Molecular Mechanisms of Ageing and Longevity

7 October, 2022 Heidelberg

Sponsors:

Boehringer Ingelheim

GSK

Roche

Conference Supporters:

abbvie

MERCK

Partners:

evotec

sanofi

Key Public Partner:

Technologiepark Heidelberg

BioRN Annual Conference 2022


Hightech
Roche in Mannheim gehört zu den Hightech-Standorten von Roche.
Wir entwickeln und produzieren diagnostische Tests und wirksame Therapien
für schwerwiegende Erkrankungen. Dies erfordert höchste Sorgfalt und Qualität.
Deshalb investieren wir in modernste Fertigungsanlagen und gewährleisten eine
lückenlose Qualitätssicherung. Mehr unter roche.de
Roche

Table of Contents

Programme

08:30	Registration
09:00	Welcome & Introduction
09:15	**Keynote**: "Novel concepts in Aging and Aging-associated Diseases" **Carien Niessen CECAD & University of Cologne**
09:50	"Cell-type specific regulatory networks to investigate disease mechanisms and ageing" **Judith Zaugg, EMBL**
10:15	Break & Exhibition
11:15	"A geriatric perspective towards geroscience" **Jürgen Bauer, Heidelberg University, Agaplesion Bethanien Hospital** "Ageing and Longevity: An investor's perspective" **Jan Adams, Apollo Health Ventures**
11:55	**'ECEI GOLD Label' and 'Cluster-Exzellenz Baden-Württemberg' Award**
12:00	Lunch & Exhibition

13:30	**Young Scientists' Pitch Competition** moderated by Carsten Hopf, Mannheim University of Applied Sciences
	"Vascular aging limits metastasis" - **Ashik Ahmed Abdul Pari,** DKFZ & Heidelberg University
	"Interferon regulates stem cells in the brain at all ages independently of its antiviral function" - **Damian Carvajal Ibañez**, DKFZ & Heidelberg University
	"A molecular marker for mechanical stress and aging in connective tissues" - **Markus Kurth**, HITS & Heidelberg University
	"Does exercise literally make the heart younger and what does sleep got to with it?" - **Carolin Lerchenmüller**, Heidelberg University Hospital & DKFZ
	"Generation and characterisation of transgenic cell and mouse lines harbouring rare protein-altering genetic variants identified in long-lived individuals" - **Larissa Smulders**, Max Planck Institute for Biology of Ageing, Cologne & CECAD
	"From injury to patterning – the role of Wnt and MAPK signaling in Hydra regeneration" - **Anja Tursch**, Heidelberg University & COS
	"Protective role of cellular heterogeneity preserving the plasticity and complexity of transcriptional networks during ageing" - **Chuan-hsin Yin**, Helmholtz Pioneer Campus (HPC), Helmholtz Zentrum München
15:00	**Keynote**: "Targeting the Cell's Stress Pathways for Therapeutic Benefit" **Peter Walter, Altos Labs and University of California**
15:40	Young Scientists' Pitch Competition: **Award Ceremony**
15:45	**Happy Hour & Speed-Networking**
17:00	Closing

Welcome

Dear BioRN Members and Friends,

On behalf of the BioRN Cluster, we would like to welcome you to the BioRN Annual Conference 2022.

Biotechnology plays a key role to solve the critical issues human being facing, such as resource efficiency, demographic trends, and health. This year´s conference theme “The molecular mechanisms of ageing and longevity” is therefore more relevant than ever. Research on ageing and longevity in the past 20 years grew exponentially and is reflected in the number of research publications. The first record in PubMed with the word “Ageing” dates back to 1925. If in 2002 PubMed collected around 9,000 research papers about aging, in 2021 this number has more than quadrupled. We are very grateful to all renowned speakers, who accepted our invitation to share their exciting results about this topic and are at the heart of the meeting. Congratulations as well to the scientists who applied for the ‘Young Scientists’ Pitch Competition’ and can pitch their cutting-edge research at the conference.

We also would like to thank our sponsors for their dedication and financial support which allowed us to organize an outstanding program.

At the interface of academia and industry, where innovation happens - BioRN enables cooperations and successful transformation of ideas into application. Our vision of making life science matter and innovation happens is supported by you – our members! Let´s keep on connecting, bridging, and exchanging ideas!

We wish you all an inspiring conference day.

Gitte Neubauer	**Michael Boutros**	**Julia Schaft**
Chair	Vice Chair	Managing Director
BioRN	BioRN	BioRN

Moderation

Julia Schaft

Managing Director
BioRN Network e.V., Germany

After completing her PhD in molecular and developmental biology at the University of Giessen and the European Molecular Biology Laboratory in Heidelberg (Germany) in 2002, Julia continued her scientific research on the differentiation of human embryonic stem cells at Genea Ltd in Sydney Australia, an IVF clinic with a strong focus on research and innovation in the IVF and human stem cell field. Julia then took over leadership responsibilities in scientific project management and the supervision of all of Genea's embryo research licences. In 2014 Julia relocated back to Germany and took on an administrative role at the European Molecular Biology Laboratory in Heidelberg (Germany) building up the philanthropic fundraising program, the Friends of EMBL. She then joined BioRN as a project manager for international R&D and translational initiatives in the life sciences sector. Since October 2018 Julia is Managing Director of BioRN where she is also taking on BioRN strategic business development and partnering responsibilities.

Carsten Hopf

Mannheim University of Applied Sciences
BioRN Network e.V., Germany

Carsten Hopf obtained his PhD in biochemistry from Tübingen University/Max-Planck-Institute for Developmental Biology. As an EMBO fellow in neuroscience, he then worked at the Johns Hopkins University School of Medicine for three years, before joining Cellzome AG in Heidelberg in 2001. There, for 13 years, he served in multiple roles in platform technology, drug discovery and business development, and eventually as part of Cellzome's leadership team till 2014. Since 2005, he is a professor of bioanalytics, proteomics and drug discovery at Mannheim University of Applied Sciences. He currently heads the Institute of Instrumental Analytics in the Biotechnology Faculty, the Center of Mass Spectrometry and Optical Spectroscopy (CeMOS) and the M2Aind partnership for innovation in health industry in Mannheim and serves as deputy chair of BioRN's Scientific Advisory Board. He is also an associated professor with both Medical Faculties of Heidelberg University.

Welcome and Wrap-up

Gitte Neubauer

Chair
BioRN Network e.V., Germany

Gitte Neubauer is a scientific founder of Cellzome. She graduated from Imperial College, London in Biochemistry and completed her PhD thesis with Matthias Mann at the European Molecular Biology Laboratory. After the acquisition of Cellzome by GSK in May 2012, Gitte Neubauer took over leadership of the company. She is Director of the Board of BioPro Baden-Württemberg, Director of the Board of the Centre for European Economic Research (Mannheim), a member of the industrial advisory board of the Biotechnology faculty of the University of Applied Sciences in Mannheim and member of the BioRN board since 2014 and chair of the BioRN executive board since 2018.

