UniversitätsTumorZentrum (UTC) Jena
Leibniz Institute for Age Research
(Fritz Lipmann Institute)
Leibniz Institute for Natural Products and
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Jena Graduate Academy

# 4th POSTGRADUATE SYMPOSIUM
## on
## Cancer Research

Saturday, April 28, 2012
Altes Schloss Dornburg (Kaisersaal)

# 4th Postgraduate Symposium on Cancer Research

On Saturday, April 28th, 2012 in Altes Dornburger Schloss
Start: 9 a.m.

**Abstract deadline**: February 20th, 2012
Later abstracts are also welcome but may not necessarily be included in the program.

**Abstract submission**:
Please submit your abstract in English by using MS-Word format including: Title, author(s), affiliation(s), to the following email address: izkfonkologie@med.uni-jena.de

**Abstract content:**

- Abstracts should be concise and informative, and should be structured under following headings: Aims (background information), Methods, Results and Conclusions.
- The first author should usually be a postgraduate student (and the last is her/his mentor), who will give the presentation.
- The title should be concise and indicate the content of the abstract and not exceed 10 words. Abstract text should not exceed 250 words.
- Use standard abbreviations. Place special or unusual abbreviation in parenthesis after the full term the first time it appears.

**Language**: The presentation should be given in English. These are 2 types of presentation: oral or poster. You may indicate on submission which type of presentation you would prefer. The final decision on the presentation type will be made by the organizing committee. Each presentation has roughly 15 minutes (10 min presentation, 5 min discussion). Contributor will be informed on the type of her/his presentation in advance.

**The program of the symposium will be distributed in April 2012.**

**Registrations** to the symposium should also be done by emailing to the above mentioned email address (please indicate name and address). Because symposium room has a limited capacity for 100 people, contributors have the highest preference for the registration. Remaining registration will be accepted on a "first-come, first-served (FCFS)" basis under the consideration of the room capacity.

There is no registration fee.

**Abstract example:**

**A p53 target gene DSC3 suppresses tumor growth and is frequently silenced by hypermethylation in human lung cancer**

T. Cui, Y. Chen, L. Yang, T. Knösel, K. Zöller, and I. Petersen
Institute of Pathology, University Hospital Jena

**Aims:** To analyse the DSC3 expression; to explore the mechanism for downregulation of DSC3, to investigate the regulation of DSC3 expression, and to analyze the functional role of DSC3 in lung cancer.

**Methods:** Expression of DSC3 was analyzed by real time RT-PCR and western blotting in lung cancer cell lines and normal bronchial epithelial cells (HBEC). In primary lung tumors, the protein expression of DSC3 was evaluated by IHC on TMA. Methylation status of DSC3 was examined by demethylation tests, MSP, and BS. The regulatory role of p53 on DSC3 was investigated by transfection. For the functional analysis, an expression vector containing the full-length cDNA of DSC3 was constructed and stably transfected into two lung cancer cell lines.

**Results:** In a majority of lung cancer cell lines, mRNA expression of DSC3 was downregulated. Expression of DSC3 was restored in 4 out 9 of lung cancer cell lines by 5'-aza-2'-DC. BS and MSP showed DNA methylation of DSC3 in the regions of promoter and exon 1. In primary lung tumors, DNA methylation of DSC3 was significantly associated with poor prognosis (P = 0.049). Transfection with p53-expression vector resulted in an increased expression of DSC3 in H2170 (DSC3 unmethylated) but not in H1299 (DSC3 methylated). However, combination of transfection with 5-aza-2-DC treatment led to increased expression of DSC3 in H1299. Additionally, table transfection of DSC3 into H2170 resulted in an increased gene expression and remarkably reduced the ability of colony formation in soft agar and the growth rate in vitro. Wound healing assay revealed that DSC3 positive transfectants exhibited decreased mobility compared to control cells.

**Conclusions:** Gene silencing of DSC3 could be explained by DNA hypermethylation. Methylation status of DSC3 predicts poor survival in patients with lung cancer. DSC3 is a potential tumor suppressor in lung cancer.

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Preferred presentation type: oral