LIVER TRANSPLANTATION MEETS CANCER

January 25-26, 2024

Symposium

ESSEN, GERMANY

An application has been made to the UEMS EACCME® for CME accreditation of this event.

PREFACE

Dear colleagues and friends,

I am pleased to extend a warm invitation to the Symposium “Liver Transplantation Meets Cancer,” organized by the Falk Foundation e.V. This symposium is scheduled to take place from January 25th to January 26th, 2024, in Essen, Germany.

In the ever-evolving landscape of hepatology and hepatobiliary surgery, this year’s symposium will focus on the intricate interplay between liver transplantation and cancer. We will explore the fields of Tumor Evolution, Tumor Host Interaction, cover the areas of Tumor Heterogeneity and complex diagnostics and will finally discuss current and novel questions in the field of Liver Transplantation for Extended Allocation.

Our goal is to present the latest breakthroughs and best practices that aim to strike the delicate balance between saving lives through transplantation and managing the complex issue of cancer recurrence in these patients.

In addition to the oral presentations, poster sessions will be scheduled on both days, providing a platform for comprehensive discussions and knowledge sharing.

Our symposium precedes the 40th Annual Meeting of the German Association for the Study of the Liver (GASL), to which I also extend a warm invitation.

I would like to express my heartfelt gratitude to all the speakers and participants for their contributions, insights, and the spirit of collaboration that they bring to this event. I wish to extend my special thanks to the Falk Foundation for their generous support and expert organization of this symposium.

I am confident that this symposium will be a source of inspiration, fostering discussions on the open questions within the interdisciplinary fields of hepatology and hepatobiliary surgery, encouraging the development of new projects and collaborations.

I eagerly anticipate welcoming you to our event at the “Haus der Technik”.

With warm regards, and looking forward to seeing you soon in Essen,

Ulf Neumann

LIVER TRANSPLANTATION MEETS CANCER

January 25–26, 2024

Scientific Organization:
Prof. Dr. Ulf Peter Neumann
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Scientific Co-Organization:
PD Dr. Dieter P. Hoyer, Essen

Congress Venue:
Haus der Technik
Hollestraße 1
45127 Essen
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For admission to scientific events your name badge should be clearly visible. Accompanying persons are not permitted during the conference at any time.

Start of Registration:
Thursday, January 25, 2024
12:00 - 18:00 h
at the congress office

Thursday, January 25, 2024

12:00	Registration and lunch with poster session
13:10	Welcome and opening remarks *Ulf Peter Neumann, Essen*

SESSION I

Tumor Evolution

Chairs: *Ali Canbay, Bochum; Thomas Seufferlein, Ulm*

13:20	Introduction to tumor evolution in the liver *Robert Schwabe, New York*
13:45	Cell death and development of inflammation and cancer in the liver *Tom Lüdde, Düsseldorf*
14:10	Commons and differences in tumor evolution of HCC & CCA *Peter Schirmacher, Heidelberg*
14:35	Mechanisms of MASLD as a leading etiology of HCC *Frank Tacke, Berlin*
15:00	Role of extracellular vesicles in cancer diagnosis and progression *Basant Thakur, Essen*
15:25	Summary
15:30	**Coffee break with poster session**

Thursday, January 25, 2024

SESSION II

Tumor Host Interaction

Chairs: *Jan Lerut, Brussels; Elke Roeb, Giessen*

16:10	Bile acids and hepatocarcinogenesis *Antonio Moschetta, Bari*
16:35	Significance of tumor-host interaction in liver cancer *Steven Olde-Damink, Maastricht*
17:00	Gut-Liver axis mediated mechanism for liver cancer *Oliver Pabst, Aachen*
17:25	Immunomodulation pre- and posttransplant *Christoph Roderburg, Düsseldorf*
17:50	Summary
18:00	**Networking and light refreshments**

Friday, January 26, 2024

8:30 Welcome
Ulf Peter Neumann, Essen

SESSION III

Tumor Heterogeneity & Diagnostics

Chairs: *Thomas Longerich, Heidelberg; Heiner Wedemeyer, Hannover*

8:40 Genomic analysis in the categorization of primary liver carcinomas (Next-Generation sequencing in liver carcinomas)
Charlotte Ng, Milan

9:00 Radiomics for early diagnosis and tumor characterization in primary liver cancer
René Hosch, Essen

9:20 Screening and histological diagnosing of CCA in PSC – challenges and developments
Christoph Schramm, Hamburg

9:40 MASH - interplay of anti-tumorigenic and pro-tumorigenic immune responses
Mathias Heikenwälder, Heidelberg