Michael Boutros

Vice Chair
BioRN Network e.V., Germany

Michael Boutros is the Head of the Division Signaling and Functional Genomics and Coordinator of the Functional and Structural Genomics Program at the German Cancer Research Center (DKFZ). He is also Professor for Cell and Molecular Biology at Heidelberg University. After his PhD at the European Molecular Biology Laboratory (EMBL), he joined Harvard Medical School in Boston as a postdoctoral fellow. In 2003, he started his independent group at the DKFZ in Heidelberg funded by an Emmy-Noether Grant of the German Research Foundation (DFG). He was also supported by the EMBO Young Investigator Program. He later became Head of Division and full Professor at Heidelberg University. Michael Boutros' research interests include functional genomic approaches to understand the regulation of cellular signaling in normal and cancer cells. His laboratory further develops and applies high-throughput screening and multi-omic data integration methodologies to dissect genetic networks and genotype-specific vulnerabilities in cancer. He is supported by the European Research Council (ERC) and is an elected member of the European Molecular Biology Organisation (EMBO). He is a member of the BioRN executive board since 2018.

Opening Keynote

Carien Niessen

Professor Department Cell Biology of the Skin, University Hospital Cologne at the University of Cologne & scientific head CECAD

Carien Niessen heads the Department Cell Biology of the Skin, University Hospital Cologne at the University of Cologne. She did her PhD with Arnoud Sonnenberg at The Netherlands Cancer Institute, Amsterdam, the Netherlands and her postdoc with Barry Gumbiner at Memorial Sloan Kettering Cancer Center in New York. She came to Germany as a group leader at the Center for Molecular Medicine Cologne, received a Cancer Aid professorship at the Department of Dermatology at the University of Cologne, and established the full professor in 2018. She was the speaker of the DFG-funded Collaborative Research Center 829 “Molecular mechanisms regulating Skin Homeostasis” until 2021, and currently serves as the scientific director of the excellence cluster CECAD focuses on Aging and Aging-associated Diseases. Her research interests focus on how biomechanical regulation of cell shape controls the structure and function of renewing epithelial barriers in health, aging and disease.

Novel concepts in Aging and Aging-associated Diseases

Modern societies are facing a dramatic demographic change with an ever-increasing life expectancy, and concurrently, a growing number of elderly and very elderly people. In Germany e.g. the percentage of 65 years and older is predicted to be over 28% by 2040. As aging comes with a range of age-associated diseases, this increase thus poses enormous socio-economic and health challenges for both individuals and their societies. The landmark discoveries that genetic or environmental interventions not only extend life span but also reduce the incidence of age-associated diseases provides unique opportunities to develop novel concepts for healthy aging. Using a multi-disciplinary team and approach, the Cologne excellence cluster on Stress Responses in Aging-associated diseases (CECAD) aims at deciphering the molecular and cellular basis of the aging process, rather than focusing research on understanding individual disease entities, with the long term aim to define new approaches for prevention, diagnosis and treatment of age-associated disease. In my talk I will highlight recent research from CECAD including my own lab how cells respond to stress, how this response affects interorgan communication and how environmental -organismal interactions control stress responses and the aging process.

Plenary

Judith Zaugg

Group Leader, EMBL

Judith Zaugg, group leader at EMBL, leads the computational systems (epi)genetics group (www.zaugg.embl.de). She has an additional faculty appointment at the Molecular Medicine Partnership Unit, a joint venture between EMBL and the University hospital Heidelberg, focused on stem cell-niche networks. Judith Zaugg studied at ETH Zurich (Switzerland), obtained her PhD at Cambridge University and EMBL-EBI (UK), and went to Stanford (USA) for her postdoctoral research. Her group at EMBL uses systems epigenetics approaches to understand basic gene regulatory principles, develops tools to integrate multiomics data* and image analysis*, and applies these tools to understand disease mechanisms. Her MMPU group studies the interaction between hematopoietic and mesenchymal stem cells in the bone marrow niche, and how aging and blood cancer affect these interactions. She was recently awarded the prestigious ERC consolidator award to continue her work on cellular interactions within the bone marrow niche. In addition to her research, Judith Zaugg serves on the Scientific Advisory Board of the Nordic Centre of Molecular Medicine (NCMM) and on the Scientific Advisory Panel of the Hungarian Centre of Excellence in Molecular Medicine (HCEMM). She acts as academic editor at LSA and editorial advisory board member of MSB. *Computational tools: https://grp-zaugg.embl-community.io/GRaNIE/; https://grp-zaugg.embl-community.io/GRaNPA/; https://git.embl.de/grp-zaugg/diffTF; https://github.com/ZauggGroup/DeePiCt.

Cell-type specific regulatory networks to investigate disease mechanisms and ageing

Phenotypic variation (including disease) across individuals has two main sources: genetic variation, and variation in environmental exposures. In the past decades the field made tremendous advances in mapping common genetic variants to complex traits and diseases. Yet, the majority of these disease-associated variants lie in the non-coding part of the genome, which makes is very difficult to understand the underlying molecular mechanisms. For the environmental impact on complex phenotypes, we still know very little, yet epigenetics may play a significant role. Our vision is to mechanistically understand how non-coding genetic variants affect gene regulation, how they interplay with epigenetic processes, and how these interplay impacts cellular signaling and cell-cell interactions. In this talk I will present our most recent work on cell-type specific and enhancer-mediated regulatory networks that integrate environmental signaling with genetic and epigenetic variation. I will show two applications of our predictive models revolving around the aging bone marrow niche and the immune system. Overall, our integrative approach with a focus on gene regulation provides a powerful tool to gain mechanistic insights into complex biological processes.

Jürgen M. Bauer

Director Center for Geriatric Medicine, Heidelberg University, Agaplesion Bethanien Hospital, and Director Network Aging Research, Heidelberg University

Prof. Dr. Jürgen M. Bauer is professor of geriatric medicine and director of the Network Ageing Research at Heidelberg University. He also keeps the position as medical director of the Center for Geriatric Medicine at the Agaplesion Bethanien Hospital Heidelberg. Before moving to Heidelberg in 2016, he was the director of the department for geriatric medicine at the university hospital in Oldenburg and also the director of the Geriatric Clinic at the Oldenburg Rehabilitation Centre. He received his doctorate and PhD at the Friedrich Alexander University Erlangen Nuremberg. His research focuses on the geriatric syndromes sarcopenia and frailty, the interaction between nutrition and functionality in older patients as well as on digital monitoring and digital interventions for older persons. He is the author of more than 200 peer reviewed publications and serves as the associate editor of the Journal of Cachexia, Sarcopenia and Muscle Wasting, of Current Opinion in Clinical Nutrition and Metabolic Care, European Geriatric Medicine and of the German Journal of Gerontology and Geriatric Medicine. From 2007 to 2015 he was a member of the Executive Board of the European Society for Geriatric Medicine (EuGMS). In 2018 he was the congress president of the EuGMS Congress in Berlin. From 2015 to 2017 he was president of the German Society for Geriatric Medicine (DGG). Already in 2007, he had received the honorary award of the DGG. In 2021 he was honoured to become a member of the Academy of Science of Baden Württemberg.

