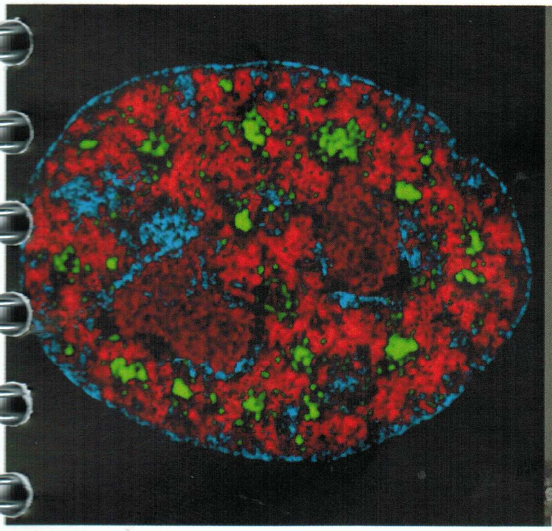
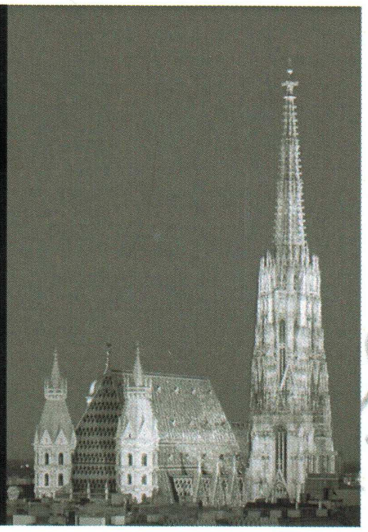
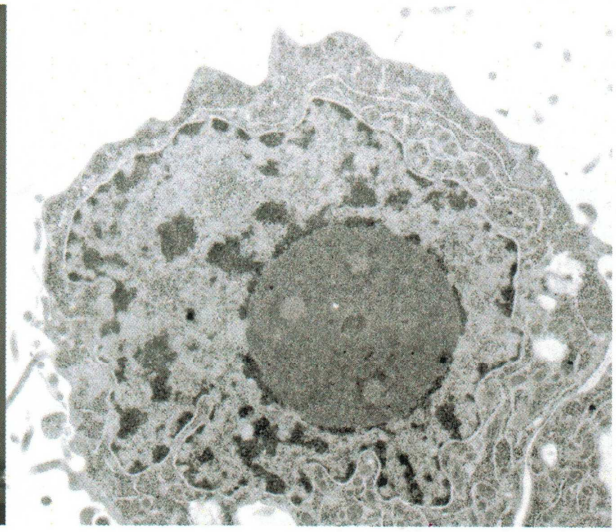
24th Wilhelm Bernhard Workshop on the Cell Nucleus







57th Symposium of the Society for Histochemistry

SFH

Vienna, Austria
Aug. 17th – Aug. 22nd 2015

13.00-14.00	Lunch
	Workshop III: Nuclear structure – nucleolus (chair: Marco Biggiogera)
14.00 - 14.30	*Evgeny Smirnov (Czech Republic):* FC/DFC units of nucleoli and rDNA replication
14.30 - 15.00	*Marion Schmidt-Zachmann (Germany):* Nucleolar dioxygenases NO52 and NO66
15.00 - 15.30	*Patrick DiMario (USA):* Complete loss of Nopp140 disrupts nuclear and ribosomal homeostasis in Drosophila melanogaster
15.30 – 16.00	Coffee break / poster viewing
16.00 - 17.00	*Meeting of the WBW International Committee*
18.30	Dinner

