



Ludwig Boltzmann Gesellschaft

**LBG MEETING FOR HEALTH SCIENCES 2016**

# **PROGRAMME**

**VIENNA, 28-29 NOVEMBER 2016**

**HOTEL PARK ROYAL PALACE VIENNA**  
Schlossallee 8, 1140 Vienna, Austria

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# DAY 1: MONDAY, 28 NOVEMBER 2016

- 09:30**                      *Registration and welcome coffee*
- 10:30-12:00**                PRE-CONFERENCE SKILLS BUILDING WORKSHOP
- ▶ ›How to publish in a top medical journal‹  
Jan Cools  
KU Leuven Center for Human Genetics, Leuven, Belgium  
Editor-in-chief Haematologica, Journal of the European  
Haematology Association
- 12:00-13:00**                *Lunchbreak (Buffet)*
- 13:00-13:10**                WELCOME NOTE
- ▶ Claudia Lingner  
Ludwig Boltzmann Gesellschaft
  - ▶ Michaela Fritz  
Medical University of Vienna
- 13:10-14:00**                PLENARY SESSION
- ▶ Key Note: ›New perspectives in the  
socioeconomics of health and well-being‹  
Simone Ghislandi  
Vienna University of Economics and Business,  
Vienna, Austria
- 14:00-15:30**                MODERATED POSTER SESSION  
*and coffee break*
- 15:30-17:00**                SESSION I: HAEMATOLOGY AND CANCER RESEARCH
- ▶ Key Note: Udo Schumacher  
Universitätsklinikum Hamburg-Eppendorf,  
Hamburg, Germany
  - ▶ Presentation of the four best abstracts

## DAY 2: TUESDAY, 29 NOVEMBER 2016

- 09:00**                      *Registration and welcome coffee*
- 09:30-11:00**                PLENARY SESSION: TECHNOLOGY PLATFORMS
- ▶ Christoph Bock  
CeMM Research Center for Molecular Medicine  
of the Austrian Academy of Sciences, Vienna, Austria
  - ▶ Irina van der Vlies  
Merck, Zwijndrecht, Netherlands
  - ▶ Karin Schütze  
CellTool, Bernried, Germany
  - ▶ Peter Kraker  
Know-Center, Graz, Austria
- 11:00-12:00**                MODERATED POSTER SESSION
- 12:00-13:00**                *Lunchbreak (Buffet)*
- 13:00-13:30**                UPDATE OPEN INNOVATION IN SCIENCE
- ▶ Lucia Malfent  
Open Innovation in Science Research and Competence Center,  
Ludwig Boltzmann Gesellschaft, Vienna, Austria
- 13:30-15:00**                SESSION II: CARDIOVASCULAR RESEARCH
- ▶ Key Note: Lina Badimon  
Centre d'Investigació Cardiovascular CSIC-ICCC,  
Barcelona, Spain
  - ▶ Presentation of the four best abstracts
- 15:00-15:30**                *Coffee break*
- 15:30-17:00**                SESSION III: REGENERATIVE MEDICINE RESEARCH
- ▶ Key Note: Gerjo van Osch  
Erasmus MC, Rotterdam, Netherlands
  - ▶ Presentation of the four best abstracts
- 17:00-17:30**                AWARD CEREMONY  
*followed by dinner (Buffet)*
- ▶ Host: Stuart Freeman  
FM4 Morning Show
  - ▶ Barbara Weitgruber  
Federal Ministry of Science, Research and Economy
  - ▶ Dejan Baltic  
Merck

**LBG MEETING FOR HEALTH SCIENCES 2016**

# **ABSTRACTS**

### New perspectives in the socioeconomics of health and well-being

► Simone Ghislandi

Vienna University of Economics and Business, Vienna, Austria

The relation between low socioeconomic status and bad health is well-documented across all countries. Despite the consensus regarding the evidence, however, the debate on the causal mechanisms behind this relation is still ongoing. Boosted by the experience of the Great Recession started in 2008 and by the double-dip recession witnessed by the Eurozone, a new interest for the topic has raised among scholars and new perspectives have emerged. In this talk we will review this new set of studies and we will try to make sense of the existing, sometimes contradictory, evidence, relying on a conceptual framework which is at the crossroad between economics, sociology, psychobiology and human genetics.

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### Metastasis research in the post-genomic era

► Udo Schumacher

Universitätsklinikum Hamburg-Eppendorf, Hamburg, Germany

Despite recent advances in the understanding of the molecular biology of cancer, mechanisms leading to distant metastasis formation are still poorly understood. As no genetic basis for metastasis formation has been identified, regulatory mechanisms must guide the seeding of malignant cells. As metastasis formation is so complex, only animal models are suited to represent the entire metastatic cascade. Over the past years our group has developed several spontaneous cancer metastasis xenograft models. As these models reflect the entire metastatic cascade, two important limitations of current cancer therapies can be analyzed namely the epithelial-mesenchymal transition (EMT) and the increased interstitial fluid pressure (IFP). The main problem in analysing the molecules responsible for metastatic spread is the fact that metastatic cells are so few; indeed even in the bloodstream, which already represents a positive selection towards metastasis, less than 99.9 % of the circulating tumor cells (CTCs) actually form metastases. In order to enter the bloodstream, cancer cells have to undergo EMT and within the large mass of the primary tumour, cancer cells which have undergone EMT and can become potentially metastasizing ones are rare. Therefore the compositions of the genome/transcriptome/proteome of these cells are overlooked in bulk analyses of primary tumours. As EMT undergone CTCs have to leave the circulation to form metastasis in distant organs, they must interact with the endothelium at the target site of the prospective metastasis. We therefore hypothesized that they use the same cell adhesion molecules (CAMs) for extravasation as leukocytes do when they leave the circulation. In order to overcome the analytical problem described above, we knocked CAMs in mice and/or tumour cells out and analysed the metastatic behaviour. Indeed, if E- and P-selectins were knocked out in immunodeficient mice, the number of spontaneous lung metastases decreased by almost 85 %. However, this reduction was not seen in every tumour entity: in our neuroblastoma model E- and P-selectins were not of importance for the metastatic spread, hence other molecules of the leukocyte adhesion cascade must be responsible for the spread. Therefore blocking this leu-

kocyte adhesion cascade might be an opportunity to prevent metastatic spread. If metastases have already developed, an approach similar to the treatment of large primary tumours is needed. Access to anti-cancer cell monoclonal antibodies and chemotherapy is hampered by an increased ISF. As long as this ISF cannot be overcome, therapeutic options for metastasized cancers seem to be limited.

