

BIH CHARITÉ CLINICIAN SCIENTIST PROGRAM

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BIH Charité
(Junior) (Digital)
(Advanced)
Clinician Scientist
Book

2025

Building on Excellence, Advancing with Purpose: Clinician Scientists for Better Patient Outcomes

Introduction by Dr. Nathalie Huber¹ and Dr. Iwan Meij²

On the occasion of the new edition of our Clinician Scientist Program book³, we took a moment to reflect on what has been happening over the past two years and a lot came to mind: While the years before 2023 saw an incredible number of unforeseeable influences, some of which had a massive impact on our program work – the corona pandemic, the sudden death of our former program director Professor Dr. Duška Dragun end of 2020 and the inauguration of our new program director Prof. Dr. Il-Kang Na beginning of 2023 – we have emerged even stronger from these challenging years with renewed vigor! It is our pleasure to give you a glimpse behind the scenes, to highlight milestones we have achieved since 2023 and to present the dynamic developments of the BIH Charité Clinician Scientist Program (CSP).

To start with, we are proud that in 2024 we could apply our expertise in the field of Clinician Scientists on a national level:

Firstly, we launched the national Translational Tandem Program for Gene- and Cell-based Therapies (nTTP-GCT): Based on its many years of experience and expertise gained from designing and implementing funding programs for early career researchers, the BIH Biomedical Innovation Academy (BIA) was commissioned by the BIH to design and implement an innovative program for promoting talents in the field of gene- and cell-based therapies. We felt honored to take on this task and developed a translational tandem program. This program is aimed at tandems consisting of a Junior Clinician Scientist and a Translational Scientist. The idea behind it is that tandems work together on a translational project in the field of GCT and actively participate in building GCT

expertise and networks. We launched the pilot call in summer 2024 and are delighted that the first nine tandems started in spring 2025 at various medical university sites in Germany. What is particularly noteworthy is that a) our BIH Charité Junior Clinician Scientist Program served as a blueprint for the clinical part of the translational tandem, that b) this program expands an important component of BIA's portfolio, i.e. the promotion of Medical Scientists and that c) through this program the BIA makes a significant contribution to the national impact of the BIH.

Secondly, we are getting closer to our goal of establishing a National Clinician Scientist Cohort (NCSC): We have been nurturing our vision of an NCSC for some time and in

1. | 2. Dr. Nathalie Huber and Dr. Iwan Meij are Co-Heads of the BIH Biomedical Innovation Academy. Dr. Huber is also Head of the Clinician Scientist Office.

3. The current version is the fifth edition of our Program Book. The first edition was published in 2016 on the occasion of the fifth anniversary of the BIH Charité Clinician Scientist Program and the corresponding Jubilee Symposium in June 2016. The second edition was published in June 2018 for the International Symposium on Translational Medicine in Berlin. In May 2021, the third edition was published for the 10th Anniversary Symposium of the BIH Charité Clinician Scientist Program and Memorial Event in Honor of Professor Duška Dragun. The fourth edition was published in June 2023 for the Clinician Scientist Retreat at Schloss Fleesensee.

4. <https://www.bihealth.org/de/translation/innovationstreiber/akademie/personenfoerderung-nationales-translationales-tandem-programm-fuer-gen-und-zellbasierte-therapien-nttp-gct>



Figure 1: Key figures from the BIH Charité Clinician Scientist Programm since 2011.

2024, BIA played a central role in designing the so-called ECLIPSE project (»ECLIPSE – Establishment of Clinician Scientist Programs at Medical Faculties: Status and Expectations«), a collaborative project with the Else Kröner-Fresenius Foundation (EKFS) and the German Association of Medical Faculties (MFT). With the help of this project, the first nation-wide online survey across CSP fellows and alumni of all Clinician Scientist Programs in Germany will be conducted. After completing the ECLIPSE project, the survey is planned to be repeated at regular intervals as a longitudinal study which will further feed the NCSC database, again in close cooperation with the MFT. In the long term the NCSC will serve as an empirical basis for career tracking of Clinician Scientists and, thus, a valuable database for evidence-based recommendations for policymakers and funding agencies as well as for counselling early career clinical researchers on possible career paths and career-related decisions.

Beyond these »go national initiatives«, we have also achieved several significant results and successes on the different funding lines under the umbrella of the BIH

Charité CSP – the key figures/main achievements are briefly listed in figure 1.