10:00 Summary

10:10 **Coffee break with poster session**

Friday, January 26, 2024

SESSION IV

Liver Transplantation for Extended Allocation

Chairs: *Felix Braun, Kiel; Martina Koch, Mainz*

10:40	Transplantation HCC out of Milan - molecular indications and contraindications? *Sandy Feng, San Francisco*
11:00	Liver transplantation for colon carcinoma metastasis – how far can we go? *Morten Hagness, Oslo*
11:20	Liver transplantation for CCC – Balancing on a razor's edge *Daniel Seehofer, Leipzig*
11:40	Robotic living related liver transplantation - donor and recipient *Dieter Broering, Riad*
12:00	Summary & Closing remarks *Ulf Peter Neumann, Essen*
12:15	**Lunch with poster session**
13:15	Opening of the annual meeting of the GASL

LIST OF SPEAKERS, MODERATORS AND SCIENTIFIC ORGANIZERS

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The event “Liver Transplantation Meets Cancer” is organized by the Falk Foundation e.V. The Foundation covers the costs for venue rental (EUR 3,900), catering (EUR 7,300), technical equipment (EUR 7,500), and accommodation of active participants (EUR 6,300). The travel expenses of active participants will be reimbursed after submission of a travel expense report.

REGISTRATION

You can register for the event via our homepage:
www.falkfoundation.org
Registration is only possible online.

CONGRESS FEES

Scientific Program of Symposium	EUR 150
Students (copy of student ID required)	EUR 75

The congress fees include:

- Refreshments during coffee breaks
- Lunch on Thursday, January 25 and Friday, January 26, 2024
- Snacks during scientific discussion on Thursday, January 25, 2024
- A copy of the final program

CONGRESS OFFICE AND REGISTRATION

Opening Hours:

Thursday, January 25, 2024	12:00 - 18:00 h
Friday, January 26, 2024	8:00 – 12:30 h

CONFLICT OF INTEREST

Members of the scientific committee declare the following potential conflicts of interest:

Ulf Peter Neumann: AstraZeneca, Falk Foundation, Merck, Roche
Dieter P. Hoyer: no potential conflict of interest to report

ARRIVAL

Haus der Technik (HDT)
Hollestraße 1
45127 Essen
Germany

Located conveniently in central Essen, directly opposite the central railway station, Haus der Technik is easily accessible by rail or road transport.

By car

From the motorway A3/A52 take the exit Essen-Zentrum/Essen-Süd; from the motorway A40, coming from the direction of Duisburg, take the exit Essen-Zentrum/Essen-Ost; and, from the motorway A40, coming from the direction of Dortmund, take the exit Essen-Huttrop.Since HDT has no car-parking facility of its own, please use the car-parks in the vicinity:

- Q Park, Gildehofstrasse 1, 45127 Essen, open 24 hours, 1,00 € per hour, 5,00 € Flat 08:00-20:00,
- 3,00 € Flat 10:00 – 20:00, 12,00 € maximum rate per day, 200 m / 3 minutes walking distance from HDT
- public parking at Hollestrasse 70, 45127 Essen, 5,00 € maximum rate per day (HDT-customer)

By plane

From Düsseldorf International Airport – Essen central railway station can be reached comfortably in about 25 minutes from Düsseldorf airport railway station, where about 300 trains (ICE, IC, EC and regional commuter trains) stop daily. At the airport take the SkyTrain or the shuttle bus to the airport station

By rail

Essen central railway station (Essen-Hauptbahnhof). The venue is a 2-minutes walk away.

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FULL CONTENT OF POSTER ABSTRACTS

Poster Numbers 1 – 14

1. A reliable guideline for evaluating NAFLD with HCC patients for the transplant centers

Metin Basaranoglu (Istanbul, TR)

Introduction: Non-alcoholic fatty liver disease (NAFLD) predisposes to cirrhosis and hepatocellular carcinoma (HCC). NAFLD-associated cirrhosis is predicted to rapidly become the leading indicator for liver transplant. The mortality rate of patients with NAFLD might differ from that in patients with virally caused cirrhosis.

Methods: We evaluated 258 cirrhotic patients with endoscopically defined high risk varices in this study. Each patient was evaluated for PVT, HCC, and mortality. Due to the etiology, patients were divided into 4 groups: Those with hepatitis B, hepatitis C, NAFLD and others related to autoimmune hepatitis, Wilson Disease, primary biliary cirrhosis, etc.

Results: HCC was detected in 14.7% of patients (38 out of 258 total study pool). The incidence rate of HCC was: 5.0% in patients with NAFLD, 26.7% in patients with hepatitis B, 34.5% in patients with hepatitis C, and 5.7% in other diseases ($p < 0.0001$). Of the 38 patients with HCC, 13% had PVT (Table 3). Moreover, HCC increased the mortality rate in almost all the groups. The mortality rate in hepatitis B group increased from 31% (17/55) in patients without HCC to 75% (15/20) in patients with HCC ($p = 0.001$). In the group with hepatitis C, the mortality rate increased from 32% (6/19) in patients without HCC to 90% (9/10) in patients with HCC ($p = 0.005$). The mortality rate in NAFLD patients increased from 47.5% during follow-up to 80% after HCC developed.