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## **Adipose tissue: from low grade inflammation to increased cardiovascular risk**

► Lina Badimon

Centre d'Investigació Cardiovascular CSIC-ICCC, Barcelona, Spain

Adipose tissue (AT), besides its role in energy storage, is a potent source of hormones, peptides, and cytokines, known as adipokines, and stem cells. AT is involved in the regulation of eating habits/behaviour (e.g. leptin), glucose metabolism (e.g. visfatin, resistin, adiponectin), lipid metabolism (e.g. cholesteryl ester transfer protein, lipoprotein lipase, adipocyte fatty acid-binding protein-4, retinol-binding protein, sirtuins), systemic blood pressure (e.g. angiotensinogen, angiotensin-II), inflammation (e.g. TNF- $\alpha$ , IL-6, macrophage chemo attractant protein-1) and thrombosis (e.g. PAI-1). The link between obesity and coronary macro- and micro-vascular disease as well as myocardial function has been attributed to the development of a pro-inflammatory/pro-thrombotic state, together with a cardiometabolic and vascular dysfunction in obese subjects, mainly due to the deregulation of the adipokines secretion profile. Therefore obesity induces a complex multifactorial cardiovascular risk phenotype with multiple comorbidities interacting and contributing to coronary macro- and micro-vascular disease. Our recent results have shown that the role of the stem cell reservoir in the adipose tissue and its differentiation state are dependent on cardiovascular risk factors and on the adipose tissue depot in the body of the host/patient. The active role of adipose tissue in organ function warrants further investigation.

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## **Approaches to use mesenchymal stem cells for repair and protection of cartilage in osteoarthritis**

► Gerjo van Osch

Erasmus MC, Rotterdam, Netherlands

Mesenchymal stem cells have a high potency for regenerative medicine application in the treatment of many diseases, including osteoarthritis. Osteoarthritis is a joint disease that leads to pain and disabilities in over 15 % of the western population. The disease is characterized by inflammation and loss of cartilage and the only available treatment is pain medication and in a late stage replacement of the joint with a prosthesis. There is no treatment that stops or cures the disease. Mesenchymal Stem Cells (MSCs) have gained interest as a possible treatment for osteoarthritis due to their capacity to differentiate into chondrocytes and form cartilage. With our research we investigate the molecular processes of chondrogenesis to find methods to regenerate cartilage from MSCs in a reproducible and reliable manner. The challenges are to find

## KEY-NOTE LECTURES / ABSTRACTS

the best source of MSCs, to expand them to get sufficient numbers and to induce formation of stable cartilage. But what is the value of repairing cartilage when the joint is still inflamed? The inflammation present in the joint will prevent formation of new cartilage and will even degenerate the newly formed cartilage. We should thus treat the inflammation first before considering a cartilage repair treatment. MSCs are, next to their differentiation capacity, known to secrete anti-inflammatory factors and could thus be applied to inhibit joint inflammation. We investigate this capacity of MSCs and methods to keep MSCs in the joint for a prolonged period of time to inhibit chronic inflammation. In this presentation I will highlight some of the challenges of the use of MSCs and explain the research approaches we have chosen to try to overcome these.

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### SESSION I: HAEMATOLOGY AND CANCER RESEARCH FOUR BEST ABSTRACTS

#### **CEBPA-mutant Acute Myeloid Leukemia is sensitive to small-molecule-mediated inhibition of the Menin-MLL interaction – Luisa Schmidt et al.**

► Presenter: Luisa Schmidt

The gene encoding for the transcription factor C/EBP $\alpha$  is mutated in 9 % of patients with acute myeloid leukemia (AML). In AML patients, *CEBPA* N-terminal mutations lead to selective loss of full length C/EBP $\alpha$  p42 expression without affecting translation of the shorter isoform p30. As a balanced ratio of C/EBP $\alpha$  isoforms is crucial for hematopoietic homeostasis, depletion of p42 leads to increased cell growth and blocks myeloid differentiation, resulting in development of AML. However, it is incompletely understood how C/EBP $\alpha$  p30 exerts its leukemogenic effects. We recently showed that p30 preferably interacts with the MLL/SET histone methyltransferase complex. Thus, the p30 variant of C/EBP $\alpha$  could act as a gain-of-function allele featuring specific molecular properties. We hypothesize that the interaction of p30 with the MLL complex is required for p30-dependent epigenetic and transcriptomic changes that contribute to leukemogenesis. Therefore, *CEBPA*-mutant AML might be particularly sensitive to perturbation of MLL/SET function. First, we confirmed that C/EBP $\alpha$  and MLL co-localize on promoters of p30 target genes. Further, myeloid progenitor cells from a *Cebpap*<sup>30/p30</sup> AML mouse model were dependent on MLL, as CRISPR/Cas9-mediated *Mll* knockout led to a decrease in cell survival. To pharmacologically interfere with MLL activity, we used MI-463, a potent and orally bioavailable small-molecule inhibitor of the Menin-MLL interaction. Inhibitor treatment lead to a time- and dose-dependent impairment of proliferation, blocked cell cycle progression and increased apoptosis in *Cebpap*<sup>30/p30</sup> cells. Further, we observed induction of myeloid differentiation upon MI-463 treatment as measured by increased cell surface levels of Mac-1 and Gr-1. This effect appears to be specific for the *Cebpap*<sup>30/p30</sup> genotype, as these cells were 2-6 fold more sensitive towards MI-463 than other leukemia cell lines of mouse and human origin. Overall, we found that *CEBPA*-mutated AML is highly sensitive to perturbation of the MLL/SET complex, either via genetic ablation of MLL or through pharmacological inhibition of the Menin-MLL interaction. These findings contribute to a better understanding of N-terminal *CEBPA* mutated AML and may inform new therapeutic strategies for leukemia treatment.

## **Characterization of a human neurodevelopmental disorder caused by mutations in HACE1 E3 ubiquitin ligase in mice reveals a role for a GTPase Rac1 – Vanja Nagy et al.**

► Presenter: Vanja Nagy

HECT-domain and ankyrin-repeat containing E3 ligase 1 (Hace1) was originally identified in Wilms' tumour and subsequently shown to be a tumour suppressor, to play a role in Golgi membrane dynamics, oxidative stress and heart function. Recently, mutations in HACE1 were shown to cause a type of autosomal recessive neurodevelopmental syndrome in young patients. Clinical features present with variable neurodevelopmental symptoms most notable of which is intellectual disability and abnormal gait. However, molecular mechanisms underlining these symptoms remain unknown. For this reason, we performed extensive, behavioural, cellular and molecular characterizations of Hace1 mutant mice. We observe that Hace1 deficient mice exhibit similar neuroradiological and behavioural features as reported for human patients. Further, we found that Rac1, a small GTPase, previously identified to be a substrate for Hace1 E3 ubiquitination, is upregulated throughout adult Hace1-deficient mouse brains. Rac1-dependent reactive oxygen species (ROS) is also strongly upregulated in these brains as compared to WT littermates. Rac1 regulates cell survival, migration and function early in development and for this reason we are currently investigating the role of Hace1/Rac1 during brain development. To verify that our findings are relevant in humans, we analysed active Rac1 levels, downstream signalling components, cellular migration and ROS production in HACE1 patient-derived fibroblasts. In conclusion, we report that Hace1 deficient mice phenocopy many neuro-pathological features described in humans with HACE1 mutations. We hypothesize that many of these features can be attributed to upregulation of Rac1.