A geriatric perspective towards geroscience

Geriatric medicine focuses on the maintenance and improvement of functionality in older persons with acute and/or chronic comorbidities. It takes the stand of the vulnerable older individual. Multimorbidity which has been defined by the simultaneous presence of multiple health conditions increases significantly with age. As a consequence, pure aging without any comorbidities has to be regarded as an exception to the rule for the older population. The concept of frailty has been developed to identify those are at a high risk for functional decline and negative health outcomes. It may even be regarded as a clinical equivalent to biological age. In frailty research it has again become evident that aging is an immensely complex life-long process which is determined by a multitude of contributing factors. While the perspective on the development of pharmacological substances for the pleiotropic prevention of age-associated degenerative disease has been tempting, there has been accumulating evidence that no single substance will be able to fulfill this expectation. Instead, it may be indicated to adopt a complex system approach that integrates the current knowledge on molecular mechanisms, cells, genes and clinical pathways as well. This approach may allow us to successfully develop multi component intervention programs instead. In this context the vulnerability of the older

population towards adverse drug reactions has to be considered. Potential pharmacological substances will have to be taken for prolonged periods by older adults that are at a high risk of age-associated functional decline. Therefore, a high safety profile will be crucial. A profound knowledge on the aging human including those with comorbidities will constitute the basis of future research when Phase I and II trials will be designed. In addition, combinations of pharmacotherapy and established approaches towards health aging like exercise and diet modification will have to be considered. The process of trial design under the above prerequisites will be challenging. While molecular parameters will most likely become a standard for the evaluation of biological age and possibly rejuvenation, the digital monitoring of physical activity may be advanced towards a highly valuable additive diagnostic concept that integrates many aspects of functionality in older individuals. From a geriatric perspective the close cooperation between geriatricians and biological gerontologists will be pivotal, if at this promising stage the transfer from bench to aging human shall be successful.

Jan Adams

Venture Partner, Apollo Health Ventures

With a career spanning venture investing, company building, strategy consulting and basic research in the life sciences, Jan joined Apollo in September 2021. At Apollo Jan's focus is on Company Creation, one of the cornerstones of Apollo's investment thesis. Prior to joining Apollo Jan was heading commercial activities at the Merck KGaA Innovation Centre in Darmstadt, looking to operationalize all business model-related topics of project teams from within the Merck Group. Before that he was a Managing Director with EMBL Ventures, a Seed and Early-stage VC that was working closely with the EMBL, Europe's flagship laboratory in the life sciences. Notable companies that Jan has been involved with on board level and as investor include Endoart (acquired by Allergan), ViraTherapeutics (acquired by Boehringer Ingelheim), Lipid Therapeutics (licensing to Nestlé Health Science and Falk Pharma), Apellis Pharmaceuticals and Arsanis Bioscience (both with an IPO at NASDAQ). Jan is trained as a biochemist with an MSc from Tübingen University and a PhD in molecular genetics from the University of Cambridge, UK. He additionally obtained a dual MBA degree from ESSEC (Paris) and Mannheim University with distinction and is a Class 11 Kauffman Fellow. Jan is furthermore a recipient of each, a Boehringer Ingelheim PhD, and an Ernst-Schering Post-doctoral Research Fellowship.

Ageing and Longevity: An investor's perspective

Anti-aging and longevity science are terms that encompass emerging yet evidence-backed approaches to delaying or reversing the onset of age-related health decline by intervening in the aging process itself. According to the latest reports, investments in anti-aging and life extension biotechnology are growing exponentially and the global anti-ageing market is predicted to skyrocket from around $190bn currently to a whopping $420bn by 2030, according to a report by P&S Intelligence. Some of the largest financial institutions and corporations, along with the world's wealthiest individuals, are financing this field with hundreds of millions of dollars being raised by investors from major corporate entities, including Alphabet, AbbVie, and BlackRock, to private individuals, such as PayPal co-founder Peter Thiel, Jeff Bezos, or Yuri Milner. Due in part to major advancements in scientific understanding and technologic capabilities, the industry focus has now shifted to an exponential-type medicine, marked by continuous innovation and testing of the limits of the human lifespan. Genome sequencing, epigenetics, exosomes, RNA transcriptomics, and other established and emerging anti-aging therapies are only the tip of the iceberg. Though a recent study has suggested that the ageing process might simply be unstoppable, proponents of biological reprogramming believe ageing is far more malleable than we think. "Ultimately, aging is a disease—a disease that many of the most powerful people on the planet believe can be slowed, stopped, even reversed," says Peter Diamandis, founder of XPRIZE Foundation, bestselling author, and key opinion leader in the space.

This short talk explores a venture capitalist's view on the field as an attractive target for a venture creation investment thesis.

Closing Keynote

Peter Walter

SVP & Institute Director, Altos Labs - Bay Area Institute of Science
Distinguished Professor Emeritus, University of California, San Francisco

Dr. Peter Walter is a Distinguished Professor in the Department of Biochemistry & Biophysics at UCSF and Howard Hughes Medical Institute Investigator. His laboratory has produced groundbreaking research related to the identification and characterisation of key proteostasis networks including the Unfolded Protein and Integrated Stress Response. In addition, Dr. Walter has the vision to leverage these observations into novel therapeutic interventions. His contributions to science have been recognised with many distinguished awards, including the 2014 Lasker Award and 2018 Breakthrough Prize. Dr. Walter has put together a select group of world-renowned experts in neuroscience, and RNA biology to make Altos Lab a reality.

Targeting the Cell's Stress Pathways for Therapeutic Benefit

From its birth in the cradle of the ribosome to its demise in the fangs of proteolytic enzymes, a protein continuously explores different folding states. In most cell compartments, molecular sensors carefully monitor protein folding and instruct down-stream effectors to take corrective actions as needed. In response, cells can make adjustments to their protein folding and degradation machineries to stay in a healthy state of homeostasis. If protein folding defects occur and cannot be corrected in a sufficient and timely manner, cells induce suicide programs. Programmed cell death is thought to protect an organism from malfunctioning rogue cells that result from an accumulation of defective protein. In various pathologies, the life/death balance can inappropriately err on either side: killing cells that would be beneficial if kept alive, or alternatively, inappropriately protecting dangerous, disease propagating cells. Studies of the regulation of proteostasis now emerge as focal points of foundational basic research that powerfully connects to a wide spectrum of unmet clinical needs. I will discuss advances in our lab's efforts to understand the molecular details of the unfolded protein response (UPR), a conserved signaling network that surveys the protein folding status in the endoplasmic reticulum. The UPR signals through three molecularly distinct branches. The development of small, drug-like molecules that selectively target each of the UPR's signaling branches has opened promising new therapeutic opportunities in areas as divergent as cancer, neurodegeneration, diabetes, inflammation, aging, and cognition. As such, the UPR emerges as a prime example of the power of fundamental cell biological discoveries to address problems of immense societal impact.

Young Scientists' Pitch Competition

How it works?