Discussion/Conclusion: Older patients were more prone to developing more cirrhosis, HCC and high mortality rates. However, the younger group had more portal vein thrombosis and fundic varices. These findings should constitute a reliable guideline for evaluating patients at the transplant center and for health policy makers to develop better strategic preventive measures against liver diseases. Our data revealed a proportionally greater rise in liver transplant candidacy due to NAFLD-associated cirrhosis with portal vein thrombosis. This could be related to the predisposition of patients with NAFLD to developing pro-coagulation and impaired blood flow, as well as a pro-inflammatory state.

2. Bone morphogenetic protein 6 exhibits protumorigenic effects on hepatocellular carcinoma cells in vitro

Hanna Ehnis (Erlangen, DE), Judith Sommer (Erlangen, DE), Anja Bosserhoff (Erlangen, DE), Claus Hellerbrand (Erlangen, DE)

Introduction: Different bone morphogenetic proteins (BMPs) have been shown to act as tumor suppressors as well as oncogenes in different types of cancer including hepatocellular carcinoma (HCC). BMP6 has been mostly studied in the context of iron metabolism but its role in HCC is largely unknown. The aim of this study was to analyze effects of BMP6 on HCC cells.

Methods: Different human HCC cell lines (Hep3B, PLC, SNU449 and HepG2) were stimulated with recombinant BMP6 (rBMP6). The expression of the BMP-type 1 receptors activin receptor-like kinases 2 and 3 (ALK2, ALK3) was suppressed using RNAi technology.

Results: Stimulation of HCC cells with rBMP6 caused a time- and dose-dependent induction of the expression of the transcription factors inhibitor of differentiation 1 and 2 (ID1 and ID2), known targets of BMP-signaling and promoters of HCC progression. Furthermore, rBMP6 induced Smad1/5/8 phosphorylation of HCC cells. ALK2 and ALK3 suppression respectively led to an approx. 50% reduction and the combined suppression of both ALK2 and ALK3 to a complete inhibition of the rBMP6 induced Smad1/5/8 phosphorylation and ID1/2 expression in HCC cells. In silico analysis of TCGA-LIHC human HCC tissues revealed that enhanced BMP6 expression correlates with poor patients' survival.

Discussion/Conclusion: Our in vitro analyses indicate that BMP6 exhibits protumorigenic effects on HCC cells. The inverse correlation of BMP6 with patients' survival further suggests BMP6 as tumor promoter in HCC. Additionally, our study implies that the protumorigenic BMP6 effects are mediated via ALK2 and ALK3. Together, our study indicates BMP6 and ALK2/3 as therapeutic targets in HCC patients.

3. Hepatic stellate cells show protumorigenic effects on melanoma cells in vitro

Verena Freutsmiedl (Erlangen, DE), Tatjana Seitz (Erlangen, DE), Anja Bosserhoff (Erlangen, DE), Claus Hellerbrand (Erlangen, DE)

Introduction: The liver represents an attractive niche for metastasis of numerous tumors including melanoma. The underlying molecular mechanisms are largely unknown however, there is evidence that hepatic stellate cells (HSCs) have a protumorigenic effect on melanoma cells. The aim of this project is to develop an in vitro model system to analyse effects of HSCs on melanoma cells.

Methods: Primary human HSC and human melanoma cell-lines were used in different in vitro systems. First, melanoma cells were incubated with conditioned media (CM) from HSCs. Second, spheroid formation of melanoma cells with or without HSCs was performed.

Results: Functional assays showed that CM from HSCs acts as a potent chemoattractant in Boyden chambers, and similarly, increased migration of melanoma cells in scratch assay. Spheroid formation assay showed that mixed spheroids of melanoma cells and HSCs formed significantly larger spheroids than the sum of each of the two cells types alone, indicating a growth-promoting effect of HSCs on melanoma cells. Furthermore, we observed that HSCs induced a significant induction of smad-1/5/8 phosphorylation in melanoma cells, indicating that HSC secreted bone morphogenetic proteins (BMPs) are involved in the protumorigenic effects of HSC on melanoma cells.

Discussion/Conclusion: The here provided in vitro data confirms protumorigenic effects of HSCs on melanoma cells and suggests BMPs as potential candidates. We propose that our in vitro model system can be used to identify further potential candidates and underlying molecular mechanisms as well as potentially diagnostic markers and therapeutic targets for hepatic metastasis of melanoma cells.

4. Longitudinal analyses of innate lymphoid cells in patients with HCC

Bernd Heinrich (Hannover, DE), Tijana Ristic (Hannover, DE), Laura Christin Kusche (Hannover, DE), Heiner Wedemeyer (Hannover, DE)

Introduction: Hepatocellular carcinoma (HCC) is a very heterogeneous type of cancer. Resection, ablation and transplant, as well as standard therapy for patients with advanced HCC, atezolizumab/bevacizumab (Atezo/Bev) interferes with the HCC microenvironment and

modifies immune responses. We hypothesize that a unique pattern of innate lymphoid cell (ILC) –frequency and –function correlates with clinical characteristics and therapy in HCC.