Wednesday, August 19th

	Workshop IV: Nuclear structure – nuclear lamina (chair: Michael Jantsch)
08.30 - 09.00	*Sandra Vidak (Austria):* Proliferation of progeria cells is enhanced by lamina-associated polypeptide (LAP) 2 alpha through expression of extracellular matrix proteins
09.00 - 09.30	*Oxana Zhironkina (Russia):* Mechanisms of nuclear lamina growth in interphase
09.30 - 10.30	***Wilhelm Bernhard Junior Lecturer - EMBO Young Investigator Lecture*** *Evi Soutoglou (France):* Spatial organization of DNA repair within the nucleus
10.30 - 11.00	Coffee break
	Workshop V: Nuclear function - replication / DNA-repair (chair: Marion Schmidt-Zachmann)
11.00 - 11.30	*Chikashi Obuse (Japan):* DNA repair pathway is controlled by differential phosphorylation of 53BP1
11.30 - 12.00	*Eva Bartova (Czech Republic):* HP1 protein and DNA repair processes
12.00 - 12.30	*Jurek Dobrucki (Poland):* Two stages of XRCC1 recruitment in response to sublethal local DNA damage induced by focused visible light
13.00 - 14.00	Lunch

DNA repair pathway is controlled by differential phosphorylation of 53BP1

S.-Y. Isobe, K. Nagao[1], Y.-N. Ohkubo[1], H. Kimura[2], C. Obuse[1]
[1]Graduate School of Life Science, Hokkaido University, Sapporo, JPN
[2]Graduate School of Bioscience and Biotechnology Tokyo Institute of Technology, Yokohama, JPN

DNA double-strand breaks (DSBs) are repaired by either of two pathways, homology-directed repair (HDR) or non-homologous end-joining (NHEJ). Rif1 (RAP1 interacting factor homolog) was recently shown to stimulate NHEJ choice through an interaction with 53BP1 (p53-binding protein 1), but the molecular mechanism underlying pathway choice remains largely unknown. Here we show that SCAI (suppressor of cancer cell invasion) also binds to 53BP1 and facilitates HDR, counteracting Rif1. Depletion of SCAI diminished HDR and the recruitment to damage sites of BRCA1 (breast cancer susceptibility gene 1) and factors for initiation of HDR. Interaction between SCAI and 53BP1 was stimulated by phosphorylation at S/TP sites in 53BP1. Dephosphorylation of 53BP1 by PP1 (protein phosphatase 1), which binds to Rif1, appears to suppress SCAI binding and HDR initiation factors. SCAI may inhibit Rif1-PP1 and thereby facilitate the HDR. Thus, SCAI and Rif1 have opposite roles in the choice of repair pathways by regulating phosphatase activity on 53BP1 at DNA damaged sites.