Moreover, looking back on the last two years, the good news is that all the »post-pandemic aftereffects« seem to have been overcome and we were able to fully resume our face-to-face events. In June 2024, after six years, we once again hosted a two-day International Symposium on Translational Medicine, which attracted around 170 participants to the »Villa Elisabeth« (Berlin Mitte). The aim of the symposium was to promote scientific exchange among the program participants, to foster interdisciplinary networking, and to establish and expand international collaborations. (Digital) Clinician Scientists had the opportunity to invite internationally renowned experts as speakers, which gave them the great opportunity to discuss their projects with eminent authorities in their own field of research. The varied program consisted of »tandem sessions« between fellows and international experts, poster sessions, and workshops. It also contained for the first time a raffle with cool prizes for the participants. The welcome speeches were given by

Prof. Dr. Ulrich Kintscher, Vice Dean for Research at Charité Universitätsmedizin – Berlin, and Prof. Dr. Christopher Baum, Chairman of the BIH Executive Board and Director of Translational Research at Charité – Universitätsmedizin Berlin. On the first evening of the International Symposium on Translational Medicine, an alumni award ceremony took place, and we were happy that Dr. Bobbert, president of the Berlin Chamber of Physicians, gave an inspiring and very appreciative welcome speech. We bid farewell to 51 alumni that evening and were particularly proud to welcome the 300th alumni of our Clinician Scientist funding lines!

Speaking of events: Our first networking event for Digital Clinician Scientists took place on October 10, 2024. Under the motto »Forging the Digital Future«, around 60 participants gathered at »Old Smithy's Dizzle« in Berlin Friedrichshain to exchange ideas and to network with each other. In addition to project pitches by the fellows, keynote speeches were given by Dr. Simon Drees from the EU Commission and Martin Peuker (former Head of IT, CIO of Charité). In addition, there was an invited presentation by Georgios Nikolis, team leader of Scientific Computing (HPC) at Charité. All in all, it was an unforgettable event that brought our community's great spirit to the surface.

All these developments would not have been feasible without the strong support of our BIA-team – a team with a wealth of experience, expertise and with great enthusiasm. Together with the program directors Prof. Dr. med. Il-Kang Na (CSP) and Prof. Dr. med. Igor M. Sauer (DCSP), we will keep on expanding and optimizing our CS funding lines.

Long-Standing Experience with Structured Clinician Scientist Programs

Clinician Scientist Programs are a modern career path within academic medicine that allow research-active physicians to pursue a structured residency with a contractually regulated share of working time (»protected time«) for research. The programs are based on a combination of a competence-oriented clinical education with a translational curriculum as well as support from both a clinical and scientific mentor. Clinician Scientists

are not considered as »clinicians light« or »researchers light«. Rather, they form the essential link within the competence triad of patient care, research and student teaching. This intertwining is the unique selling point of Clinician Scientists and patients in particular benefit from it (see Dragun et al. 2019).⁵ Today, almost every university hospital in Germany has its own Clinician Scientist Program.

Founded in 2011, the BIH Charité CSP is one of the oldest CSP in Germany and is recognized internationally for its pioneering role. It was awarded »best practice model« by the German Research Foundation (DFG) in 2015.⁶ We participate in several committees (for example in the MFT Working Group Clinician Scientists) and advise university hospitals in terms of structures and measures for promoting up-and-coming translational talents. This has allowed us not only to be continuously involved in national and international debates but also to set benchmarks and quality standards for the structured career pathway of a Clinician Scientist in Germany.

5. Dragun, Duška, Huber, Nathalie, Rösen-Wolff, Angela, Blomberg, Richard. »Clinician Scientists: Ärzte mit Kompetenz-Trias«. Dtsch Arztebl. 2019;116(50): A-2339 / B-1922 / C-865.

6. Empfehlungen der Ständigen Senatskommission für Grundsatzfragen in der Klinischen Forschung der Deutschen Forschungsgemeinschaft: »Etablierung eines integrierten Forschungs- und Weiterbildungsprogramms für »Clinician Scientists« parallel zur Facharztweiterbildung« (2015): <https://www.dfg.de/resource/blob/171670/2bb0d87b5a7ad90839fa5ae3fa8ce9b5/empfehlungen-clinician-scientists-0415-data.pdf>

7. Huber, Nathalie et al. »Stark in Klinik und Forschung: Exzellenzschmiede Clinician Scientist Programm. Berliner Ärzt:innen(2024):<https://magazin.aekb.de/beruf-kompetenz/stark-in-klinik-und-forschung-exzellenzschmiede-clinician-scientist-programm>