Methods: We performed flow-cytometry of PBMCs derived from 75 HCC patients with viral and metabolic etiology of liver disease with and without cirrhosis. We compared ILCs, NK and T cell frequencies between four study groups: healthy subjects, patients with HCC before and after therapy with Atezo/Bev and tumor-free patients after ablative or surgical therapy at the time of analysis. Clinical parameters were correlated with flow-cytometry results and therapy response.

Results: Subgroup analysis of ILCs revealed a significant increase of CD8+ ILC1s and decrease of CD4+ ILC1s after 3 months treatment compared to baseline HCC patients. For further phenotypical analysis, we stained ILC1s for PD-1, CTLA-4 and PD-L1 showing the highest median fluorescence intensity of CTLA4 in three months follow-up compared to baseline HCC and first follow-up patient group. PD-1 and PD-L1 were very low expressed in all groups. CD56brightNK cells showed a decrease in frequency after systemic therapy in parallel with an increase of CD56dimNK cells. Interestingly, NK cells frequencies were similar in healthy controls and recurrence-free patients, including a high frequency of a previously described cytotoxic NKp80+NK cell subgroup.

Discussion/Conclusion: Our results suggest that HCC development and treatment alters ILCs, NK and T cell composition in PBMCs. Composition of ILCs indicates a more mature but exhausted phenotype after immunotherapy. Further analyses may reveal connections between ILCs and the underlying liver diseases and the response to treatment and may also help to stratify patients before and after transplantation.

5. Overall survival after liver transplantation of incidental cholangiocellular carcinoma patients compared to HCC patients

Sophia Heinrich (Hannover, DE), Katrin Vollmann (Hannover, DE), Kateryna Shmanko (Mainz, DE), Simone Boedecker-Lips (Mainz, DE), Theresa Kirchner (Hannover, DE), Emily Bosselmann (Hannover, DE), Bastian Engel (Hannover, DE), Christian Lange (Munich, DE), Arndt Weinmann (Mainz, DE), Richard Taubert (Hannover, DE)

Cholangiocellular carcinoma (CCA) is the second most common liver malignancy with a dismal prognosis. Previous trials for OLT for unselected CCA showed limited 5-year survival rates from 18–45%. Recent studies suggest that patients with very early CCA may benefit from OLT. We aimed to analyze overall survival (OS) and disease-free survival (DFS) of patients with CCA undergoing OLT and compared them to patients transplanted with HCC.

Methods: We have screened histopathology reports of liver explants from patients transplanted between 2002 and 2021 in three German transplant centers (Hanover, Mainz, Munich) and have retrospectively analyzed OS and DFS. Biliary cancers were classified by TNM staging and location. We further compared OS as well as DFS with those of patients listed and transplanted with HCC.

Results: In total 21 CCA patients and 151 HCC patients have been included in this study. Median follow-up of CCA patients was 53 months. Most patients were male, median age at OLT was 52 and the major underlying liver disease was primary sclerosing cholangitis (68%). Median OS after OLT was 61 months for patients with CCA and 146 months for patients with HCC. OS differed significantly in tumor location (median OS for iCCA 133, pCCA 151and dCCA 5 months, p = 0.04*) and there was a trend in the TNM staging (median OS CCA T1 133 versus T2 61 months). Most importantly, there was no significant OS benefit in HCC patients compared to CCA patients staging T1 (OS HCC 146 months, CCA 133 months, p = 0.88).

Discussion/Conclusion: Carefully selected CCA patients may have a benefit from OLT in terms of OS and do not show a significantly worse OS rate compared to HCC patients. These data support a discussion regarding an adjustment of the current German transplantation guidelines in the future.

6. Harnessing the potential of microRNAs to affect tumor metabolism in hepatocellular carcinoma

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Background: Hepatocellular carcinoma (HCC) is amongst the leading causes of cancer-related deaths worldwide. More effective and novel therapeutic targets are needed to address the high mortality observed in HCC patients. Small non-coding microRNAs (miRNAs) are deregulated in many human cancers, including HCCs. Specific miRNAs have been shown to function as tumor suppressors or oncogenes in cancers and may be therapeutically relevant.

Methods: Since hepatocytes readily take-up nucleic acids, including miRNAs, we studied the therapeutic potential of overexpressing tumor suppressor miRNAs or inhibiting oncogenic miRNAs, in vivo in oncogene-driven transgenic mouse models of HCC via adeno associated virus (AAV)-mediated delivery.

Results: Our results show that modulation of specific miRNAs can affect tumor metabolism, leading to attenuation of liver tumor progression and prolonging overall survival of liver tumor-bearing mice.

Discussion/Conclusion: Targeting miRNAs thus shows promising therapeutic potential in the treatment of HCC, currently, still a largely intractable disease.