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## **Hydroxyurea inhibits the survival of BCR-ABL1 T315I+ CML sub-clones in vitro and in vivo and synergizes with ponatinib in killing TKI-resistant CML cells – Mathias Schneeweiß et al.**

► Presenter: Mathias Schneeweiß

In chronic myeloid leukemia (CML), BCR-ABL1 T315I is associated with resistance against all BCR-ABL1 tyrosine kinase inhibitors (TKI) except ponatinib. However, resistance against ponatinib may develop in sub-clones carrying compound-mutations in BCR-ABL1. Therefore, alternative therapies have to be considered for these patients. Hydroxyurea (HU) has been used for (palliative) treatment of CML over many years. However, the effects of HU on TKI-resistant mutant sub-clones have not been examined so far. We analyzed the in vivo response of primary cells carrying BCR-ABL1 T315I to HU in four TKI-resistant CML patients who were treated with HU (1-3 g/day) for up to 18 months. In all four patients, white blood counts and total BCR-ABL1 remained stable for three to twelve months. Surprisingly, in three of four patients, the leukemic sub-clone expressing BCR-ABL1 T315I was no longer detectable after HU-treatment as assessed by ligation-dependent PCR. After three months, two of the four patients received an allogenic stem cell transplant. In the other two patients, the disease remained stable for six and twelve months, respectively. In in vitro studies HU was found to inhibit the

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growth of human CML cell lines (K562, KU812, KCL22), primary CML cells, Ba/F3 cells harbouring native BCR-ABL1 (IC50: 236±49 µM), and Ba/F3 cells expressing BCR-ABL1 T315I or T315I-including compound-mutations (IC50: 50-100 µM). Moreover, HU and ponatinib were found to synergize in counteracting the growth of CML cell lines and Ba/F3 cells carrying BCR-ABL1 T315I. In cell mixing experiments, ponatinib exerted strong growth-inhibitory effects on Ba/F3-T315I cells but not on Ba/F3-T315I/E255V cells, whereas HU was found to produce stronger effects on Ba/F3-T315I/E255V cells, and only the combination of ponatinib and HU resulted in complete suppression of both clones. Together, we show that HU exerts strong, sub-clone-specific, anti-neoplastic effects in TKI-resistant CML cells and synergizes with ponatinib in producing growth inhibition. Our observations may pave the way for the development of more effective sub-clone eradicating but also for palliative or bridging-to-SCT concepts in advanced CML. Clinical studies are now warranted to define the exact value of the drug combination ponatinib+HU in TKI resistant CML.

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### **134 novel anti-cancer therapies were approved between Jan 2009 and April 2016: What is the level of knowledge at the time of approval?** – Nicole Grössmann et al.

► Presenter: Nicole Grössmann

**Background:** In the last decade an increasing number of high-priced, new cancer treatments received marketing authorisation in Europe. What is actually known about the clinical benefit of those therapies at the time of approval needs to be elucidated in order to inform decisions about the use and reimbursement of these novel treatment options. **Materials and methods:** To assess the benefit of new interventions as well as expanded indications, we extracted the median gain of the two study endpoints: progression-free survival (PFS) and overall survival (OS). Information is based on approval documents provided by the European Medicine Agency (EMA) and assessments from the Austrian Horizon Scanning programme (HSO). We included all cancer therapies approved in Europe between 2009 (January 1st) and 2016 (April 15th). **Results:** Cancer drugs for 134 new indications approved since 2009 were identified. In the case of 37 indications (27 %), no data was available for PFS or for OS. A gain in median overall survival was reached by 76 licensed indications (55.5 %); 22 (16 %) of them achieved a gain of more than three months. Regarding the study endpoint progression-free survival, an improvement was shown in 90 indications (65.2 %). **Discussion:** Scarce knowledge regarding the clinical benefit of anti-cancer therapies is available at the time of approval. In addition, the survival benefit of the approved indications is less than three months in the majority of approved therapies.

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## **SESSION II: CARDIOVASCULAR RESEARCH** **FOUR BEST ABSTRACTS**

### **Inflammatory response of decellularized small diameter vascular grafts from placenta; in-vitro and in-vivo studies – Karl Heinrich Schneider et al.**

► Presenter: Karl Heinrich Schneider



The future development of vascular prosthetic grafts requires a better understanding of fundamental biological mechanisms like inflammatory response and wound healing. In this study, we investigated arteries from human placenta chorion, decellularized with two different detergents, for their potential use as small diameter vascular grafts (SDVG) and their induced inflammatory response. Arteries from placenta chorionic plate were decellularized using different detergents (Triton X-100 or SDS) under pulsatile perfusion. For improved in-vivo hemocompatibility, grafts were modified by heparin crosslinking. Topography, structure and biomechanical properties of the grafts have been investigated using SEM and a uniaxial BOSE ElectroForce LM1 testbench system, respectively. Macrophage specific gene expression and expression of pro-inflammatory (TNF- $\alpha$ , IL-1 $\alpha$ , CD80, CD86, CCR7) and anti-inflammatory (IL-10, CD163, CD206) cytokines were evaluated in-vitro (macrophage culture) and in-vivo subcutaneous implants in nude rats (n=28, one and four weeks) using real-time PCR. The phenotype of the host cells, which repopulated the graft, was characterized by immunohistochemistry (vWF, CD44, CD68, CD163). The acellular conduits of both groups showed appropriate tensile strength, suture retention strength and compliance for their in-vivo application. Expression of pro-inflammatory cytokines (TNF- $\alpha$ , IL-1 $\alpha$ ) was down regulated in repopulating cells of both grafts. Anti-inflammatory cytokine, IL-10, and M2-macrophages were significantly up regulated in Triton X-100 treated grafts. Both implants showed no foreign body reaction in-vivo and host cells repopulated the grafts. Implantation of decellularized grafts as a functional prosthesis in a rodent model showed good surgical applicability and indicates that this material could be an excellent alternative to synthetic graft materials for small diameter vascular applications.

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## **Thrombectomy for ischemic stroke: previously unnoticed adverse events – Robert Emprechtlinger et al.**

► Presenter: Robert Emprechtlinger

Following coronary heart disease (21 %), stroke (12 %) is the second most common cause of death in Europe. A distinction is made between ischemic stroke and hemorrhagic stroke. With 87 % percent, the ischemic stroke is significantly more common than the haemorrhagic stroke. Mechanical thrombectomy with stent retrievers has shown to be an effective treatment for certain patients with ischemic stroke. Results of recent meta-analyses report that the treatment is effective and safe. However, the endpoints recurrent stroke, vasospasms, and subarachnoid hemorrhage have not been evaluated sufficiently. Hence, we extracted data on these outcomes from the five recent thrombectomy trials in which mainly stent retrievers were used (MR CLEAN, ESCAPE, REVASCAT, SWIFT PRIME, and EXTEND IA published in 2015). Subsequently, we conducted meta-analyses for each outcome. Three studies reported data on recurrent strokes. While the results did not reach statistical significance in the random effects model (despite a three times elevated risk), the fixed effects model revealed a statistically significant higher rate of recurrent strokes after thrombectomy. Four studies reported data on subarachnoid hemorrhage. The higher pooled rates in the intervention groups were statistically significant in both, the fixed and the random effects model. One study reported on vasospasms. We recorded 14 events in the intervention group and none in the control group. The efficacy of mechanical thrombectomy is not questioned, yet our results indi-

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cate an increased risk for recurrent strokes, subarachnoid hemorrhage, and vasospasms post-treatment. Therefore, because thrombectomy is going to play an important role in the routine care, we strongly recommend a thorough surveillance concerning these adverse events in routine registries and future clinical trials in order to assess the real safety risks and to allow for an early reaction against these possible side effects.