8. Wissenschaftsrat drs. 5663-16: »Perspektiven der Universitätsmedizin« (2016): <http://www.wissenschaftsrat.de/download/archiv/5663-16.pdf>

A key component of the ongoing success of the BIH Charité Clinician Scientist Program has been the close collaboration with the Berlin Chamber of Physicians (Ärztammer Berlin). A jointly developed competence-based educational program for fellows of all disciplines was established allowing »protected time for research« during the regular residency. The cooperation allows to integrate research activities into specialist training and thereby avoids a significant extension of the training period. Consequently, (Digital) Clinician Scientists can be credited with up to 18 months of research time as part of their specialist training. Data from the Berlin Chamber of Physicians show that (D)CSP fellows perform as well as colleagues who do full-time clinical work during their board examination. Our shared experiences and cornerstones of the successful cooperation with the Berlin Chamber of Physicians were compiled in an article published in 2024.⁶

The BIH Charité CSP is by far the largest CSP in Germany. The number of participants has grown impressively from eight participants in the pilot program in 2011 to 149 active participants in 06/2025. Figure 2 illustrates the cumulative number of fellows taken up through the CSP

funding lines since 2011. We are proud to announce that within the framework of the second call in 2024 we could include the 500th fellow in our program!

The success of the program also depends heavily on the competitive selection of the most promising candidates. Due to the continued high demand for our program with its different funding lines, we have expanded our pool of reviewers quite substantially in 2024. The reviewers are either members of the Clinician Scientist Board (for reviewing the (J)CSP applications) or the Digital Clinician Board (for reviewing the (J)DCSP applications). We are very grateful to all the members of both the Clinician Scientist Board and the Digital Clinician Scientist Board supporting the program as reviewers during the two-stage selection process twice a year!

Finally, it is worth mentioning that today approximately 9% of all senior Charité residents and 7% of all junior Charité residents receive »protected time« for research through our program. This fits nicely with the recommendations⁸ by the German Science and Humanities Council.

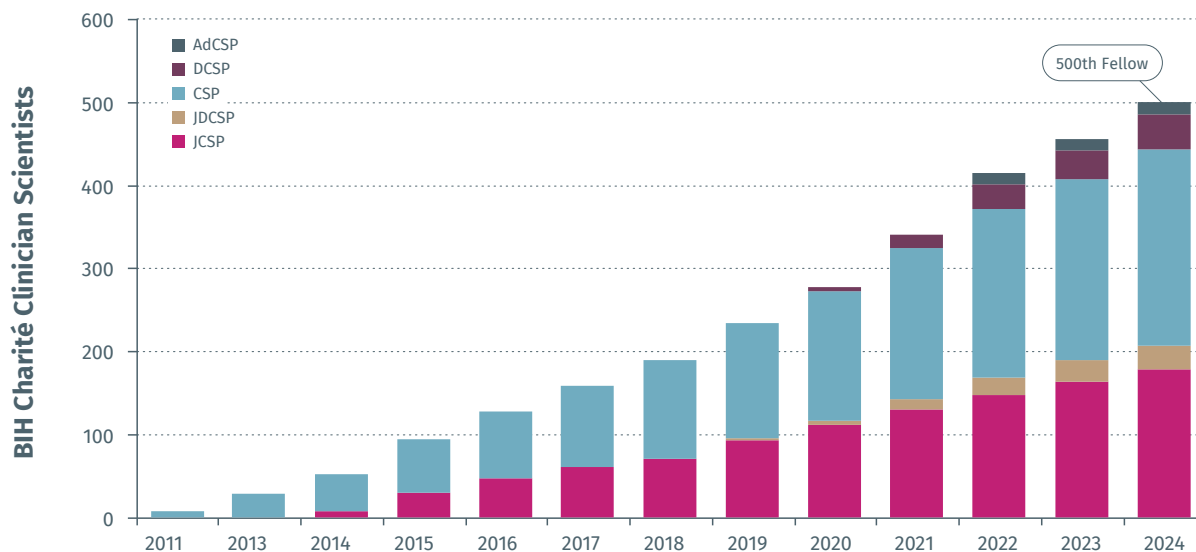


Figure 2: Cumulative number of fellows taken up through the CSP funding lines since 2011.

Interview with Program Directors Prof. Dr. med. Il-Kang Na and Prof. Dr. med. Igor M. Sauer



What does it mean to you to be Director of the BIH Charité (Digital) Clinician Scientist Program? How would you describe your most important responsibilities?