1. Young Scientists have replied to a call of abstracts launched in early summer and now present their research live in a short pitch at 13:30 about their research work (10')
2. The onsite audience selects the best research pitch, through live voting:
    - Go to slido.com and include the code #BioRNConference to access the live voting system or scan the QR code here under
    - Select your favorite Research Pitch and submit your vote – Voting closes at 15:30.
3. The winning Scientist will be announced in the Winner Ceremony after the Closing Keynote

Slido.com

#BioRNConference

#1 - Vascular aging limits metastasis

Ashik Ahmed Abdul Pari[1,2], Mahak Singhal[1,2], Christian Moritz Heyer[3], Anja Gampp[1,2], Nils hebach[4], Carolin Mogler[5], Benjamin Schieb[1,2], Denise Grieshober[1,2], Matthia Karreman[4], Moritz Felcht[1,2], Frank Winkler[4], Matthias Schlesner[3], Hellmut G. Augustin[1,2]

[1]Division of Vascular Oncology and Metastasis, German Cancer Research Center, Heidelberg, Germany. [2]Department of Vascular Biology and Tumor Angiogenesis, European Center for Angioscience, Medical Faculty Mannheim, Heidelberg University, Mannheim, Germany. [3]Biomedical Informatics, Data Mining and Data Analytics, Augsburg University, Augsburg, Germany. [4]Clinical Cooperation Unit Neurooncology, German Cancer Consortium (DKTK), German Cancer Research Center (DKFZ), 69120 Heidelberg, Germany. 5Institute of Pathology, TUM School of Medicine, Munich, Germany. 5Division of Cancer Epigenomics, DKFZ, Heidelberg, Germany.
Presenting author.

Research group- Vascular Oncology and Metastasis, German Cancer Research Center LINK

The host microenvironment communicates dynamically with the tumor cells and actively guides the process of metastasis1. Yet, how this communication network is affected by the process of aging has not been systematically addressed2. Employing a broad array of preclinical models and longitudinal intravital imaging, here we discover that organismal aging impairs metastasis by reducing the colonization potential of tumor cells. Correspondingly, aged breast cancer patients (>50 years) had a significantly longer disease and metastasis-free survival compared to younger cancer patients (<50 years). Single-cell analysis of the aged microenvironment showed that endothelial cells (EC) were the most transcriptionally variable stromal cell-type during aging. In line, focal induction of endothelial aging in vivo by promoting telomere dysfunction was sufficient to reduce metastasis and enhance overall survival in a spontaneous metastasis model. Global transcriptomic and epigenomic profiling of aged endothelial cells identified the APLN-APLNR signaling system as a regulator of vascular aging, with APLNR being strongly downregulated in the aged vasculature. Genetic and pharmacological targeting of the apelinergic axis in young mice suppressed metastasis and prolonged overall survival in preclinical metastasis models. Conversely, restoring the apelinergic axis in aged mice enhanced metastatic colonization. Moreover, serum apelin levels in clinical metastatic tumor patient samples inversely correlated with disease progression. Taken together, the experiments identified the aging endothelium as a roadblock to metastatic colonization with APLN-APLNR signaling acting as a critical determinant of the vascular control of metastasis during aging that may serve as a novel target to therapeutically interfere with metastatic progression.

References

1. Abdul Pari, A. A., Singhal, M. & Augustin, H. G. Emerging paradigms in metastasis research. J Exp Med 218, doi:10.1084/jem.20190218 (2021).

Aunan, J. R., Cho, W. C. & Soreide, K. The biology of aging and cancer: A brief overview of shared and divergent molecular hallmarks. Aging Dis 8, 628-642, doi:10.14336/AD.2017.0103 (2017).

#2 - Interferon regulates stem cells in the brain at all ages independently of its antiviral function

Damian Carvajal Ibañez[1,2,*], Maxim Skabkin[1,*], Jooa Hooli[1,2,3,*], Santiago Cerrizuela[1], Manuel Göpferich[1,2], Simon Anders[4], Anna Marciniak-Czochra[3,5], Ana Martin-Villalba[1]

[1]Division of Molecular Neurobiology, German Cancer Research Center (DKFZ), Heidelberg 69120, Germany; [2]Faculty of Biosciences, Heidelberg University, Heidelberg 69120, Germany; [3]Institute of Applied Mathematics, Heidelberg University, Heidelberg 69120, Germany; [4]Bioquant, Heidelberg University, Heidelberg 69120, Germany; [5]Interdisciplinary Center of Scientific Computing (IWR) and Bioquant, Heidelberg University, Heidelberg 69120, Germany
*These authors contributed equally; presenting author.

Division of Molecular Neurobiology, DKFZ (Prof. Dr. Ana Martin-Villalba) - LINK
Applied Analysis and Modelling in Biosciences, Heidelberg University (Prof. Dr. Anna Marciniak-Czochra) - LINK
Bioinformatics tools for omics data, Heidelberg University (Dr. Simon Anders) – LINK

Interferons are the first line of defense against viral infections across organisms1. Unlike differentiated cells, stem cells display an intrinsic interferon response, which protects them from viruses2. Intriguingly, interferons in the ageing brain impair the activity of stem cells hampering regeneration and plasticity3. Whether the viral protective and the stem cell regulatory functions of interferons are present at all ages and whether these functions are connected is unknown.
We combine single-cell transcriptomics, Ribo-Seq and animal models of interferon to show that this pathway is important for proper stem cell function at all ages. Molecularly, interferon orchestrates cell cycle and mTOR activity to post-transcriptionally repress Sox2 and drive the exit from stem cell activation. The interferon response then decreases in subsequent maturation states, leaving late neural progenitors vulnerable to viral infections. This suggests interferon as a regulator of the increased susceptibility to viral infections of progenitor cells in the developing brain.
Additionally, mathematical simulations indicate that the interferon response is beneficial for the young and harmful for the old brain. Our therapeutic models suggest that blocking interferon signalling at around 11 months of age in mice (40 years in human) prevents the age-related decline of neurogenesis for the rest of the individual's lifespan.
Our study establishes molecular mechanisms of interferon in stem cells and interferons as genuine regulators of stem cell homeostasis and a potential therapeutic target to repair the ageing brain.

References

1. Stanifer ML *et al.* Importance of Type I and III Interferons at Respiratory and Intestinal Barrier Surfaces. *Front Immunol.* **11**:608645. (2020).
2. Wu, X. *et al.* Intrinsic Immunity Shapes Viral Resistance of Stem Cells. *Cell* **172**, 423-438.e25 (2018).
3. Kalamakis, G. *et al.* Quiescence Modulates Stem Cell Maintenance and Regenerative Capacity in the Aging Brain. *Cell* **176**(6):1407-1419.e14. (2019).

#3 - A molecular marker for mechanical stress and aging in connective tissues

Markus Kurth[1,2,3,*], Frauke Gräter[1,2,3]

[1] Heidelberg Institute for Theoretical Studies, Schloß-Wolfsbrunnenweg 35, 69118 Heidelberg, Germany; [2] Centre for Advanced Materials (CAM), Heidelberg University, Im Neuenheimer Feld 225, 69120 Heidelberg, Germany; [3] Interdisciplinary Center for Scientific Computing (IWR), Heidelberg University, Im Neuenheimer Feld 205, 69120 Heidelberg, Germany
Presenting author.