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### **Development of novel FP-based probes for live-cell imaging of nitric oxide dynamics – Emrah Eroglu et al.**

► Presenter: Emrah Eroglu

Nitric oxide (NO) is a free radical with a wide range of biological effects, but practically impossible to visualize in single cells. Here we report the development of novel multicoloured fluorescent quenching-based NO- probes by fusing a bacteria-derived NO-binding domain close to distinct fluorescent protein variants. These genetically encoded NO-probes, referred to as geNOps, provide a selective, specific and real-time read-out of cellular NO-dynamics and, hence, open a new era of NO-bioimaging. The combination of geNOps with a Ca<sup>2+</sup> sensor allowed us to visualize NO and Ca<sup>2+</sup> signals simultaneously in single endothelial cells. Moreover, targeting of the NO-probes was used to detect NO-signals within mitochondria. The geNOps are useful new tools to further investigate and understand the complex patterns of NO-signalling on the single (sub)cellular level.

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### **Non-invasive determination of pulmonary hypertension with impedance cardiography – Michael Pienn et al.**

► Presenter: Michael Pienn

Background: Pulmonary hypertension (PH) is a rare and fatal disease with symptoms similar to many other lung diseases. Time from the first visit of a physician to diagnosis of PH is typically two years. Recently, a number of methods have been proposed for earlier diagnosis of PH, however, they usually depend on expensive equipment or highly trained personal. Therefore, we investigated whether hemodynamic changes in patients with PH can be detected by impedance cardiography (ICG), a fast and easily applicable method. Methods and Materials: Recordings of ICG curves were acquired from patients, suspected of PH, during right heart catheterization and from healthy subjects. The datasets were anonymized, transferred to an independent workstation, visually checked for signal quality and analysed with an in-house developed algorithm. ICG curves for individual heart beats were identified by triggering to the simultaneously recorded electro cardiogram (ECG) and averaged over 20 to 100 heart beats. Subsequently, several characteristic points were identified in the curves and used for analysis. Differences between patients with and without PH were tested with Mann-Whitney test. Power to discriminate patients with and without PH was examined with ROC analysis. Results: Datasets of 67 subjects (47 with PH; 20 without PH, including ten healthy controls) were retrospectively analysed. There were no significant differences in age and body surface area between subjects with and without PH (p=0.2 each). Subjects with

PH showed a significantly lower peak height in the ICG signal than subjects without PH ( $2.6\pm 0.7$  vs.  $1.4\pm 0.5$  Ohm/ms, respectively;  $p<0.001$ ), while there was no difference in rise time ( $p=0.5$ ). Hence, the average slope of the ICG signal between minimum and maximum was significantly lower in subjects with PH than in subjects without PH ( $0.015\pm 0.004$  vs.  $0.008\pm 0.003$  Ohm/ms<sup>2</sup>, respectively;  $p<0.001$ ). ROC analysis showed areas-under-the-curve of 0.94 and 0.90 for ICG peak height and average slope of the ICG signal, respectively. Conclusion: Evaluation of peak heights and slopes in ICG curves allows distinction between subjects with and without PH. Thus, ICG may be a fast, cheap and easy method to identify patients with PH, leading to an earlier diagnosis of the disease.

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### **SESSION III: REGENERATIVE MEDICINE RESEARCH FOUR BEST ABSTRACTS**

#### **Collagen-fibrin blend engineered neural tissue: a novel approach for peripheral nerve regeneration – Christina Schuh et al.**

► Presenter: Christina Schuh

Patent pending – abstract not available online

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#### **Implantation of human amniotic membrane ameliorates recurring perineuronal adhesions in a rat sciatic nerve fibrosis model – Angela Lemke et al.**

► Presenter: Angela Lemke

Inflammation, fibrosis and painful adhesions with the surrounding tissue represent major complications associated with surgery of peripheral nerves. Despite improvements in surgical techniques and postoperative rehabilitation programs, clinical symptoms, such as restricted mobility and impaired nerve function, often reappear due to recurring

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adhesions. To avoid these, the most effective method would be the use of a barrier to the surrounding tissue. It should be easy to handle, biodegradable and reduce scar formation and adhesion to a minimum without interfering with the healing tissue. Although several new therapeutic approaches were tested, they could in most cases not satisfyingly hinder the recurrence of symptoms. In this study we aimed to reduce reoccurring fibrotic adhesions between the sciatic nerve and the surrounding tissue by implantation of human amniotic membrane (hAM), the innermost layer of the fetal membranes. Featuring extraordinary flexibility and a thickness of less than 0.05 mm it could represent the ideal candidate for a biological adhesion-preventing barrier. Also, hAM is known to exhibit anti-inflammatory and anti-fibrotic properties, and showed anti-adhesive effects as mesh coating for hernia defects, without causing adverse immune reactions. Therefore we used our newly established rat model for the induction of intraneural fibrosis and perineural adhesions. After generating severe neural damage, the nerve was re-exposed and wrapped with hAM. At different time points (one, four and twelve weeks post implantation), the nerve itself and the recurrence of perineural adhesions were analyzed, using histological, electrophysiological and functional techniques, and compared with controls. As a result, we could show that the implantation of hAM significantly reduced reoccurring adhesions between sciatic nerve and the surrounding tissue, which was examined via morphological and histological evaluation. Also, functional improvement could be verified by the analysis of the rat gait and the calculation of the sciatic functional index. Concluding our study, the implantation of human amniotic membrane represents a very promising therapeutic approach for the prevention of recurring perineural adhesions, hopefully leading to a life without constant pain.

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### **The hybrid vector: a double-active plasmid system for osteoinductive therapy – Ara Hacobian et al.**

► Presenter: Katja Posa-Markaryan

Patent pending – abstract not available online

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## Radiofrequency denervation for sacroiliac and facet joint pain – Katharina Rosian et al.