7. Exogenous fatty acids induce tumorigenic and prometastatic characteristics of colon cancer cells

Tatjana Itzenhaeuser (Erlangen, DE), Fulya Suzan Schildt (Erlangen, DE), Judith Sommer (Erlangen, DE), Claus Hellerbrand (Erlangen, DE)

Introduction: Obesity and hyperlipidemia are risk factors for the development of hepatic metastases in patients with colorectal cancer (CRC). Furthermore, they are associated with a poor prognosis after resection of hepatic metastases. The underlying mechanisms are only incompletely understood. The aim of this study was to assess the impact of hyperlipidemic conditions on tumorigenic and prometastatic behaviour of colon cancer cells in vitro.

Methods: Free fatty acids (FFA) complexed to albumin were added to the cell culture medium of different human colon cancer cell lines to mimic hyperlipidemia, when fatty acids released from adipose tissue are bound to albumin in the circulation. FFA uptake and triglyceride (TAG) accumulation and their functional effects on colon cancer cells were analyzed.

Results: FFA are taken up in a dose and time dependent manner by CRC cells leading to TAG accumulation and enhanced beta-oxidation. These metabolic changes induced the proliferation as well as the migratory activity of CRC cells. Furthermore, fatty acid uptake induced the colony formation of CRC cells.

Discussion/Conclusion: Our data indicate that fatty acid uptake promotes tumorigenic and prometastatic behaviour of colon cancer cells. We propose that our in vitro model for exogenic high-fat supply can be used to further evaluate the molecular mechanisms of these

protumorigenic effects to develop diagnostic markers and potential therapeutic targets for (hepatic) metastasis of CRC patients.

8. Expression and function of G protein-coupled receptor 37 in hepatocellular carcinoma

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Introduction: G protein-coupled receptors (GPRs) play a critical role in different types of cancer, including hepatocellular carcinoma (HCC). GPR37 is associated with development and progression of some types of cancer, but its role in liver disease and liver cancer is unknown. This project aimed to analyze the expression and function of GPR37 in HCC.

Methods: The expression of GPR37 was analyzed at RNA and protein level in different human HCC cells (Hep3B, HepG2, PLC and SNU449) compared to primary human hepatocytes (PHHs). Furthermore, the expression of GPR37 was suppressed in human HCC cells by RNA interference (RNAi).

Results: Expression of GPR37 was significantly higher in HCC cells compared to PHHs, and in HCC tissues compared to non-tumorous livers. In silico analyses showed that increased GPR37 expression in HCC correlated with poor prognosis of patients. Successful RNAi-mediated suppression of GPR37 expression was demonstrated. The suppressed cells exhibited reduced expression of the proliferation markers cyclin D1 and MKI67, which are known to be associated with poor prognosis in HCC patients. Furthermore, GPR37 suppression resulted in reduced proliferation of HCC cells.

Discussion/Conclusion: Elevated GPR37 in HCC cells and correlation of high GPR37 with poor patient survival indicates this G protein-coupled receptor as a protumorigenic factor in HCC, which is supported by initial in vitro analyses. Further studies are required to identify the ligands that act via GPR37 on HCC cells and to assess their as well as GPR37's potential as a therapeutic target in HCC.

9. Pharmacological inhibition of JNK/c-Jun-signalling in S. mansoni-infected mice

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Introduction: Schistosomiasis, a neglected tropical disease, affects over 240,000,000 people worldwide1 (annual mortality 11,500)2. Paired adult worms produce about 300 eggs daily, eventually provoking granulomatous liver-fibrosis. c-Jun, a transcription factor of the AP1-transcription-complex, supports hepatocellular regeneration, proliferation and apoptosis.

Moreover, c-Jun is permanently induced by S. mansoni-infection3. This study aimed to characterize the effect of pharmaceutical inhibition of c-Jun in S. mansoni-infected mice.

Methods: 12 eight-week-old C57BL/6J-mice were infected with 100 cercariae (♂+♀) each in a water bath. Six weeks post-infection, mice were either supplied with JNK-inhibitor SP600125 (n = 6; SP/Sm) or 0.9% NaCl (n = 6; Sm) via an osmotic pump; the latter served as control.

Equally treated non-infected littermates served as controls and supercontrols. Nine weeks post-infection, hepatic damage and biomolecular alterations were examined by western

blotting, RT-qPCR, immunostaining and other functional tests. Differences were statistically analysed via one-way ANOVA (SPSS29.0.0.0).

Results: Serum ALT levels significantly increased in infected mice (130 U/L) compared to non-infected ones (30 U/L; $p < 0.001$) and increased stronger under JNK-inhibition (165 U/L, $p < 0.001$). Similarly, stronger increases were observed for TH2- ($p < 0.05$; IL4x-fold Sm = 205, SP/Sm = 334; IL13x-fold Sm = 189, SP/Sm = 270) and proliferation-related markers ($p < 0.05$).