Prof. Dr. med. Il-Kang Na (IKN): When I was first asked to take on the role of Director of the BIH Charité Clinician Scientist Program, I felt a deep sense of responsibility. Supporting so many talented young clinician scientists – helping to create the space, time, and resources they need to grow – felt like a big task, and it still is. But it's also one I care about deeply. Whether it's access to infrastructure, training opportunities, or building meaningful networks, my aim is to make sure they have what they need to thrive. At the same time, I believe this role also comes with a responsibility to lead by example – not only in terms of academic and clinical excellence, but also by standing for values like integrity, curiosity, and balance between work and personal life. These things matter just as much in shaping a sustainable career. Now that I'm in the role, I'm continuously inspired by the people in our program. They are driven, reflective, and often already very clear about the direction they want to take. That makes it an absolute joy to work with them – to be someone they can turn to for support, encouragement, or simply to help clear a path where needed. It's a privilege to be part of their journey.

Prof. Dr. med. Igor M. Sauer (IMS): In addition to providing top-level clinical care, a university hospital has a fundamental commitment to teaching and, in particular, to research. However, due to financial constraints and the increasing focus on efficiency in clinical work, research

is increasingly being neglected – not intentionally, but as a result of practical realities. It is essential, though, that medical research is also driven by physicians. Ensuring this and upholding the role of the physician-scientist is our responsibility. In the context of the digital transformation of medicine and the emergence of entirely new scientific fields – such as the integration of AI and robotics into medical practice – I am especially passionate about promoting and enabling research in these areas. In addition to creating protected time for research, we place particular emphasis on fostering interdisciplinary networking among our fellows. Here, like-minded individuals – physicians who question the status quo, who are curious and exceptionally committed – come together across disciplinary fields. This not only benefits the research activities at Charité but also has very practical advantages: it strengthens interdisciplinary collaboration in day-to-day clinical work. In my view, this is a crucial aspect – not to be underestimated, especially in an institution as large as Charité.

What are your primary program goals for the next 2-3 years?

IKN: One of my key goals is to ensure that future generations of Clinician Scientists continue to benefit from protected time for research and an outstanding scientific environment. We see how much talent and motivation there is among early-career Clinician Scientists, and I would love to expand the number of positions we can offer – because the demand is not only high, it's growing. Another major focus for me is to open up new perspectives for long-term careers in academic medicine. We

need to think beyond the traditional path toward becoming a clinical director and create a broader range of target positions – ones that reflect different strengths, passions, and research profiles. That means helping to shape alternative career trajectories that also offer visibility, recognition, and impact – and that allow people to stay true to their scientific identity while still contributing meaningfully to clinical care.

IMS: The demand for the program is enormous – and it is expected to grow further, as limited personnel budgets in clinical departments leave little room for assigning physicians from clinical duties. Therefore, securing additional funding from foundations and third-party sponsors is essential. This is particularly relevant for the DCSP, where the DFG funding is coming to an end. A key issue in the context of the DCSP is how it will be integrated into or aligned with the ›traditional‹ CSP going forward. At present, the two programs differ somewhat in their conceptual design – for example, the inclusion of a third, digital mentor in the DCSP – as well as in the criteria used to assess track records, such as the recognition of certain conference papers in the field of computer science. However, both programs now operate under the same budget. Therefore, adjustments will be necessary to ensure alignment without compromising the specialized focus and specific needs of projects in digital medicine. However, it is essential that we preserve this ›brand‹ – especially in terms of the program’s public profile and Charité’s visibility, as well as in the context of securing additional third-party funding to support future fellows.

What is your long-term vision for our fellows and for (Digital) Clinician Scientists in general?

IKN: A major milestone we’ve already achieved is the recognition of Clinician Scientists as a distinct professional group – one with its own identity, values, and community. For our fellows, this sense of belonging is essential: they need to feel seen, understood, and supported in a role that bridges two demanding worlds – medicine and science. Looking ahead, one of my long-term goals is to further expand and internationalize the Clinician Scientist network. Nationally, we’re already well connected, but I believe that real growth and inspiration often come from global exchange. That’s why building strong partnerships across Europe and beyond is so important – to enable mobility, collaboration, and new perspectives. Personally, I’ve benefited enormously from international experiences. They’ve shaped my thinking, opened doors, and helped me grow – not just professionally, but also personally. I want our fellows to have

those same opportunities: to connect globally, challenge themselves, and to truly define their own paths in academic medicine.