► Presenter: Katharina Rosian

Within twelve months, a quarter of the Austrian population is affected by chronic low back pain (2014). Reliable epidemiological data on the proportion of facet or sacroiliac (SI) joint pain is missing. We analysed the effectiveness and safety of radiofrequency denervation (RFD), a minimally invasive procedure, in adult patients with chronic, facet joint- or SI joint low back pain and a positive response to diagnostic block. A radiofrequency generator produces an alternating electrical current through an insulated needle. The heat from the tip of the device is used to produce a lesion in the nerves suspected of contributing to the pain. We included a total of 15 randomised controlled trials in this review. Ten randomized controlled trials (RCTs) for facet joint pain (679 patients) and 2 RCTs for SI joint pain (79 patients) fulfilled the inclusion criteria for the effectiveness assessment. Eight of twelve RCTs were placebo controlled. Further, 3 RCTs (comparing different RFD methods) were included for safety considerations. Overall, the level of evidence for the estimated effects is very low to low. RFD for facet joint pain compared to placebo might reduce pain in the short term ( $\leq 1$  month); it might increase functional status between six to twelve months; it might not improve quality of life (QoL) at three months, but might lead to a global improvement in the intermediate term ( $> 1$  up to 6 months). RFD for facet joint pain compared to steroid injections might reduce pain up to twelve months post intervention. RFD for SI joint pain compared to placebo might not reduce pain or improve functional status in the short term ( $\leq 1$  month); it might not increase QoL up to 1, but up to 3 months. No evidence is available for all critical outcomes during an observation period of  $> 3$  months. The current evidence is not sufficient to prove that RFD in adult patients with chronic ( $> 3$  months, facet joint- or SI joint) low back pain who had a positive response to diagnostic block is more effective than, and as safe as, the comparator(s). Therefore, we currently recommend against the inclusion of RFD for SI or facet joint pain in the BMG catalogue of procedures and suggest a re-evaluation in 2019.

**LBG MEETING FOR HEALTH SCIENCES 2016**

# **POSTER WALKS**

## MODERATED POSTER SESSION, DAY 1

Monday, 28 November 2016, Start: 14:00

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## HAEMATOLOGY AND CANCER RESEARCH

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### 1st Poster Walk

Hall Edison I

Moderator: Richard Moriggl, LBI Cancer Research

- 8078 **The Ki-1 antigen (CD30), a novel target in neoplastic canine mast cells, is downregulated by interleukin-4 – Karin Bauer et al.**  
▶ Presenter: Karin Bauer
- 8079 **CAR, a novel marker of erythroid differentiation and migration, is down-regulated in erythropoietic progenitor cells in MDS – Karin Bauer et al.**  
▶ Presenter: Karin Bauer
- 8176 **Comparison of microRNA Patterns in Saliva- versus Serum-derived Exosomes – Ulrike Kegler et al.**  
▶ Presenter: Ulrike Kegler
- 8292 **The pan-BCL-2-blocker obatoclax and the PI3-Kinase/mTOR-inhibitor BEZ235 produce synergistic growth-inhibitory effects in ALL cells – Gabriele Stefanzi et al.**  
▶ Presenter: Gabriele Stefanzi
- 8333 **Targeting STAT5 Oligomerisation in Hematopoietic Cancer – Anna Orlova et al.**  
▶ Presenter: Anna Orlova
- 8334 **Functional Analysis of the Protein Interactome of NUP98-Fusion Proteins in AML – Stefan Terlecki-Zaniewicz et al.**  
▶ Presenter: Stefan Terlecki-Zaniewicz
- 8421 **Melanoma invasion and proliferation is regulated by activation or genetic loss of STAT3 controlling the SOX10-MITF pathway – Alexander Swoboda et al.**  
▶ Presenter: Alexander Swoboda
- 8507 **Generation of novel cellular models for clinically relevant genetic combinations to investigate epigenetic and transcriptomic effects of CEBPA mutations in Acute Myeloid Leukemia – Elizabeth Heyes et al.**  
▶ Presenter: Elizabeth Heyes
- 8521 **Gene hunting: powerful next-generation sequencing (NGS) approaches aimed at identifying new gene defects in patients with primary immunodeficiency disorders – Ana Krolo et al.**  
▶ Presenter: Ana Krolo
- 8525 **The corepressor NCOR1 regulates the development of conventional and innate-like T cell lineages – Lena Müller et al.**  
▶ Presenter: Lena Müller
- 8531 **Loss of c-Jun enhances tumor formation in a mouse model of prostate cancer – Astrid Aufinger et al.**  
▶ Presenter: Astrid Aufinger



- 8532 **Oncogenic role of Kmt2c in prostate cancer – Tanja Limberger et al.**  
▶ Presenter: Tanja Limberger
- 8258 **Comparative analysis of antineoplastic activities from statins and bisphosphonates – Heidrun Karlic et al.**  
▶ Presenter: Heidrun Karlic
- 8259 **Do leukemic cells interact with the osteoblastic niche? – Franz Varga et al.**  
▶ Presenter: Heidrun Karlic
- 8565 **Crosstalk between heme oxygenase, nitric oxide synthase and NADPH oxidase in macrophages – Andrea Müllebner et al.**  
▶ Presenter: Andrea Müllebner
- 8459 **PDGFRB function in Anaplastic Large Cell Lymphoma – Ines Garces de los Fayos Alonso et al.**  
▶ Presenter: Ines Garces de los Fayos Alonso

## 2nd Poster Walk

### Hall Edison I

Moderator: Karoline Gleixner, Medical University of Vienna

- 8551 **An Activating STAT5 Mutation Drives T Cell Lymphomas – Barbara Maurer et al.**  
▶ Presenter: Barbara Maurer
- 8556 **JAK2 in non-small cell lung cancer, friend or foe? – Julian Mohrherr et al.**  
▶ Presenter: Julian Mohrherr
- 8558 **The role of STAT3 $\beta$  in myeloproliferative neoplasms – Petra Aigner et al.**  
▶ Presenter: Petra Aigner
- 8573 **An interdisciplinary approach to identify novel causal genes in rare diseases – Julia Pazmandi et al.**  
▶ Presenter: Julia Pazmandi
- 8588 **Anti-proliferative, cytotoxic and pro-apoptotic effects of novel resveratrol-salicylate hybrid molecules on Jurkat leukemia CD4<sup>+</sup> T cells – Jenny Breitenbach et al.**  
▶ Presenter: Jenny Breitenbach
- 8093 **Oncogenic lipogenesis and signaling – how do they interact in ovarian cancer? – Thomas W. Grunt et al.**  
▶ Presenter: Thomas W. Grunt
- 8341 **Targeting BRD4 as a potential therapeutic approach in JAK2 V617F<sup>+</sup> myeloproliferative neoplasms – Alexandra Keller et al.**  
▶ Presenter: Alexandra Keller
- 8453 **EGF receptor in KRAS driven lung tumors: still a therapeutic target? – Herwig Moll et al.**  
▶ Presenter: Herwig Moll
- 8355 **Identification of a novel STAT5 inhibitor to interfere with the oncogenic activities of STAT5 in AML – Bettina Wingelhofer et al.**  
▶ Presenter: Bettina Wingelhofer
- 8481 **Simultaneous inhibition of STAT3 and STAT5: a novel approach to overcome drug resistance in chronic myeloid leukemia – Karoline Gleixner et al.**  
▶ Presenter: Karoline Gleixner

