Ph.D. in Biological Sciences with a specialization in cell biology from the Institute of Cytology of the Russian Academy of Sciences. His current work focuses on chromosome stability mechanisms, the regulation and structure of mitotic chromatin, as well as genomic and proteomic aspects of mitotic chromosome organization.

CC: Did you expect your paper to become highly cited, or is this surprising to you?

AS: I did expect it to happen. The work was really original and at the same time rather conclusive, which always sparks good following. I was surprised, though, that it got featured in *Nature Reviews* immediately following publication in *Cell Cycle*.

CC: Would you give us a brief history of the paper?

AS: The core observation of this work was made back in 2000, when my lab, upon discovering re-localization of condensin to the nucleolus in mitosis, began screening a large collection of conditional mutants for ones that interfere with this property of condensin. At that time there was no comprehensive assay for condensin function in vivo (which is still the state of affairs now). Therefore, the changed condensin localization in the course of cell cycle, used as an indirect assay for condensin activity in living cell, has given a tremendous boost to our efforts to elucidate both the biological function of chromosome condensation and its regulation. Mutations in the *CDC14* gene were the most prominent hit in this screening. These results were included in my talk at the FASEB meeting the same year, but I received virtually no feedback from the expert audience, as it was apparently difficult to comprehend that such a drastic difference (with regard to condensin loading) would exist between cdc14 and cdc5 mutants on one hand (disrupted condensin loading) versus cdc15 and lte1 mutant on the other (normal condensin loading). All these genes were regarded as working in the same pathway (mitotic exit network) at the time. However, upon formulation of the FEAR concept (fourteen early anaphase release) by A. Amon lab in 2002–2003, it became evident to me that our discovery was just ahead of its time. We quickly relaunched work on this project and found that it is the FEAR pathway that is crucial for the facilitation of condensin loading to rDNA.

CC: What has been its impact on the field?

AS: I believe it had the biggest impact with respect to revitalizing interest in the nucleolus and rDNA, which was somewhat fading at the time. It also has led to several breakthroughs in our understanding of nucleolar biology and to the formation of new concepts: for example, one of competition between transcription and chromosome segregation. Furthermore, as we see now, the nucleolus puzzle is yet to be solved, with its many biological functions remaining uncharacterized. One prominent example is the recent discovery of the CDC14B and nucleolar involvement in DNA damage response in human cells (Bassermann et al., *Cell* 2008).

CC: What are major discoveries in the field since your publication?

AS: There were many exciting developments in the field in the past five years, both with respect to cell cycle regulation mediated by the nucleolar organizer as well as regarding functional and structural organization of the nucleolus itself. It would take sizable space to describe them all. Among the most prominent ones in the cell cycle aspects of nucleolar function - the already mentioned breakthrough publication on CDC14B. The field of SUMO-mediated regulation of cell cyle acquired a very interesting nucleolar angle, upon discovery of a functional interface between the polysumoylation turnover and the nucleolus. Regarding the organization of rDNA, I was most impressed by the 2005 work from Kobayashi lab on the control of rDNA amplification by specialized transcription (Kobayashi and Ganley, 2005). Also, our own work (as well as the Amon and Aragon labs) contributed greatly to solving the paradox of functional compartmentalization between continuing RNA PolI transcription and sister chromatid separation in yeast mitosis. This extensive work has led to a compelling model, which we proposed for the functional role of silent rDNA repeats as "segregation domains" of actively transcribed nucleoli (Wang et al., 2006).

Dr. Strunnikov's highly cited paper can be found at:
http://www.landesbioscience.com/journals/6/article/1003/

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