of skin is removed from the dorsal area of a SCID mouse to engraft a skin equivalent set up with tumour or normal keratinocytes. To date, we have obtained fast-growing and massively invasive tumours with one cSCC keratinocyte population. We are currently using this model to test whether the specific PLK1 inhibitors identified *in vitro* are efficacious with topical application *in vivo*.

094
Ultraviolet B-dependent changes in the expression of fast-responding early genes is modulated by huCOP1 in keratinocytes

B. Fazekas,[1] H. Polyánka,[2] A. Bebes,[1] G. Tax,[1] K. Szabó,[2] K. Farkas,[2] Á. Kinyó,[1] F. Nagy,[3] L. Kemény,[1] M. Széll[2] and É. Ádám[3]

[1]*Department of Dermatology and Allergology, Faculty of Medicine, University of Szeged, Szeged, Hungary,* [2]*MTA-SZTE Dermatological Research Group, University of Szeged, Szeged, Hungary and* [3]*Institute of Plant Biology, Biological Research Centre of the Hungarian Academy of Sciences, Szeged, Hungary*

Ultraviolet (UV) B is the most prominent physical carcinogen in the environment leading to the development of various skin cancers. We have previously demonstrated that the human orthologue of the *Arabidopsis thaliana* constitutive photomorphogenesis (COP)1 protein, huCOP1, is expressed in keratinocytes in a UVB-regulated manner and is a negative regulator of p53 as a post-translational modifier. However, it was not known whether huCOP1 plays a role in mediating the UVB-induced early transcriptional responses of human keratinocytes. In this study we found that stable small-interfering (si) RNA-mediated silencing of huCOP1 affects the UVB response of several genes within 2 h of irradiation indicating that altered huCOP1 expression sensitizes the cells toward UVB. Pathway analysis identified a molecular network in which 13 out of the 30 examined UVB-regulated genes were organized around three central proteins. As the expression of the investigated genes was upregulated by UVB in the siCOP1 cell line, we hypothesize that huCOP1 is a repressor of the identified pathway. Several members of the network have been implicated previously in the pathogenesis of nonmelanoma skin cancers; therefore, clarifying the role of huCOP1 in these skin diseases may have clinical relevance in the future.

095
Contribution of collagen XIII and XXIII to squamous cell carcinoma development

S. Santoleri,[1] G.M. Nieto,[1] A. Heikkinen,[1] R. Heljasvaara,[1] A.-M. Auvinen,[1] T. Pihlajaniemi[1] and M. Koch[2]

[1]*Oulu Center for Cell-Matrix Research, Biocenter Oulu, Faculty of Biochemistry and Molecular Medicine, University of Oulu, Oulu, Finland and* [2]*University of Cologne, Cologne, Germany*

Collagens are important not only as structural components but also by regulating cell behaviour. Our work addresses the roles of the structurally homologous collagens XIII and XXIII in skin tumour formation. Both collagens are transmembrane proteins, which may also occur as shed protein at cell–extracellular matrix interphases (Heikkinen A, Tu H, Pihlajaniemi T. Collagen XIII: a type II transmembrane protein with relevance to musculoskeletal tissues, microvessels and inflammation. *Int J Biochem Cell Biol* 2012; **44**: 714–17). Their expression is induced in malignant transformations and they have been suggested to play a role in tumour progression and metastasis (Väisänen T, Väisänen M-R, Autio-Harmainen H, Pihlajaniemi T. Type XIII collagen expression is induced during malignant transformation in various epithelial and mesenchymal tumours. *J Pathol* 2005; **207**: 324–35; Banyard J, Bao L, Hofer MD, Zurakowski D *et al*. Collagen XXIII expression is associated with prostate cancer recurrence and distant metastases. *Clin Cancer Res* 2007; **13**: 2634–42; Spivey KA, Banyard J, Solis LM *et al*. Collagen XXIII: a potential biomarker for the detection of primary and recurrent non-small cell lung cancer. *Cancer Epidemiol Biomarkers Prev* 2010; **19**: 1362–72; Spivey KA, Chung I, Banyard J *et al*. A role for collagen XXIII in cancer cell adhesion, anchorage-independence and metastasis. *Oncogene* 2012; **31**: 2362–72). We have recently generated collagen XIII knockout mice (Latvanlehto A, Fox MA, Sormunen R *et al*. Muscle-derived collagen XIII regulates maturation of the skeletal neuromuscular junction. *J Neurosci* 2010; **30**: 12230–41) and a collagen XXIII knock-in mouse line with markedly reduced expression (unpublished). Both collagens are expressed in normal skin, and to study their roles during malignant processes we have used a classical, chemically induced cutaneous squamous cell carcinoma (SCC) model of mouse skin carcinogenesis in single mutant mice as well as double mutants for these alleles. All mice developed tumours but the number of tumours was significantly reduced in collagen XXIII deficiency, while lack of collagen XIII resulted in more aggressive malignant conversion in short term. This project will lead to the identification of pathways in skin carcinogenesis regulated by collagens XIII and XXIII together and separately, and the findings may lead to the development of novel diagnostic tools and therapeutic targets.

096
Preliminary results of the examination of gene abnormal methylation in T-cell cutaneous lymphoma and parapsoriasis

N. Potekaev,[1] D. Zaletaev,[2] I. Khamaganova,[3] V. Lysenko[1] and O. Shvec[1]

[1]*Moscow Scientific and Practical Centre for Dermatology and Cosmetology, Moscow, Russia,* [2]*Research Centre of Medical Genetics, Moscow, Russia and* [3]*Department of Skin Diseases and Cosmetology, Russian National Research Medical University named after N.I. Pirogov, Moscow, Russia*

The epigenetic regulation of gene activity, which is based on abnormal methylation/demethylation, may be essential in cancerogenesis. The imbalance of methylation was shown in neoplastic cells. Abnormal methylation of the promoter CpG islands takes place simultaneously with genome hypomethylation. The result is inactivation of the gene. The same type of methylation is described in genes that take part in apoptosis, angiogenesis, signal transmission, DNA reparation, metastatic