## POSTER WALKS

- 8489 **Establishing high-content imaging based drug screening for rare disease zebrafish models – Caterina Sturtzel et al.**  
▶ Presenter: Caterina Sturtzel
- 8533 **Uncoupling INK proteins from CDK4/CDK6 HSCs – uncoupling proliferation from senescence – Michaela Prchal-Murphy et al.**  
▶ Presenter: Michaela Prchal-Murphy
- 8629 **The role of the protocadherin CDHR5 in colorectal cancer – Monira Awad et al.**  
▶ Presenter: Monira Awad
- 8028 **Identification, characterization and targeting of putative leukemic stem cells in human mast cell leukemia – Gregor Eisenwort et al.**  
▶ Presenter: Gregor Eisenwort

## 3rd Poster Walk

### Hall Edison II

Moderator: Gregor Hörmann, Medical University of Vienna

- 8568 **Human DOCK2 mutations underlie a pleiotropic immunodeficiency syndrome with early onset, invasive infections – Cecilia Dominguez Conde et al.**  
▶ Presenter: Cecilia Dominguez Conde
- 8346 **Characterization and target expression profiles of CD34+/CD38- and CD34+/CD38+ stem- and progenitor cells in acute lymphoblastic leukemia – Katharina Blatt et al.**  
▶ Presenter: Katharina Blatt
- 8345 **The in vitro effects of various targeted drugs on plasma cells and putative neoplastic stem cells in patients with multiple myeloma– Katharina Blatt et al.**  
▶ Presenter: Katharina Blatt
- 8555 **Functional Dissection of the Transcriptional Network Regulated by NUP98 Translocations in Acute Myeloid Leukemia – Johannes Schmoellerl et al.**  
▶ Presenter: Johannes Schmoellerl
- 8571 **Early onset protein losing Enteropathy, Bowel Inflammation, and Thrombosis in patients with complement regulator deficiency – Rico Chandra Ardy et al.**  
▶ Presenter: Rico Chandra Ardy
- 8063 **The Normalization Visualization Tool: a novel bioinformatic resource to identify optimal normalization strategies for RNA-Seq experiments – Thomas Eder et al.**  
▶ Presenter: Thomas Eder
- 8020 **The First Report of Metastatic Ewing Sarcoma Mouse Model driven by oncogenic fusion of EWS/FLI1 – Tahereh Javarehi et al.**  
▶ Presenter: Tahereh Javarehi
- 8069 **CD44 is a RAS-regulated invasion molecule that is overexpressed in neoplastic mast cells and triggers disease expansion in advanced mastocytosis – Niklas Mueller et al.**  
▶ Presenter: Niklas Mueller
- 8206 **Identification of BRD4 as a Novel Drug Target in Ph+ CML: The BRD4/MYC blocker JQ1 overrides TKI resistance in CML cells – Barbara Peter et al.**  
▶ Presenter: Barbara Peter

- 8488 **(A-)typical cannabinoid receptors GPR55 and CB1 have differential roles in colon cancer – Carina Hasenoehrl et al.**  
▶ Presenter: Carina Hasenoehrl
- 8495 **The methyltransferase SETD2 is required for MLL-rearranged Acute Myeloid Leukemia – Anna Skucha et al.**  
▶ Presenter: Anna Skucha
- 8505 **Palbociclib treatment of FLT3-mutated AML uncovers a kinase-dependent transcriptional regulation of FLT3 and PIM1 by CDK6 – Iris Uras et al.**  
▶ Presenter: Iris Uras
- 8529 **Does Notch-signaling induced by cocultured leukemia-cells modulate differentiation of osteoblasts? – Thomas Heugl et al.**  
▶ Presenter: Thomas Heugl
- 8560 **The TYK2-STAT1 pathway in aggressive T-cell lymphoma – a novel therapeutic intervention site? Nicole Prutsch et al.**  
▶ Presenter: Nicole Prutsch
- 8561 **Identification of prostate cancer biomarkers by DNA-Methylation analysis – Thomas Dillinger et al.**  
▶ Presenter: Thomas Dillinger

## 4th Poster Walk

### Hall Edison II

Moderator: Lukas Kenner, LBI Cancer Research

- 8174 **STAT5BN642H is a driver mutation for leukaemia – Ha T. T. Pham et al.**  
▶ Presenter: Ha T. T. Pham
- 8464 **The multi-kinase inhibitor DCC-2618 counteracts growth and survival of neoplastic cells in systemic mastocytosis – Mathias Schneeweiß et al.**  
▶ Presenter: Mathias Schneeweiß
- 8633 **Oncogenic signaling and epigenetic deregulation – the function of DNMT1 in NPM-ALK driven lymphomagenesis – Elisa Redl et al.**  
▶ Presenter: Thomas Dillinger
- 8254 **Exploring druggability and drug-gene interactions in primary immunodeficiencies – Birgit Höger et al.**  
▶ Presenter: Birgit Höger
- 8295 **Delineation of effects of ponatinib on vascular endothelial cells: a potential basis and explanation for the occurrence of vascular adverse events in CML patients treated with ponatinib – Emir Hadzijusufovic et al.**  
▶ Presenter: Emir Hadzijusufovic
- 8482 **Resveratrol and a resveratrol-salicylate hybrid molecule: a comparative study in CD4+ T-cells – Katrin Goldhahn et al.**  
▶ Presenter: Katrin Goldhahn
- 8520 **Hypermineralization and increased accumulation of Zinc in osteosarcoma tumor matrix – Phedra Messmer et al.**  
▶ Presenter: Phedra Messmer

## POSTER WALKS

- 8634 **HDAC1 genetic deletion accelerates NPM-ALK+ lymphoma formation by activating anti-apoptotic protein expression – Alexandra Zisser et al.**  
▶ Presenter: Alexandra Zisser
- 8460 **Identification of CD25 (IL-2RA) and CD26 (DPPIV) as novel markers and targets in CD34+/CD38- LSC in Ph+ CML – Irina Sadovnik et al.**  
▶ Presenter: Irina Sadovnik
- 8530 **Comparison of extracellular vesicles isolated from human tumor cells – Marta Blank et al.**  
▶ Presenter: Marta Blank
- 8457 **Non-viral CRISPR/Cas9-mediated genome editing and rearrangement: A powerful tool for the generation and functional investigation of large acute myeloid leukemia fusion proteins – Fabio Liberante et al.**  
▶ Presenter: Fabio Liberante
- 8508 **BTK-inhibition is able to suppress IgE-mediated activation and histamine release in human basophils and mast cells – Dubravka Smiljkovic et al.**  
▶ Presenter: Dubravka Smiljkovic
- 8590 **μ-Crystalline is a Hormone Antagonist in Prostate Cancer – Osman Aksoy et al.**  
▶ Presenter: Osman Aksoy
- 8599 **Glibenclamide: an old anti-diabetic drug with anti-cancer activities? – Silvia Loebisch et al.**  
▶ Presenter: Silvia Loebisch
- 8329 **Effects of Histamine Receptor 1 antagonists on growth and survival of canine neoplastic mast cells – Susanne Gamperl et al.**  
▶ Presenter: Susanne Gamperl