Nevertheless, hepatic fibrogenesis has not been significantly influenced, while elevated pSTAT3 levels ($p = 0.019$) propose possible alternative pathways. Serum triglyceride levels and expression levels of fat-metabolism-related marker-genes showed tendencies to decrease stronger under JNK-inhibition.

Discussion/Conclusion: Inhibiting JNK/c-Jun-signalling in S. mansoni-infected mice seems to alter immune responses and metabolism, indicating the crucial role of c-Jun in various hepatic functions. However, the mechanistic basis for increased hepatocellular damage following S. mansoni-infection remains ambigous.

10. Lipopolysaccharide increases risks for liver transplantation inducing neovascularization and immunosuppression

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Introduction: It is believed that endotoxin – lipopolysaccharide (LPS) and cytokines in the area of liver transplantation are important factors influencing circulatory dynamics and ischemia reperfusion injury, and the LPS during the perioperative period can be a biological index that sensitively reflects changes in graft liver function. Although LPS levels generally increase in both living donors and recipients, through measurement of LPS, rejection and infections can be differentiated or predicted, allowing for earlier diagnosis and treatment of such complications. Previous studies showed that LPS is both angiogenic and immunosuppressing, thus promoting metastatic growth (MG). However, the role of LPS as a possible therapeutic target is mostly unclear. We hypothesized that anti-LPS therapy may decrease liver transplantation complications and improve follow up.

Methods: Murine model including 3 groups (25 each) of adolescent mice was used. Metastatic process was modeled by i.v. injection of 200 Ql spontaneously metastasizing mammary adenocarcinoma cell culture suspension. Control group (CG) animals received 200 Ql sterile saline intraperitoneal (i.p.), experimental group 1 (EG1) – 200 Ql suspension of 10 Qg LPS per mouse, experimental group 2 (EG2) – same plus 20 Qg at 0.5 ml anti-LPS monoclonal antibodies. MG evaluated histochemically within lung metastases.

Results: EG1 showed significantly higher ($p < 0.001$) MG compared with the control. MG was characterized by 61.2% higher mitotic index (MI) in the EG1 and 42.3% lower apoptotic index (AI). MI/AI ratio in the EG1 was 3.2 times higher ($p < 0.001$) than control. LPS injection resulted in reliably ($p = 0.002$) higher levels of serum VEGF than in control with strong positive correlation ($r = 0.971$) between circulating VEGF and LPS levels. Addition of anti-LPS monoclonal antibodies significantly decreased MG, MI and increased AI with respective change of MI/AI ratio. VEGF becomes insignificantly higher than in control whilst LPS concentration decreased reliably ($p = 0.014$).

Discussion/Conclusion: Despite the well-established role of LPS as pro-inflammatory, pro-proliferator and pro-neovascularization factor, its role in carcinogenesis remains under

evaluated. Our findings show that targeted anti-LPS therapy may impact tumor growth due to prevention of neovascularization and inflammation as well as inducing apoptosis. Data from this study supports idea of anti-LPS biological therapies following liver transplantation for cancer.

11. Adipocyte secreted factors induce tumorigenic and prometastatic characteristics of melanoma cells

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Introduction: The liver is a frequent site of melanoma metastasis. Obesity is a risk factor for melanoma metastasis and also for poor prognosis of patients after surgical resection of hepatic metastases.

Obesity is characterised by enlargement of adipose tissue and soluble factors derived from visceral adipose tissue reach the liver sinusoids via the portal vein in high concentrations.

The aim of the study was to get first insights whether adipocyte derived factors exhibit protumorigenic effects on melanoma cells and herewith may provide a potential mechanism how obesity affects hepatic metastasis and prognosis of patients.

Methods: Differentiation of the murine embryonic fibroblast cell line 3T3-L1 into adipocytes is an established model to mimic adipogenesis. After differentiation, we generated conditioned medium (CM) of adipocytes. Adipocyte maintenance medium was used as control. Subsequently, different human melanoma cell lines were stimulated with CM or control medium to analyse gene expression and functional effects.

Results: Stimulation with CM induced the expression of vascular endothelial growth factor (VEGF), which is a known pro-angiogenetic factor and a known marker for poor prognosis of melanoma patients with hepatic metastases. Furthermore, adipocyte derived CM induced the expression of heme oxygenase 1 (HMOX1), indicative for oxidative stress, which is a known promotor of prometastatic behavior of melanoma cells. Functional analyses revealed that adipocyte CM induced proliferation of melanoma cells. Moreover, CM from adipocytes induced the migratory activity of melanoma cells in boyden chamber assays.

Discussion/Conclusion: Adipocytes secrete factors that promote protumorigenic and prometastatic behaviour of melanoma cells, which provides a potential mechanism how obesity and enlarged visceral adipose tissue promote (hepatic) metastasis. The here developed in vitro system may be used to identify the protumorigenic adipocyte derived factors as potential diagnostic and therapeutic targets for patients with (hepatic) metastasis.