IMS: I hope they will succeed, and in turn, help ensure that future generations can continue to engage in research – despite the increasing intensity of clinical workloads. Given the significance and success of the program, it may be necessary to fundamentally reconsider how academic medicine is structured and funded. At present, I often have the impression that, due to financial constraints, the true uniqueness of academic medicine is being neglected, as the traditional pillars of Charité – research, teaching, and healing – are increasingly out of balance. I hope that our fellows once they move into positions of leadership and begin shaping policy – will be able to help correct this course.

(Junior) (Digital) Clinician Scientists are at a very challenging stage in their careers. What is your personal career advice for our fellows?

IKN: There’s a quote by Marie Curie that I often come back to: »Life is not easy for any of us. But what of that? We must have perseverance and above all confidence in ourselves. We must believe that we are gifted for something, and that this thing must be attained.« Choosing the path of a Clinician Scientist is already a strong statement – it means you’ve recognized one of your own gifts: the drive to ask meaningful questions and to improve medicine through science. Surround yourself with people who support and challenge you – mentors, peers, collaborators – and don’t be afraid to reach out, to ask for help, or to change direction if needed. Careers in academic medicine aren’t linear, and they don’t have to be. Stay curious, stay persistent, and protect your enthusiasm.

IMS: Network, co-operate with other like-minded people! Form rope teams to climb the mountain! Don’t be protective with your ideas and visions – engage in dialogue. Together, something bigger emerges. Nobody will take anything away from you (and if, contrary to expectations, they do: no problem – you are clever, creative and new ideas will come to you)!

To end with: In three words – how would you describe the BIH Charité Clinician Scientist Community?

IKN: Smart, eager, open-minded

IMS: Curious, collaborative, clever

Our different funding lines under the umbrella »BIH Charité Clinician Scientist Program«

The BIH Charité Clinician Scientist Program is an open-topic program and has continuously developed and differentiated since it started almost 15 years ago. Today, all career phases of Clinician Scientists can be supported with the help of tailored funding lines (see figure 4). All CS funding lines are professionally managed by the BIA, ensuring long-term structural support. Based on the program's fruitful experiences, we have adopted our organizational mechanism to ensure sustainability. Over the last five years, we had around 105 applications per year. The average funding rate across all funding lines is currently 40%, which implicates that not all high-quality applications can be funded. Currently we recruit almost 40 fellows per year (see figure 3).

(Junior) Clinician Scientist Program

Junior Clinician Scientists selected for the program are allotted 20% of their working time as »protected time« for 2 years to exclusively conduct research. For Clinician Scientists, this amounts to 50% for 3 years. Applicants are eligible to apply for the Junior Clinician Scientist Program (JCSP) during the first three years of their residency training. Candidates who have completed more than three years are eligible for the Clinician Scientist Program (CSP). A close clinical and a scientific mentoring with

progress and feedback meetings ensures continuous guidance and support not only for the research project but also for the individual career development. Fellows of the CSP are expected to complete their residency training and their »Habilitation« by the end of the program. New fellows are recruited twice a year following a highly competitive two-stage selection procedure.

(Junior) Digital Clinician Scientist Program

Our (Junior) Digital Clinician Scientist Program ((J)DCSP) is an innovative track for research-active physicians with a special interest in digital medicine which has become increasingly important. In 2018, we successfully secured additional funding from the German Research Foundation (DFG) that also recognized this need. The ((J)DCSP track is set up along the same structure as the »classic« ((J)CSP with regard to the amount and duration of »protected time« for research and the recruiting procedure. However, what sets it apart is a focus on digital medicine and the addition of a digital mentor to the regular mentoring team.

In 2022, we successfully applied for a follow-up funding by the DFG, securing the digital track until at least 2026.