## MODERATED POSTER SESSION, DAY2

Tuesday, 29 November 2016, Start: 11:00

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## CARDIOVASCULAR RESEARCH

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### 1st Poster Walk

Hall Edison I

Moderator: Grazyna Kwapiszewska, LBI Lung Vascular Research

- 8472 **Force spectroscopy on red blood cells of different species: a comparative approach – Dina Baier et al.**  
▶ Presenter: Dina Baier
- 8269 **Differential expression of the plasminogen receptor Plg-RKT in monocyte and macrophage subsets – possible functional consequences in atherogenesis – Barbara Thaler et al.**  
▶ Presenter: Barbara Thaler
- 8276 **Gender difference in the mouse model of systemic sclerosis – Valentina Biasin et al.**  
▶ Presenter: Valentina Biasin



- 8458 **Nitric oxide encapsulated poly( $\epsilon$ -caprolactone) small diameter vascular graft – Marjan Enayati et al.**  
▶ Presenter: Marjan Enayati
- 8484 **Isolation and Characterization of Endothelial Extracellular Vesicles from Cell Culture Supernatants – Carina Hromada et al.**  
▶ Presenter: Carina Hromada
- 8485 **In-Vitro Measurement of Forces acting on Transvalvular Aortic Cannulas – Martin Stoiber et al.**  
▶ Presenter: Martin Stoiber
- 8497 **Small diameter vascular grafts with adjustable mechanical properties – Christian Grasl et al.**  
▶ Presenter: Christian Grasl
- 8303 **„Fit for Transplantation“ – Frieda-Maria Kainz et al.**  
▶ Presenter: Frieda-Maria Kainz
- 8526 **Pulmonary Hypertension in Hypersensitivity Pneumonitis – Adrienn Tornyos et al.**  
▶ Presenter: Adrienn Tornyos
- 8536 **Diffusion capacity for nitric oxide in pulmonary hypertension and lung diseases – Balasz Odler et al.**  
▶ Presenter: Balasz Odler
- 8534 **Piloting patient involvement strategies in European HTA: a focus group approach with cardiac patients – Sabine Ettinger et al.**  
▶ Presenter: Sabine Ettinger

## 2nd Poster Walk

### Hall Edison I

Moderator: Philipp Hohensinner, Medical University of Vienna

- 8544 **Increased expression of RGS5 in pulmonary vascular disease – Neha Sharma et al.**  
▶ Presenter: Neha Sharma
- 8554 **Evaluation of Periodic Pump Speed Variations in Ventricular Assist Device Performance by Using Numerical Simulation – Mojgan Ghodrati et al.**  
▶ Presenter: Mojgan Ghodrati
- 8569 **The role of Cartilage Oligomeric Matrix Protein in the pathogenesis of lung fibrosis – Bakytbek Egemnazarov et al.**  
▶ Presenter: Bakytbek Egemnazarov
- 8579 **The regulatory role of Tenascin C on matrix metalloproteases expressions induced by hypoxia and reoxygenation in H9C2 cardiomyocytes cell line – Inês Fonseca Gonçalves et al.**  
▶ Presenter: Inês Fonseca Gonçalves
- 8581 **Influence of Tenascin C on Cardiac Reverse-Remodeling – an Aortic Banding-Debanding Model – Philipp Kaiser et al.**  
▶ Presenter: Philipp Kaiser
- 8635 **SMAD3 contributes to lung vascular remodeling in pulmonary arterial hypertension via MRTF disinhibition: a new pathomechanism – Diana Zabini et al.**  
▶ Presenter: Diana Zabini

## POSTER WALKS

- 8512 **S-Nitroso Human Serum Albumin dose-dependently leads to vasodilation and alters reactive hyperaemia in coronary arteries of an isolated mouse heart model – Paul Haller et al.**  
▶ Presenter: Paul Haller
- 8572 **Short- and Long-Term Mortality within different Age Cohorts of Patients with ST-elevation Myocardial Infarction – Paul Haller et al.**  
▶ Presenter: Paul Haller
- 8564 **Adherence to Current ESC Heart Failure Treatment Guidelines in a Tertiary Referral Centre and University Teaching Hospital in Central Europe – Christina Hauser et al.**  
▶ Presenter: Christina Hauser
- 8065 **Ezetimibe-Statin Therapy compared to Statin Therapy alone – Barbara Nußbaumer-Streit et al.**  
▶ Presenter: Barbara Nußbaumer-Streit
- 8557 **Continuous monitoring of physical activity and cardiac hemodynamics of patients with a left ventricular assist device implanted – Christoph Gross et al.**  
▶ Presenter: Christoph Gross

## 3rd Poster Walk

### Hall Edison I

Moderator: Johann Wojta, LBC Cardiovascular Research

- 8577 **Effects of purified coagulation factor concentrates on hypoperfusion related endotheliopathy – Nikolaus Hofmann et al.**  
▶ Presenter: Nikolaus Hofmann
- 8574 **Experimental models of endotheliopathy: Impact of shock severity – Nikolaus Hofmann et al.**  
▶ Presenter: Nikolaus Hofmann
- 7550 **Angiotensin-II-induced tissue inflammation and fibrosis are distinct from its hemodynamic effects and involve TNFR1 signaling – Sandra Haudek et al.**  
▶ Presenter: Magdalena Mayr
- 8547 **Acute phase cytokines do not contribute directly to liver damage through mitochondrial reactive oxygen species during systemic inflammation – Andras T. Meszaros et al.**  
▶ Presenter: Andras T. Meszaros
- 8257 **Importance of kynurenine in pulmonary hypertension – Bence Nagy et al.**  
▶ Presenter: Bence Nagy
- 8288 **Visualisation of post-mortem vessels in MRI: Development of an imaging approach to improve detection of cardiovascular causes of death – Bridgette Webb et al.**  
▶ Presenter: Bridgette Webb
- 8503 **Targeted antioxidant treatment with mitochondrial Ros-Scavengers SKQ1 and Mitotempo is detrimental in the mouse abdominal sepsis – Pia Rademann et al.**  
▶ Presenter: Pia Rademann
- 8280 **Protective effects of interleukin-33 in critically ill patients – Stefan Stojkovic et al.**  
▶ Presenter: Stefan Stojkovic

- 8281 **Head-to-head comparison of GDF-15 and soluble ST2 in prediction of sudden cardiac death in patients with dilated cardiomyopathy – Stefan Stojkovic et al.**  
▶ Presenter: Stefan Stojkovic
- 8317 **Risk stratification in acute coronary syndrome: evaluation of the GRACE and CRUSADE scores in the setting of a tertiary care centre – Katharina Tscherny et al.**  
▶ Presenter: Katharina Tscherny
- 8619 **Copeptin levels in patients with chest pain with type 1 and type 2 myocardial infarction – Mona Kassem et al.**  
▶ Presenter: Mona Kassem