12. Peroxisome proliferator-activated receptor gamma activation credibility of use as a part of neoadjuvant protocol in liver transplantation for cholangiocarcinoma

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Introduction: Cholangiocarcinoma (CCA), which represents about 10% of all hepatobiliary malignancies, stands as the second most common primary hepatic tumour of the liver, following hepatocellular carcinoma. Radical resection offers acceptable 5-year survival rate,

however, most tumours are diagnosed at an advanced stage. In a 12-centre US study it was found that patients with CCA who were treated with neoadjuvant therapy followed by liver transplantation had an overall 65% 5-year survival rate, and only 20% developed recurrence. It is a very low figure compared to patients who underwent transplantation without any neoadjuvant protocol, which commonly ranged from 53% to 84%. Therefore, development of such protocols is plausible. Peroxisome proliferator-activated receptor gamma – NR1C3 (PPARG) plays an important role in various biological processes including lipid and glucose metabolism. PPARG agonists have been used in treatment of different metabolic disorders and non-alcoholic steatohepatitis decreasing steatosis, inflammation, and fibrosis. Recent studies show its pro-apoptotic and antiproliferative effect. The aim of the study was to clarify the perspectives for cholangiocarcinoma (CCC) targeted therapy with thiazolidinediones as a part of neoadjuvant protocol.

Methods: CCC cell line HuCCT-1 was cultivated in modified medium with 10% fetal bovine serum seeded onto well plates. PPARG agonist pioglitazone 0.5 to 10 mmol/L added in study group cultures. General cells count and nuclei morphology were visualized with the TUNEL-staining protocol and cells viewed with a fluorescence microscope (magn. ×400). The number of apoptotic cells calculated in percentage of total nuclei. Apoptosis related cytokines were analyzed by Western blotting.

Results: Activation of PPARG by pioglitazone caused marked growth inhibition in a time- and dose-dependent manner. Pioglitazone inhibited growth of cholangiocarcinoma cell lines by inducing apoptosis and by cell cycle regulation, and this was associated with caspase-3, -6 and caspase-9 activation. These changes were similar to changes observed in hepatocellular carcinoma Hep G2/Hep 3B lines used for control with comparable pro-apoptotic effect during same time interval but different pioglitazone doses.

Discussion/Conclusion: This study shows plausibility of PPARG activation, which shows positive pro-apoptotic and antiproliferative effect on CCC as well as other tumour types. Molecular targeting with thiazolidinediones, nuclear receptor ligands, may be a promising strategy for treating cholangiocarcinoma as a part of neoadjuvant protocol. However, our previous study of PPARG agonists and hepatocellular carcinoma raised question of individual susceptibility/resistance for this approach, which remains unclear. Moreover, this study has multiple limitations based on the small number of the cell lines and its in vitro design.

13. Targeting vascular endothelial growth factor in HCC may modify the paradigm for patients' selection for transplantation

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Introduction: Hepatocellular carcinoma (HCC) contributes to cancer-related mortality, and liver transplantation is the ideal treatment for this malignancy. The paradigm for the patient selection for transplantation shifts from morphologic criteria towards a combination of biologic, histologic, and morphologic criteria, leading to the establishment of a model for predicting post-transplantation recurrence and outcomes. This approach raises the importance of understanding the biological behavior of HCC, and the response to locoregional and targeted therapies. Existing database shows that inhibition of vascular endothelial growth factor (VEGF) signaling may affect tumor growth through several mechanisms. However, clinical studies involving patients with HCC present various and often disappointing results. We hypothesized that VEGF inhibiting may not be equally effective due to different HHC types.

Methods: Human HCC cells lines Hep 3B, Hep G2, and Sk-hep-1 were cultivated in modified media seeded onto well plates. VEGF-targeting drug sorafenib 0.05 mg/ml added in study group cultures. General cells count and nuclei morphology were visualized with the TUNEL-staining protocol and cells viewed with a fluorescence microscope (magn. 400). The number of apoptotic cells calculated in percentage of total nuclei. Apoptosis related cytokines were analyzed by Western blotting.

Results: Sorafenib related changes become evident in Hep G2/Hep 3B cell lines after 48 hours of treatment leading to a significant time-dependent reduction of cell numbers of 67.9–83.2% ($p < 0.01$). Cells became sparse, rounded, and detached from the dishes representing morphologic signs of apoptosis. This correlated with activation of caspase-9, caspase-3, and caspase-6. However, Sk-hep-1 cell culture responded much worse with only 36.7–43.7% reduction during same time interval.

Discussion/Conclusion: VEGF-targeted therapy may act through parallel mechanisms that have more or less important role depending on tumor type. In certain malignancies VEGF-targeted therapy has significant activity, whereas in other has no clinical benefit. Our study gives explanation to the fact of variations in clinical response rate of VEGF-targeted therapy. Different subtypes of HCC have different sensitivity to VEGF-targeted therapy. However, more studies are needed as it remains unclear how obtained data may influence the recurrence rate and transplantation outcome.