Molecular Biomechanics groups' "Mechanoradicals in collagen" project: LINK
Molecular Biomechanics laboratory at the Centre for Advanced Materials (CAM), Heidelberg University: LINK
Molecular Biomechanics group on Twitter: LINK

Aging slowly damages the material we are made of. Cartilage and tendons steadily lose stability, with severe consequences, from tendon rupture to meniscus operations. Collagen is responsible for the physical strength of these connective tissues. Collagen fibers are elastic to some extent, but if over-stretched they can rupture. How collagen-based materials biochemically respond to mechanical stress and how this relates to aging is poorly understood.
We recently uncovered mechanoradicals in collagen as a key response of connective tissue to high stretch. When mechanically stressing tail & Achilles' tendons and meniscus cartilage, we observed the formation of stable dihydroxyphenylalanine (DOPA) radicals (Zapp et al., 2020; Kurth et al., 2022). Using mass spectrometry, we then identified DOPA as a post-translational modification by oxidation of tyrosine and to a smaller extent of phenylalanine in collagen. These radicals are ultimately degraded to hydrogen peroxide, an important oxidative stress molecule involved in tissue hemostasis.
Strain measurements of tendons sampled from animals of different ages indicate that aging renders tendons stiffer. Here, also changes in the crosslinking in collagen may play a role as they change the mechanical properties of the tissues. In this light, we hypothesize an increase in mechanoradical formation and a larger DOPA "footprint" with age in tendon, cartilage and other collagen-rich tissues.
Our accumulated data propose collagen to act not only as a mere force-carrying material but as a huge redox relay system for mechanical stress, and pave new routes towards combating oxidative stress related aging.

References

1. C. Zapp, A. Obarska-Kosinska, B. Rennekamp, M. Kurth, D. M. Hudson, D. Mercadante, U. Barayeu, T. P. Dick, V. Denysenkov, T. Prisner, M. Bennati, C. Daday, R. Kappl, F. Gräter, Nat. Commun. 2020, 11, 2315.
2. M. Kurth, U. Barayeu, H. Gharibi, A. Kuzhelev, K. Riedmiller, J. Zilke, K. Noack, V. Denysenkov, R. Kappl, T. Prisner, R. Zhubarev, T. Dick, F. Gräter, 2022, in preparation

#4 - Does exercise literally make the heart younger and what does sleep got to with it?

Carolin Lerchenmüller[1,2,3], Ana Vujic[4], Sonja Mittag[1,2,3], Annie Wang[4], Charles P. Rabolli[1,3], Chiara Heß[1], Fynn Betge[1], Ashraf Y. Rangrez[1], Malay Chaklader[5], Christelle Guillermier[6,7], Frank Gyngard[6,7], Jason D. Roh[3,6], Haobo Li[3,6], Matthew L. Steinhauser[6,7,8], Norbert Frey[1,2], Beverly Rothermel[5,9], Christoph Dieterich[1,2], Anthony Rosenzweig[3,6*], and Richard T. Lee[4,10*]

[1] Department of Cardiology, University Hospital Heidelberg, 69120 Heidelberg, Germany. [2] German Center for Cardiovascular Research (DZHK), Partner Site Heidelberg/Mannheim, Heidelberg, Germany.[3] Cardiology Division and Corrigan Minehan Heart Center, Massachusetts General Hospital, Boston, MA 02114, USA. [4] Department of Stem Cell and Regenerative Biology and the Harvard Stem Cell Institute, Harvard University, Cambridge, MA 02138, USA. [5] Department of Internal Medicine, Division of Cardiology, The University of Texas Southwestern Medical Center, Dallas, TX 75390, USA. [6] Harvard Medical School, Boston, MA 02115, USA. [7] Center for NanoImaging and Division of Genetics, Brigham and Women's Hospital, Cambridge, MA 02115, USA. [8] Aging Institute, University of Pittsburgh School of Medicine, Pittsburgh, PA 15219, USA. [9] Department of Molecular Biology, The University of Texas Southwestern Medical Center, Dallas, TX 75390, USA. [10] Division of Cardiovascular Medicine, Department of Medicine, Brigham and Women's Hospital, Boston, MA 02115, USA. [·]Presenting author.

Dr. med. Carolin Lerchenmüller, Labor für Kardiales Remodeling und Regeneration, Universitätsklinikum Heidelberg, Abteilung für Kardiologie, Angiologie, Pneumologie: LINK

Average life expectancy increased throughout the world, and it is expected that ~25% of the European and ~15% of the worldwide population will be older than 65 by the year 2050. Age is an independent risk factor for the development of cardiovascular diseases, and heart failure represents a leading cause of hospitalization and death in older adults. Because loss of cardiomyocytes contributes to heart failure, it is crucial to explore stimuli of endogenous cardiac regeneration to favorably shift the balance between loss and birth of new cardiomyocytes (cardiomyogenesis), especially in the aged heart. However, the human heart has very limited capacity to generate new cardiomyocytes and this capacity further declines with age. We have previously shown that cardiomyogenesis can be activated by exercise in the young adult mouse heart. In this study, we aimed to investigate whether exercise also induces cardiomyogenesis in aged hearts. Aged mice were subjected to a voluntary running protocol, and age-matched sedentary animals served as controls. Cardiomyogenesis was assessed based on 15N-thymidine incorporation and multi-isotope imaging mass spectrometry (MIMS), followed by advanced histology and imaging to account for ploidy and nucleation of the cell. Cardiomyogenesis was observed at a significantly higher frequency in exercised compared with sedentary aged hearts, where no cardiomyogenesis could be detected. The calculated rate of new cardiomyocytes in aged exercised mice was 2.3% per year. This compares to our previously reported annual rate of 7.5% in young exercised mice and 1.63% in young sedentary mice. Transcriptional profiling of young and aged exercised hearts and their sedentary controls revealed that exercise induces pathways related to circadian rhythm, irrespective of age. One known oscillating transcript, however, that was exclusively upregulated in aged exercised hearts, was RCAN1.4, whose regulation and functional role were explored further. Our data demonstrate that voluntary running partially restores cardiomyogenesis in aged mice and suggest that pathways associated with circadian rhythm may play a crucial role in physiologically stimulated cardiomyogenesis.

References

1. Lerchenmüller C*, Vujic A*, Mittag S, Wang A, Rabolli CP, Heß C, Betge F, Rangrez AY, Chaklader M, Guillermier C, Gyngard F, Roh JD, Li H, Steinhauser ML, Frey N, Rothermel B, Dieterich C, Rosenzweig A, Lee RT (2022). Restoration of Cardiomyogenesis in Aged Mouse Hearts by Voluntary Exercise. Circulation 2022;0:10.1161/CIRCULATIONAHA.121.057276 *contributed equally
2. Vujic A*, Lerchenmüller C*, Wu TD, Guillermier C, Rabolli CP, Gonzalez E, Senyo SE, Liu X, Guerquin-Kern JL, Steinhauser ML, Lee RT, Rosenzweig A (2018). Exercise induces new cardiomyocyte generation in the adult mammalian heart. Nature Communications 9:1659, 2018 *contributed equally
3. Roh JD, Houstis N, Yu A, Chang B, Yeri A, Li H, Hobson R, Lerchenmüller C, Vujic A, Chaudhari V, Damilano F, Platt C, Zlotoff D, Lee RT, Shah R, Jerosch-Herold M and Rosenzweig A (2020). Exercise training reverses cardiac aging phenotypes associated with heart failure with preserved ejection fraction in male mice. Aging Cell 19:e13159, 2020

#5 - Generation and characterisation of transgenic cell and mouse lines harbouring rare protein-altering genetic variants identified in long-lived individuals

Larissa Smulders[1], Ramona Jansen[1], Joris Deelen[1,2]

[1]Max Planck Institute for Biology of Ageing, Cologne, Germany. [2]Cologne Excellence Cluster on Cellular Stress Responses in Ageing-Associated Diseases (CECAD), University of Cologne, Cologne, Germany. Presenting author.