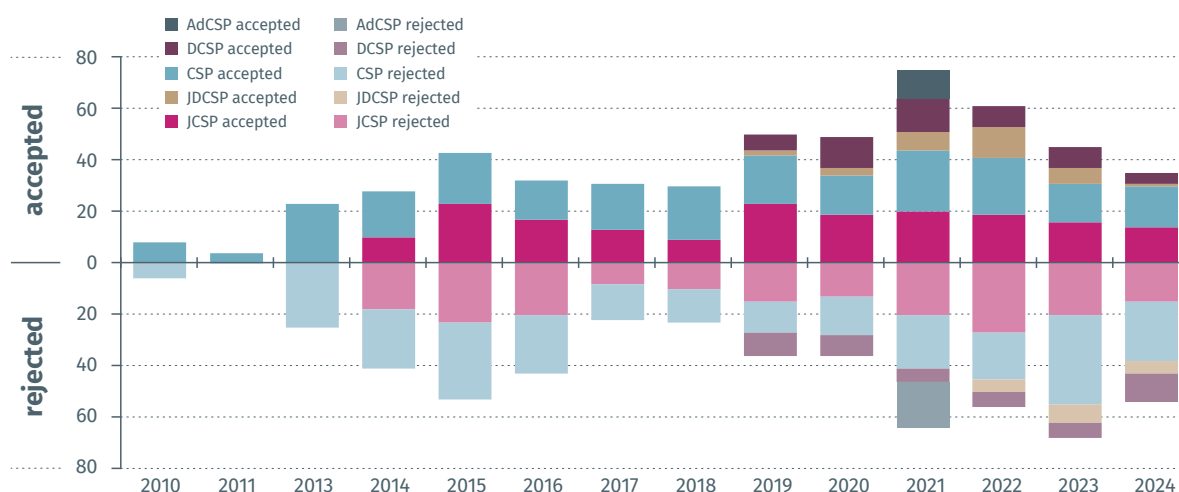


Figure 3: Number of applicants per year accepted (top) and rejected (bottom) per CS funding line

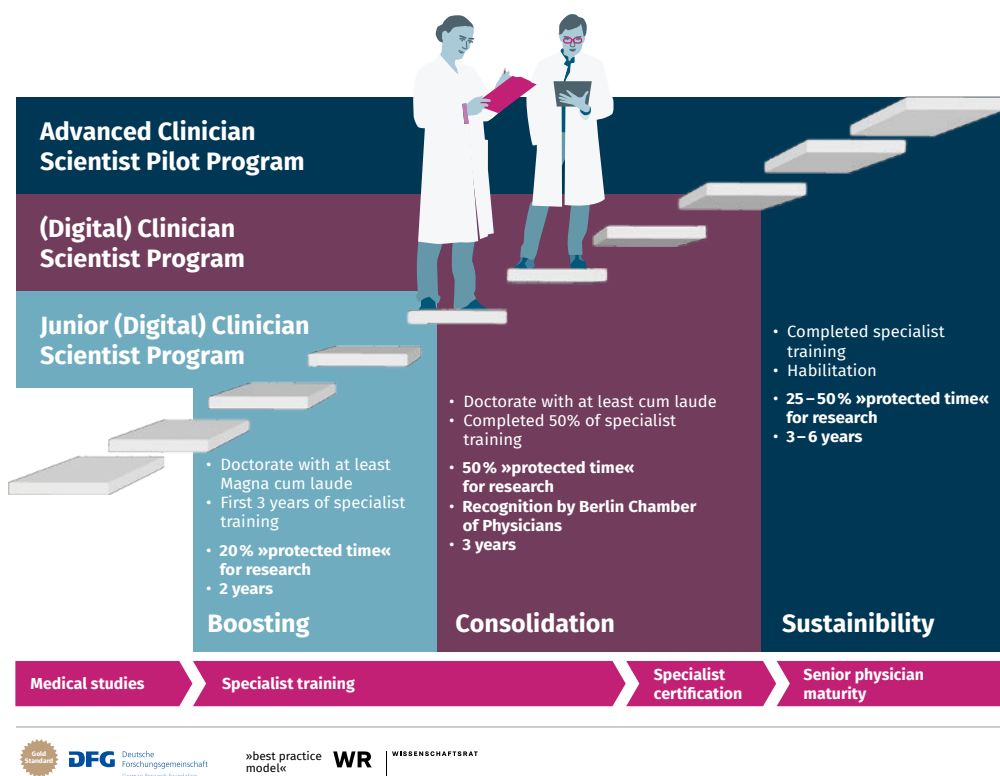


Figure 4: Structured career paths for Junior (Digital) Clinician Scientists, (Digital) Clinician Scientists and Advanced Clinician Scientists spanning different stages of career beginning from medical school.

Moreover, the Stiftung Charité has agreed to fund four Digital Clinician Scientists with a specific focus on digital translation i.e. fellows who are primarily involved in the integration of state-of-the-art digital technologies, in particular artificial intelligence (AI) and extended reality (XR), into everyday clinical care.