## 4th Poster Walk

### Hall Edison II

Moderator: Helga Bergmeister, Medical University of Vienna

- 8578 **Potential role of platelet-leukocyte aggregation in trauma-induced coagulopathy: ex vivo findings – Johannes Zipperle et al.**  
▶ Presenter: Johannes Zipperle
- 8580 **Activation of Protein C and the formation of Neutrophil Extracellular Traps in non-traumatic hyperfibrinolysis – Johannes Zipperle et al.**  
▶ Presenter: Johannes Zipperle
- 8316 **Gender-Related Differences in the Perception of Prodromes of Out-of Hospital Cardiac Arrest caused by Myocardial Infarction – Elisabeth Lobmeyr et al.**  
▶ Presenter: Elisabeth Lobmeyr
- 8511 **In vitro effects of anti-anginal drugs on inflammation and coagulation in endothelial cells. A comparative study of nicorandil, trimetazidine and ranolazine – Max Lenz et al.**  
▶ Presenter: Max Lenz
- 8559 **Therapeutic potential of APOSEC against trauma/haemorrhage-induced inflammation, organ failure, and mortality in rats – Arian Bahrami et al.**  
▶ Presenter: Arian Bahrami
- 8567 **Shock wave treatment of 3D cardiac model systems activates ERK1/2 signaling pathway and influences cardiomyogenesis – Christiane Fuchs et al.**  
▶ Presenter: Christiane Fuchs
- 8584 **Characterization of early left ventricle dysfunction in a relevant model for human rheumatoid arthritis – Kiss Attila et al.**  
▶ Presenter: Kiss Attila
- 8587 **Impact Of The HeartWare Ventricular Assist Device Lavare Cycle On Intraventricular Flow Patterns – Philipp Aigner et al.**  
▶ Presenter: Philipp Aigner
- 8570 **Impact of Time of Admission on Short- and Long-term Mortality in the Vienna-STEMI-Registry – Maximilian Tscharre et al.**  
▶ Presenter: Maximilian Tscharre
- 8563 **Use of P2Y12-inhibitors in patients with acute coronary syndrome undergoing PCI in Austria: Insights from the ATTAIn registry – Maximilian Tscharre et al.**  
▶ Presenter: Maximilian Tscharre

## POSTER WALKS

- 8562 **Epicardial Adipose Tissue and its Predictive Effect on Cardiovascular Outcome in Patients with Acute Coronary Syndromes Undergoing Percutaneous Coronary Intervention – Maximilian Tscharre et al.**  
▶ Presenter: Maximilian Tscharre

## 5th Poster Walk

Hall Edison II

Moderator: Heinrich Schima, LBC Cardiovascular Research

- 8502 **A (poly)trauma hit requires functional verification of its immunoinflammatory characteristics and outcome effect upon secondary sepsis – Susanne Drechsler et al.**  
▶ Presenter: Susanne Drechsler
- 8509 **Homocysteine modulates mineralization of murine cell cultures – Norbert Hassler et al.**  
▶ Presenter: Norbert Hassler
- 8535 **Development of a Flow Estimator for Left Ventricular Assist Devices – Martin Maw et al.**  
▶ Presenter: Martin Maw
- 8527 **Age dependent changes in lung vessel morphology in healthy women and men – Michael Pienn et al.**  
▶ Presenter: Michael Pienn
- 8636 **The new St. Thomas Hospital polarized cardioplegia: improved efficacy of myocardial protection in pigs – Anne-Margarethe Kramer et al.**  
▶ Presenter: Anne-Margarethe Kramer
- 8506 **Contribution of mitochondrial nitrite reductase to the regulation of hemodynamics – Peter Dungal et al.**  
▶ Presenter: Peter Dungal
- 8487 **Angiotensin-Like Protein 4 (ANGPLT4): a potential regulator of pulmonary fibrosis – Anita Sahu-Osen et al.**  
▶ Presenter: Anita Sahu-Osen
- 8620 **Circulating copeptin and high-sensitivity troponin I in patients with chest pain after a recent syncope – Kris Vargas et al.**  
▶ Presenter: Kris Vargas
- 8514 **Standardized Telephone Intervention Algorithm for Improved Ventricular Assist Device Outpatient Survival and Reduced Adverse Event Rates – Thomas Schöglhofer et al.**  
▶ Presenter: Thomas Schöglhofer
- 8623 **Increased Platelet Reactivity in Dyslipidemia in Patients on Dual Anti-Platelet Therapy – Bernhard Jäger et al.**  
▶ Presenter: Bernhard Jäger

## MODERATED POSTER SESSION, DAY2

Tuesday, 29 November 2016, Start: 11:00

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### REGENERATIVE MEDICINE RESEARCH

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#### Poster Walk

Hall Edison II

Moderator: tba

- 8538 **Noval human placenta substrates for Neovascularization**  
– Johannes Hackethal et al.  
▶ Presenter: Johannes Hackethal
- 8519 **In-depth characterization of vital human amniotic membrane**  
– Asmita Banerjee et al.  
▶ Presenter: Asmita Banerjee
- 8553 **Adipose Tissue-Derived Therapeutic Cells – Towards a Non-Enzymatic Procedure** – Eleni Priglinger et al.  
▶ Presenter: Eleni Priglinger
- 8552 **Extracorporeal Shock Wave Therapy in situ – a novel approach to obtain an activated fat graft**– Eleni Priglinger et al.  
▶ Presenter: Eleni Priglinger
- 8621 **Extracorporeal shockwave therapy accelerates motor axon regeneration despite a phenotypically mismatched environment** – David Hercher et al.  
▶ Presenter: David Hercher
- 8470 **Inpatient Rehabilitation of painful Shoulder Diseases with and without additional therapeutic Nuclear Magnetic Resonance (NMRT)** – Barbara Stritzinger et al.  
▶ Presenter: Barbara Stritzinger
- 8296 **Fast T2-mapping at 7T in patients after posterior medial meniscectomy under loading** – Sebastian Röhrich et al.  
▶ Presenter: Sebastian Röhrich
- 8504 **Effects of low level light therapy on endothelial cells and vasculogenesis**  
– Peter Dungal et al.  
▶ Presenter: Peter Dungal
- 8524 **Fast and label-free single cell analysis in regenerative medicine** – Heidi Kremling et al.  
▶ Presenter: Heidi Kremling
- 8185 **Spatially resolved determination of Glycosaminoglycan content in bone and cartilage** – Sonja Gamsjaeger et al.  
▶ Presenter: Sonja Gamsjaeger
- 8478 **Geometric morphometrics: discovering the influence of genes on morphology**  
– Uwe Yacine Schwarze et al.  
▶ Presenter: Uwe Yacine Schwarze
- 8575 **Generation of a 3D fibrin scaffold with aligned skeletal muscle-like tissue based on the application of stain** – Babette Maleiner et al.  
▶ Presenter: Babette Maleiner





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## **LBG MEETING FOR HEALTH SCIENCES**

The bi-annual LBG Meeting for Health Sciences promotes and supports young scientists by offering them a stage to present their work and giving them a chance to assert themselves within the scientific setting. This year's conference focuses on the translation of research results from theory into practice. Abstracts on preclinical, clinical and implementation research were submitted. The three thematic sessions on Haematology and Oncology, Cardiovascular Diseases, and Regenerative Medicine include a key-note lecture by an international expert and oral presentations of the four best submitted abstracts. For the first time, these presentations will cover the whole translational research process. The conference is organised by Ludwig Boltzmann Gesellschaft in cooperation with the Medical University of Vienna.

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