Research Group Deelen, Genetics and Biomarkers of Human Ageing: LINK

Ageing is the primary risk factor for many chronic diseases, yet, exceptionally long-lived individuals often seem to escape or at least compress late-life morbidity. We, and others, have previously shown that the heritable component of longevity is likely not explainable by relatively common genetic variants. Therefore, our ongoing research aims to functionally characterise rare genetic variants, which we identified in long-lived individuals from the Leiden Longevity Study and the German Longevity Study. To this end, we use a unique approach, which allows us to study the variants both in vitro (cell lines) and in vivo (mice). As a proof of concept, we currently focus on protein-altering variants in different genes of the insulin/insulin-like growth factor-1 signalling (IIS) pathway.

We generated several transgenic cell and mouse lines by introducing the variants via CRISPR-Cas9. The initial characterisation of the transgenic cell lines has shown that the transcript levels of the genes carrying the genetic variants are decreased, although the corresponding protein levels are only reduced for some variants. The investigation of the IIS pathway has revealed that phosphorylation of S6K is reduced upon insulin stimulation in all examined cell lines. Moreover, an untargeted analysis of these cell lines uncovered a shared proteomic signature. A preliminary assessment of the tissues from the transgenic mouse lines replicates some of these results in a genotype, tissue- and sex-specific manner.

In conclusion, we created several transgenic cell and mouse lines harbouring mutations identified in long-lived individuals that show shared downstream effects, which we are currently exploring in more detail. Our research has the potential to identify the molecular mechanisms encoded by genetic variants in the genome of long-lived individuals. These mechanisms could subsequently be targeted using pharmacological compounds to eventually improve healthy ageing in the general population.

References

1. Deelen, J., Evans, D. S., Arking, D. E., Tesi, N., Nygaard, M., Liu, X., Wojczynski, M. K., Biggs, M. L., van der Spek, A., Atzmon, G., Ware, E. B., Sarnowski, C., Smith, A. V., Seppala, I., Cordell, H. J., Dose, J., Amin, N., Arnold, A. M., Ayers, K. L., . . . Murabito, J. M. (2019). A meta-analysis of genome-wide association studies identifies multiple longevity genes. Nat Commun, 10(1), 3669. https://doi.org/10.1038/s41467-019-11558-2

Baghdadi, M., Hinterding, H. M., Partridge, L., & Deelen, J. (2022). From mutation to mechanism: deciphering the molecular function of genetic variants linked to human ageing. Brief Funct Genomics, 21(1), 13-23. https://doi.org/10.1093/bfgp/elab005

#6 - From injury to patterning – the role of Wnt and MAPK signaling in Hydra regeneration

Anja Tursch[a], Natascha Bartscha[b], Moritz Mercker[c], Anna Marciniak-Czochra[c], Suat Özbek[a], and Thomas W. Holstein[a]

[a]Molecular Evolution & Genomics, COS, Heidelberg University, INF 230, 69120 Heidelberg, Germany.
[b]Department of Biological Sciences, Bergen University, Thormohlens gate 53, 5020 Bergen, Norway
[c]Institute of Applied Mathematics, Heidelberg University, INF 205, 69120 Heidelberg, Germany Presenting author.

Group of Prof. Dr. Thomas Holstein: LINK
Group of Prof. Dr. Anna Marciniak-Czochra: LINK

Wnt signal activation in response to injury is a shared trait between early metazoans and humans. Yet, the molecular cues triggered by injury resulting in Wnt expression have so far been elusive. The basal metazoan Hydra with its unlimited regeneration capacity that depends on Wnt signaling is an ideal model to dissect the temporal sequence of molecular events leading from injury to tissue regeneration. Using a combination of protein biochemical, genetic and in silico analyses, my work elucidated how generic wound signals are transmitted to position-specific axial regeneration in Hydra. My results showed that injury initially led to a release of reactive oxygen species (ROS) and calcium, which in turn activated ERK, p38, and JNK by increasing their phosphorylation levels. MAPK activation was then crucial to promote indiscriminate Wnt expression in early head and foot regenerates of dissected animals. This early injury-related role of Wnt signaling was required to drive the tissue into a regeneration-competent state, which later allowed patterning of the head tissue by the sustained activity of Wnt9/10c, Wnt3, and Wnt7 that was absent in the foot regenerate. We speculated that these stage specific functions of Wnt resulted from the interplay of the wound signal with the “source density”, a graded regenerative competence of the tissue along the oral-aboral axis of Hydra, which was postulated on the basis of classical regeneration experiments. Our hypothesis was validated experimentally by the transformation of presumptive foot tissue into head structures upon incubation with recombinant Wnt or ectopic stabilization of β-catenin. Given the high degree of conservation of the analyzed pathways in the animal kingdom, my work may contribute to a more profound understanding of injury induced signaling circuits that could also play a role in regeneration processes of higher vertebrates.

References

1. Cazet et al. (2021) : Generic injuries are sufficient to induce ectopic Wnt organizers in Hydra, eLife 10:e60562; https://doi.org/10.7554/eLife.60562

Gufler et al. (2018) : β-Catenin acts in a position-independent regeneration response in the simple eumetazoan Hydra, Developmental Biology, Volume 433, Issue 2, Pages 310-323, https://doi.org/10.1016/j.ydbio.2017.09.005.

#7 - Protective role of cellular heterogeneity preserving the plasticity and complexity of transcriptional networks during ageing

K. Yin[1*], M. Büttner[2*], I.K. Deligiannis[1], C. Ogris[2], M. Strzelecki[1], D. Odom[3], F. Theis[2] and C.P. Martinez-Jimenez[1,5]

[1]Helmholtz Pioneer Campus (HPC), Helmholtz Zentrum München, 85764 Neuherberg, Germany. [2]Institute of Computational Biology, Helmholtz Zentrum Munich, 85764 Neuherberg, Germany. [3]German Cancer Research Center (DKFZ), Neuenheimer Feld 280, 69120 Heidelberg, Germany. [4]TUM School of Medicine, Technical University of Munich, Munich, Germany. [*]These authors contributed equally to this work; presenting author.

Martinez-Jimenez group: LINK
Theis group: LINK
Odom group: LINK

The dysregulation of transcriptional networks and the increase in transcriptional variability[1] are crucial components of ageing. In order to investigate the role of cellular heterogeneity in response to a long-term perturbation and its impact on the transcriptional networks of individual cells, three hemizygous knock-out (KO) mouse strains were generated, two of them lacking one copy of a liverspecific transcription factor, Hnf4a and Cebpa respectively, and another missing Ctcf as ubiquitous factor. Here, we perform single-nucleus RNA-seq2[2] in young and aged mice showing higher abundance of polyploid hepatocytes and steatosis in aged C57BL/6J livers. We also observe an increase in transcriptional variability in most of the cell types, and differential transcriptional profiles between aged polyploid hepatocytes. Furthermore, hemizygous KO mice show increase inhepatocyte ploidy and complex transcriptional phenotypes due to the increase in cellular heterogeneity. Importantly, aged livers from all three hemizygous KOs present no relevant steatosis indicating a conserved transcriptional program that rescue liver function upon a long-term perturbation. Our results demonstrate that hepatocyte polyploidization constitutes a non-canonical mechanism that protects against transcriptional dysregulation and associated chronic liver diseases in the context of ageing.