Advanced Clinician Scientist Pilotprogram

To close the existing gap in the support of academic career paths after residency, we piloted an Advanced Clinician Scientist Program (AdCSP) in 2020. The overall aims of this funding line are to enable AdCSP fellows establishing an independent clinical and scientific focus area in their departments with the help of (1) »protected time« for research, (2) comprehensive mentoring and (3) a tailored curriculum and to propagate the academic capabilities and structures within the medical departments. In 2024, the BIH Charité AdCSP was evaluated by an internationally renowned external review panel. The reviewers confirmed the outstanding career development of the fellows as well as the beneficial impact on the

scientific translational culture of the departments. It is highly encouraging that in addition to two APL professorships already five of the ten originally funded fellows have obtained full professorships since the program has started and the remaining AdCSP funding could be allocated to qualified follow-up candidates of the same department.

In order to sustain the support of Advanced Clinician Scientists, the program directors of the (D)CSP and the BIA leadership joined forces to apply for the call for Advanced Clinician Scientist Programs in 2024 by the Federal Ministry of Research and Education (BMBF) which since then was restructured and renamed into the Federal Ministry of Research, Technology and Space (BMFTR). Under the running title »proHealth – Promoting and Maintaining Health« we designed a career program for Advanced Clinician Scientists with a focus on prevention, which is a key priority of Charité's Strategy 2030. We are very happy to announce that our application was recommended for funding by the review committee, and we were invited to submit a final funding request to the BMFTR. Depending on the outcome, a next Advanced Clinician Scientist Call might go online in 2026!

CSP »Excellence Track«

In recent years, several fellows of the BIH Charité (D)CSP have acquired excellent junior investigator funding, which gave rise to the idea of an CSP »Excellence Track«. Inclusion in the »Excellence Track« is intended for Principal Investigators of excellent third-party funding (e.g. ERC Starting Grants Program, DFG Emmy Noether Program or BMBF Research Group). Clinician Scientist Fellows of the »Excellence Track« do not have to go through the official two-stage selection process of the BIH Charité (D)CSP as they have already prevailed in a highly competitive external selection process. They can be associated to the (D)CSP and have the same rights and duties as the regularly funded (D)CSP Fellows, including recognition of research time by the Berlin Chamber of Physicians. The only difference is that the research portion of their position is not funded by program funds. Currently there are 3 fellows in the CSP »Excellence Track«.

Living Interdisciplinarity: Building Bridges Across Disciplines and Departments

Our fellows come from a wide variety of clinical and diagnostic disciplines creating an inspiring translational community and fostering transdisciplinary collaboration (cf. Dragun/Huber 2017).⁹ Thus, within the CSP community, interdisciplinarity is not just a phrase but is actually lived through various community-building measures: monthly Jours Fixes, where fellows present their research projects to other fellows and the program leadership. It is also a great opportunity for presenting and discussing new program developments. At regular intervals a retreat is held for all Clinician Scientists and their mentors and clinical or institutional directors. The overall aim of the retreat is to provide a communication platform to discuss both scientific and strategic topics relevant to Charité,