14. Selective cyclooxygenase-2 inhibiting may have pro-apoptotic and anti-proliferative effects on the hepatocellular carcinoma, potentially aleviating the post-transplantation risks and expanding indications

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Introduction: Previous experience shows that liver transplantation in patients with advanced tumors was a futile effort as reccurence and malignancy dependent mortality at short term was a rule. Therefore, approaches to diminish metastatic growth and malignant process are of potential interest. Prostaglandins and thromboxanes are bioactive lipids that regulate many physiological responses and are recognized as players in inflammation and carcinogenesis, including hepatic tumors. Moreover, prostaglandins may control gene transcription through the activation of nuclear receptors of the peroxisome proliferators-activating receptor family. Several studies showed that selective cyclooxygenase-2 (COX-2) inhibitors suppress growth of cancer cells and have chemopreventive potential towards colonic cancerogenesis. However, it is still debatable whether COX-2 contributes to the malignant growth and whether inhibition of COX-2 modifies the malignant potential of hepatic tumors. It is even more important emphasizing the outcome of liver transplantation for the HCC. The aim of the study was to clarify the pro-apoptotic and anti-proliferative effect of selective COX-2 inhibition on the hepatocellular carcinoma (HCC) cells.

Methods: HCC cells lines Hep G2/Hep 3B were cultivated in modified media seeded onto well plates. Selective COX-2 inhibitor celecoxib 50 Qmol/L was added in study group cultures. Apoptosis related cytokines were analyzed by Western blotting. Apoptotic nuclei (apoptotic DNA fragmentation) were visualized with the TUNEL-staining (Terminal Deoxynucleotidyl Transferase dUTP Nick End Labeling) protocol and cells viewed with a fluorescence microscope (magn. ×400). The number of apoptotic cells calculated in percentage of total nuclei.

Results: Selective cyclooxygenase-2 inhibition led to changes that become evident in Hep G2/Hep 3B cell lines after 48 hours of treatment leading to a significant time-dependent reduction of cell numbers of up to 80% ($p < 0.05$). Microscopically, cells became sparse, rounded, and detached from the dishes representing morphologic signs of apoptosis. This correlated with activation of caspase-9, caspase-3, and caspase-6 cytokines in media. However, exposure of cell cultures to 3 g/mL PgE2 eliminated the COX-2 inhibiting and pro-apoptotic effect on cells. This indicates that the antineoplastic properties of COX-2 inhibiting are dependent on reduced conversion of arachidonic acid to PGE2 attributable to COX-2 inhibition.

Discussion/Conclusion: Selective inhibition of cyclooxygenase-2 is capable of causing marked growth restriction of human liver tumor cells, based on the induction of apoptosis and inhibition of proliferation. The mechanism by which COX-2 inhibiting-related apoptosis is realized remains unclear as well as involvement of other factors into antiproliferative effect of COX-2 inhibitors. Furthermore, it is interesting whether obtained data may influence the liver transplantation, expanding indications and improving patients' follow-up.

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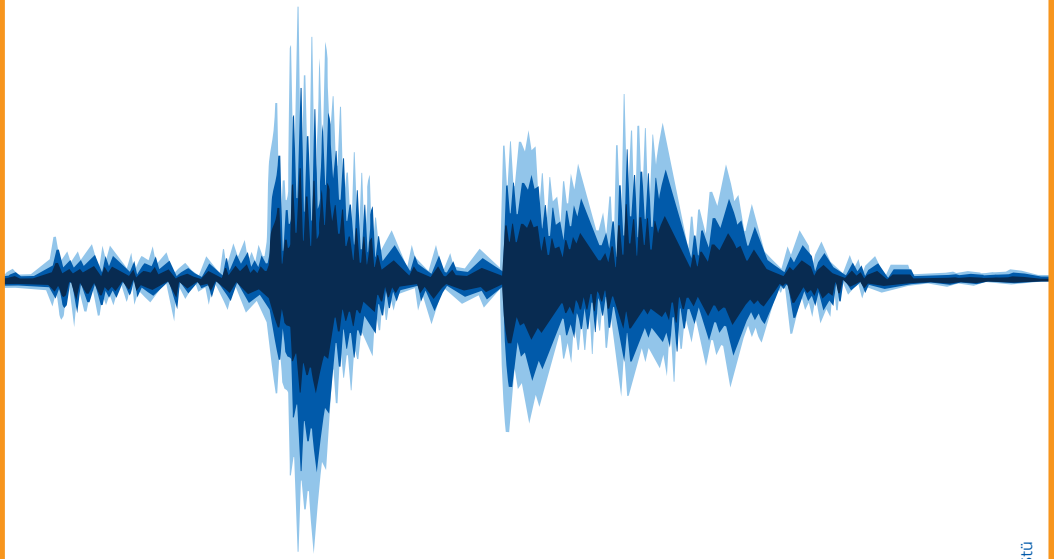

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