References

1. Martinez-Jimenez, C. P., N. Eling, H. C. Chen, C. A. Vallejos, A. A. Kolodziejczyk, F. Connor, L. Stojic, T. F. Rayner, M. J. T. Stubbington, S. A. Teichmann, M. de la Roche, J. C. Marioni, and D. T. Odom. 2017. 'Aging increases cell-to-cell transcriptional variability upon immune stimulation', Science, 355: 1433-36.
2. Richter, M. L., I. K. Deligiannis, K. Yin, A. Danese, E. Lleshi, P. Coupland, C. A. Vallejos, K. P. Matchett, N. C. Henderson, M. Colome-Tatche, and C. P. Martinez-Jimenez. 2021. 'Single-nucleus RNA-seq2 reveals functional crosstalk between liver zonation and ploidy', Nat Commun, 12: 4264.

Exhibit & Pitch

ACADEMICLABS

Booth no. 1
Pitch in the exhibition area at 10:30

AcademicLabs.com is the leading scientific landscaping and expert identification solution. It is used by Business Development, Search & Evaluation, External Innovation, Competitive Intelligence teams and R&D leadership to quickly grasp who is doing what and working with whom on any given topic, as well as to systematically find and assess scientific collaborators, KOLs, rising stars, emerging startups, senior scientists, academic partners and R&D activities from big pharma, literature, early-stage unpublished research, newly funded projects, clinical trials, patents…
Service and solutions providers for the life science market use AcademicLabs to find highly targeted customers and relevant scientific champions in pharma, biotech and academia in no time.

https://www.academiclabs.com/

@academiclabsco

https://www.linkedin.com/company/academiclabs/?originalSubdomain=be

Booth no. 2
Pitch in the exhibition area at 10:40

BioLabs - Let's grow together & join the Heidelberg life-science ecosystem.

BioLabs is a membership-based network of laboratory and office spaces located in key geographies with proven biotech innovation clusters. BioLabs offers beautifully designed coworking environments that pair fully equipped and supported lab, office, and event spaces with relevant programming and unparalleled access to capital and industry partners.
These fertile, supportive ecosystems allow young companies to shift their focus from start-up operations to experimentation and innovation so they can reach their scientific potential quickly and achieve business success. Companies can start with a single lab bench and scale-up as they grow. BioLabs regards itself as more than just a useful element of infrastructure.
Through its competitive approach, BioLabs generates a forward-looking culture of entrepreneurship, directly promoting the creation of networks and cooperation between regional and international companies and venture capitalists. As such, it is an important facilitator of growth, bridging the gap between biotech research and commercial success.
The expanding BioLabs and Affiliates US network of labs now comprises sites throughout the U.S. and is continuously developing additional domestic and international sites. In 2022 their first European site was opened in Heidelberg (Germany), and further sites in France will follow.
BioLabs Heidelberg will be catalyst for growth and entrepreneurship in Germany's Rhine-Main-Neckar area, enabling the development of tomorrow's life-science
technologies. BioLabs Heidelberg will contribute significantly to the goal of bringing together all relevant stakeholders for sound growth in the region, thus providing an additional boost for Heidelberg as a top research location, for the Rhine-Neckar region as a life-sciences industry hub and to the city development in Heidelberg.

www.biolabs.io

@BiolabsHD

https://www.linkedin.com/company/biolabs-heidelberg/

Cell Signaling TECHNOLOGY®

Booth no. 3
Pitch in the exhibition area at 10:50

Founded by research scientists in 1999, Cell Signaling Technology (CST) is a private, family-owned company with over 400 employees worldwide. Active in the field of applied systems biology research, particularly as it relates to cancer, CST understands the importance of using antibodies with high levels of specificity and lot-to-lot consistency. That's why we produce all of our antibodies in house and perform painstaking validations for multiple applications. And the same CST scientists who produce our antibodies also provide technical support for customers, helping them design experiments, troubleshoot, and achieve reliable results.

www.cellsignal.com

@CellSignal

https://www.linkedin.com/company/cell-signaling-technology/

DIVERSA

Booth no. 4
Pitch in the exhibition area at 11:00

DIVERSA is a biotechnology company promoting the transfer to the clinic of new therapeutic molecules through the application of drug delivery nanotechnology. We want to build a bridge between molecules and patients, between drug discovery and clinical application. DIVERSA aspires to become the company of reference for drug delivery, opening new opportunities for innovative medicines and improving patients' lives.

150,000 people die every day due to diseases. Many of them have no treatment available: of the 7,000 rare diseases identified, only 5% have a treatment at all. The main limitation for the arrival of new therapies is not, however, the discovery of new targets and therapeutic molecules, but the translation of these molecules into the clinic. Less than 3% of the new molecules that are discovered will make it to clinical stages.

With our patented technology of lipid nanoemulsions, we can efficiently bind new drug molecules and transport them to their place of action in their therapeutically active form, so that they can be effective and thus generate new opportunities for drug discovery. It is the most versatile and simple platform on the market, and we have demonstrated its ability to associate and release both biomolecules (proteins, peptides, aptamers, antibodies, DNA or RNA) and hydrophobic small molecules.

We want to make our technology available to the entire research community and CROs, through its sale in the form of kits. These kits will help researchers advance their candidate molecules into further clinical testing stages. We also offer contract research & development services to biotech and pharma companies that need delivery systems for their candidate molecules. With our technology being patented we have the possibility of establishing non-exclusive licenses for specific indications. We are also developing our own R&D projects to create new products and test the technology further.

https://www.diversatechnologies.com/

@DIVERSATechnlgs

https://www.linkedin.com/company/diversatechnologies/

Booth no. 5
Pitch in the exhibition area at 12:30

Evotec is a life science company with a unique business model that delivers on its mission to discover and develop highly effective therapeutics and make them available to the patients. The Company's multimodality platform comprises a unique combination of innovative technologies, data and science for the discovery, development, and production of first-in-class and best-in-class pharmaceutical products. Evotec leverages this "Data-driven R&D Autobahn to Cures" for proprietary projects and within a network of partners including all Top 20 Pharma and over 800 biotechnology companies, academic institutions, as well as other healthcare stakeholders. Evotec has strategic activities in a broad range of currently underserved therapeutic areas, including e.g. neurology, oncology, as well as metabolic and infectious diseases. Within these areas of expertise, Evotec aims to create the world-leading co-owned pipeline for innovative therapeutics and has to-date established a portfolio of more than 200 proprietary and co-owned R&D projects from early discovery to clinical development. Evotec operates globally with more than 4,500 highly qualified people. The Company's 16 sites offer highly synergistic technologies and services and operate as complementary clusters of excellence.

www.evotec.com

@Evotec

https://www.linkedin.com/company/evotec/

Booth no. 6
Pitch in the exhibition area at 10:40

Genscript is the world's leading biotech company providing life sciences services and products. With gene synthesis, peptide, protein, antibody and preclinical drug development service capabilities, we are providing life sciences services and products to over 200,000 scientists in over 100 countries worldwide. We have four major platforms including

(i) life-science services and products platform to provide one-stop solutions to global research communities,
(ii) a biologics contract development and manufacturing organization (the "CDMO") platform,
(iii) an industrial synthetic products platform, and
(iv) an integrated global cell therapy platform.