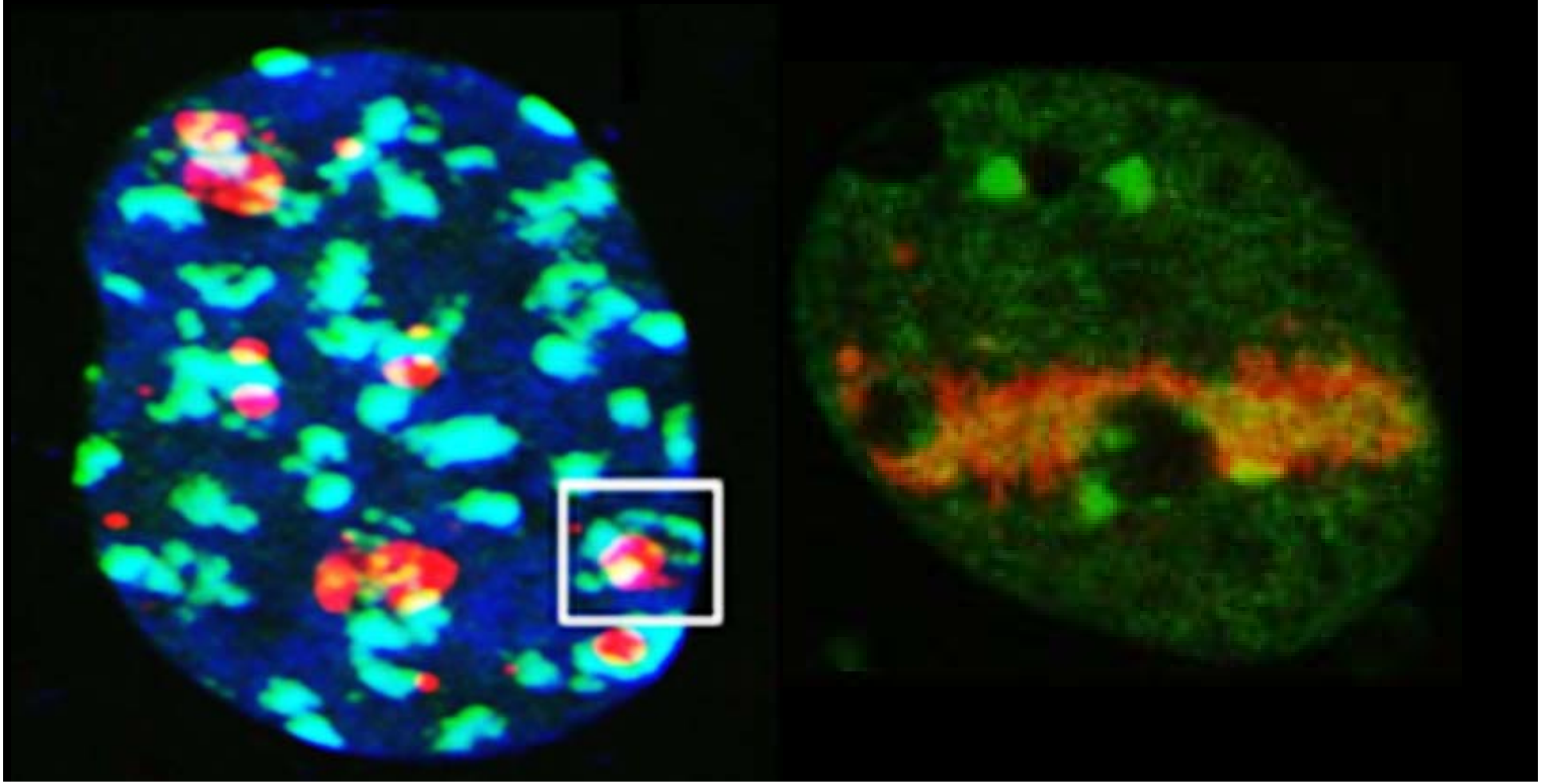
HP1 protein and DNA repair processes

E. Bartova[1], S. Legartova[1], L. Stixova[1], P. Sehnalova[1], J. Suchankova[1]
[1]Institute of Biophysics, Academy of sciences of the Czech Republic, v.v.i., Brno, CZE

We analyzed the DNA damage response after ultraviolet (UVA) and γ-irradiation of human and mouse cells. We focused on HP1 beta protein, which plays an important role in regulation of gene expression and processes of heterochromatinization. After local micro-irradiation by UVA lasers we confirmed that HP1 beta accumulates at local induced DNA lesions, which is ATP-dependent. Moreover, HP1 beta appeared at DNA lesions simultaneously with protein of the nucleolus, UBF1. By FRET technique we observed an interaction between HP1 beta and UBF1 at both nuclear and nucleolar DNA lesions. This was mediated by chromo shadow domain (CSD) of HP1 beta and it was found in parallel with an appearance of cyclobutane pyrimidine dimers (CPDs). Accumulation of HP1 beta at UVA-induced DNA lesions was abrogated by inhibition of histone deacetylases (HDACs). In non-treated cells, H3K9 deacetylation was found at UVA-damaged chromatin, but status of phosphorylation of histone H2AX was not changed. However, injury of A-type lamins at nuclear periphery by UVA laser did not affect a pronounced recruitment of HP1 beta to DNA lesions. Only 53BP1 pattern at DNA lesions was changed by damage in nuclear lamina.

HP1β protein and DNA repair processes

Eva Bártová
Institute of Biophysics Academy of Sciences of the Czech Republic
Brno



GA ČR

THE ACADEMY OF SCIENCES OF THE CZECH REPUBLIC

Andrea Horáková, Gabriela Šustáčková, Lenka Stixová, Sona Legartová, Petra Sehnalová, Jana Suchánková