With global support from its loyal customers and over 5000 employees located across the globe, GenScript continues to strive towards their vision of being the most reliable biotech company in the world, in service of a better and healthier future.

https://www.genscript.com

@GenscriptE

https://www.linkedin.com/company/genscript-europe/

HEALEX

Booth no. 7

Healex is a clinical research data-focused software health IT company based in Cologne and Berlin evolving into one of the leading digital health companies.
With our products, we enable breakthrough innovation through access to clinical (anonymized) patient data and ensure that projects are well managed and costings are profitable.

Clincial Site is a collaborative tool for managing organizational data of a trial, particularly data which is relevant for both site as well as sponsor allowing data to be utilized as a single precise source for all processes related to trial conduct on site - among others recruitment, physician registration and delegation logs. This solution also comes with our Studget extension with which study cost calculations are simplified while taking into account site specific differences to ensure tailored resourcing for successful trial conduct.

Our on Premise Integration and Documentation System supports all processes related to patient enrolment, source data extraction and clinical data management:

- Our Subject Management Application allows sites to manage the enrolment process for all trials on a site - irrespective of sponsors and sponsor systems - in a standardized way very effectively and in the process enabling sites to easily maintain broad accurate data for feasibility.
- Our Integration HUB allows source data integration from the EMR for Research Projects
- Our Clinical Documentation Solution ingests the source data and provides an embedded auxiliary documentations filling data gaps not provided by the EMR

https://healex.systems/

@healex_systems

https://www.linkedin.com/company/healex/

HWI group

Booth no. 8
Pitch in the exhibition area at 12:50

The HWI group is a family-owned, independent service partner for pharma & biotech industries. Founded in 1991, HWI today offers a service portfolio ranging from formulation development, analytical development and quality control, manufacturing of clinical batches and small market batches to non-clinical development, pharmacovigilance and GxP services.
With 150 colleagues at sites in Ruelzheim, Frankfurt and Appenweier HWI looks back on 30 years of experience and a large number of successfully completed projects and drug approvals on behalf of its clients.
HWI is specialised with its technology platform for parenteral, nasal and oral applications on the development of innovative and affordable drugs with a special focus on poorly soluble small and large active ingredients. Our purpose is to discover new ways to improve and extend people‘s life. With novel technologies and innovative services, HWI aims to advance the development of highly effective medicines for everyone.

www.hwi-group.de

https://www.linkedin.com/company/hwi-pharma-services-gmbh/

Booth no. 9
Pitch in the exhibition area at 13:00

Sino Biological is dedicated to providing the scientific community with high-quality recombinant protein and antibody reagents to promote life science research and drug discovery. With the largest validated recombinant protein bank (6000+), a broad range of antigens are covered, including viral proteins, cytokines and growth factors, immune checkpoints, antigens for CAR-T cell therapy, Fc receptors, biomarker proteins, etc, as well as a comprehensive collection of high quality mono- and polyclonal antibodies.
Based on our rich experiences and expertise, several robust recombinant protein expression platforms and antibody discovery methods have been established for customized contracted research projects.

https://www.sinobiological.com/

@SinoInc

https://www.linkedin.com/company/sino-biological-inc/

Surprise Booth (…with wheels)

Founding is a great thing! Everyone improves our world a little bit in their own way. In doing so, you need full attention for your innovation.

But especially during the growth phase, questions like the following often get in your way:

- ? What's happening with my insurance when I'm self-employed?
- ? Who can help with my questions all about social insurance?
- ? What do I need to consider when hiring new employees?
- ? My employees demand benefits, but I have no budget for that, what should I do?

We have the right answer to all of these questions, of course tailored to your individual situation!

Website for Startups https://socialpizza.tk.de/

VectorBuilder

Booth no. 10
Pitch in the exhibition area at 13:10

VectorBuilder is a rapidly growing biotechnology organisation. Our revolutionary online Vector Design Studio has become popular with researchers around the world for its rich functionalities and highly intuitive user interface. We specialise in a range of research products and services such as vector cloning, virus packaging, library construction, stable cell line generation, COVID-19 research reagents, AAV capsid evolution and biodistribution and GMP manufacturing of clinical-grade plasmid DNA and viruses, taking your design right through to therapy.

https://en.vectorbuilder.com/

@VectorBuilder

https://www.linkedin.com/company/vectorbuilder/

About the Organiser

BioRN is a science and industry innovation cluster with a unique combination of all relevant innovation stakeholders in the Life Sciences around Heidelberg at the border between Baden-Württemberg, Rhineland-Palatinate and Hesse, and connected by the rivers Rhein, Main and Neckar.

BioRN is a non-profit network counting more than 130 members. It includes top universities, research institutions and Technology Parks. Ten global pharmaceutical companies have R&D sites or are active in the BioRN network. The ecosystem is completed by a large range of small and medium-sized enterprises as well as local government organizations and interest groups.

With the vision to make life science matter and innovation happen, the cluster management established a clear strategy to become the leading European life science cluster, attracting global investment and talent.

BioRN Cluster management establishes initiatives to nurture and extend networks, stands for the promotion and visibility of the Life Science region and fosters connections to other regions of innovation worldwide. BioRN initiates and manages different programmes to support the development of (academic) research ideas through all necessary development stages. BioRN recently supported the establishment of the Evotec's BRIDGE beLAB2122, a new public-private partnership to fund and execute novel disease modifying therapeutic projects in the region. BioRN is also initiator, project manager and Founding Partner of Biolabs Heidelberg, part of the global BioLabs network of co-working space for startups.

General Information

Social Media

We strongly encourage the use of social media in and around the conference.
Follow the conference on Twitter (@BioRNCluster) and use the hashtag **#BioRNConference** for this conference.
You are welcome to discuss the conference and what you are hearing and seeing, but please refrain from sharing raw data presented, as this may preclude subsequent publication of the data in a scholarly journal.

Young Scientists' Pitch Competition

The onsite audience can select the best research pitch, through live voting: go to slido.com and type: #BioRNConference

Photo and Video Recording

You are participating in an event during which the organizers will be taking photographs and videos that may contain your recognizable image. We will use these photographic and video recordings online, internally or externally (e.g. in press releases, on the BioRN websites, and in the LinkedIn and Twitter social networks) for a period of 10 years. The recordings will be deleted after 10 years. For more information about our privacy policy, please go to the BioRN websites (www.biorn.org). You may, of course, object to the use of the photographic and video recordings at any time during the event by talking to the photo/video team, or after the event by contacting

BioRN Cluster Management GmbH
Im Neuenheimer Feld 582
69120 Heidelberg
Email: info@biorn.org

Notes

Working with the life-science community to be first4patients

We set the bar high for innovation. Inspired by patients, driven by science and powered by our growing global community of partners, we're working hard to discover and develop the next generation of transformative medicines. Together we can learn more, do more and achieve more and get the next breakthroughs to patients faster.

WORKING TOGETHER IN:

Cardiometabolic Diseases | CNS Diseases | Immunology | Oncology & Cancer Immunology
Respiratory Diseases | Retinal Health | Research Beyond Borders | Technologies

FIND OUT MORE

Boehringer Ingelheim

DIGITAL CONFERENCE BOOK

BioRN Life Science Cluster Rhine-Neckar

Im Neuenheimer Feld 582
69120 Heidelberg
Germany

Phone: +49-6221-4305-111
E-Mail: info@biorn.org
Internet: www.biorn.org