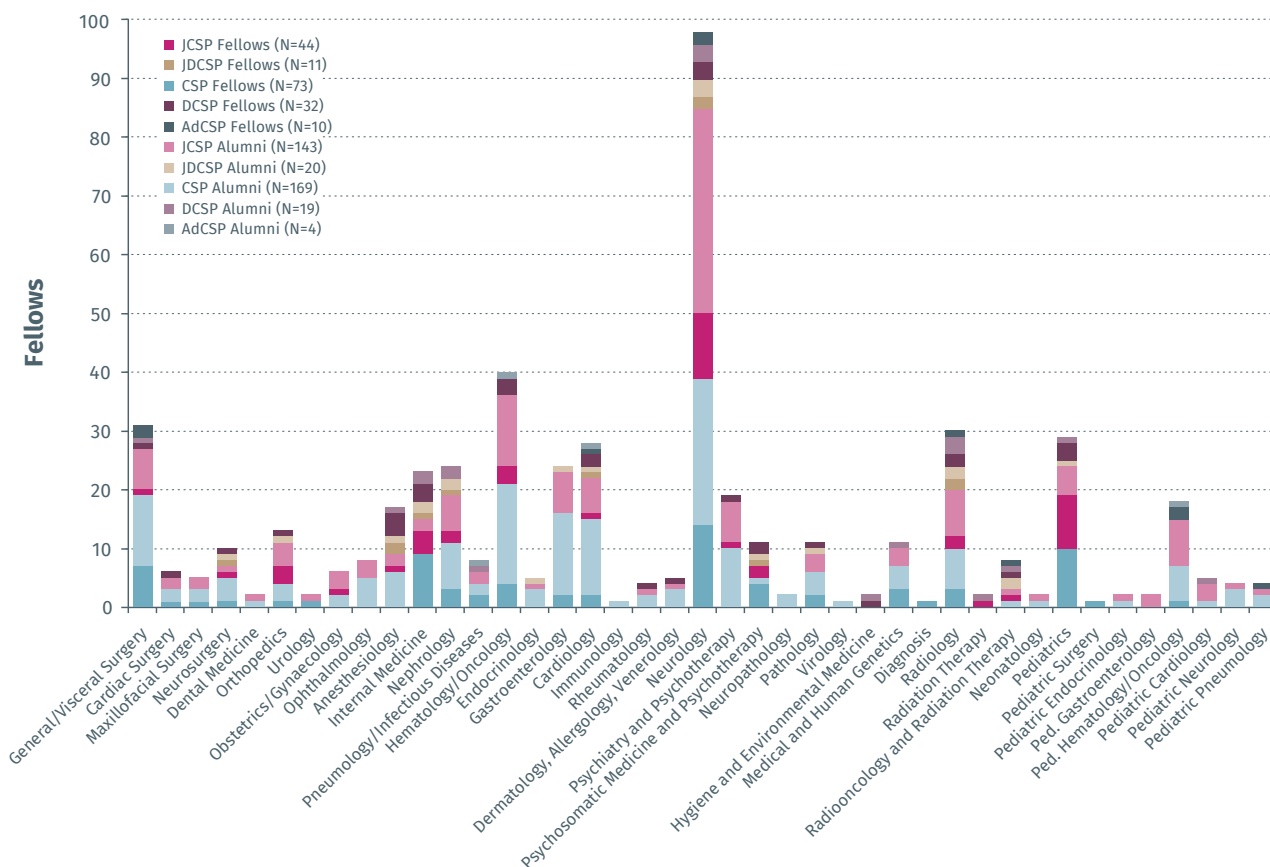


Figure 5: Broad distribution of (J)(D)(Ad)CSP fellows and alumni across clinical disciplines at Charité – Universitätsmedizin Berlin.

BIH and beyond. In addition, a two-day Clinician Scientist Symposium on Translational Medicine is held generally every two years in Berlin, to which fellows can invite internationally renowned experts as speakers. By all these measures the CSP promotes vivid networking and acts as an integrative force within the BIH Charité ecosystem.

As can be seen from the distribution graph (see figure 5), our CSP funding lines bring together fellows from almost all clinical disciplines of Charité. At the same time, we are also gaining increasingly positive experience with integrating topic specific third party-funded CS positions within our BIH Charité CSP, and this approach is proving highly effective in providing additional support for Clinician Scientists with specific focus areas. Examples include rare diseases (Alliance4Rare), pediatrics (German Center for Child and Adolescent Health, DZKI), and dentistry (Prof. Herbert Harnisch & Brigitte Harnisch Foundation).

Equal Opportunities and Family Friendliness as Attractiveness Factors

As a general policy, we actively encourage women to apply and to provide them with the best possible support throughout the whole application and selection process. Among other things, we offer an application and presentation coaching specifically for female candidates. In 2021, we implemented a gender-segregated selection process using identical quality standards. Furthermore, our programs offer flexible working options enabling parental leave for up to 18 months (per child) and part-time work within the program period through flexible budgeting. In addition, we offer on-site childcare for our monthly Jour Fixes and for all our program events. Over the years we have observed a noticeable increase in both the number of applications by women and in the number of female program participants. Today, approximately 48% of our fellows across all funding lines are female which is depicted in table 1.

Expansion of Training Offers and Networking Opportunities

Tailored support for Clinician Scientists in training and education during residency is multifaceted and designed to foster both scientific excellence and professional development. Core components of our competence-based curriculum for the (D)CSP include regular participation in the Jours Fixes, Retreats, and Symposia. In addition to these mandatory elements, the curriculum offers a range of elective modules focusing on methodological

Funding line	Percentage of Women
JCSP	46 %
CSP	36 %
JDCSP	36 %
DCSP	35 %
AdCSP	50 %

Table 1: Percentage of female fellows and alumni across the different funding lines, cumulatively since the start of the program.

expertise, key competencies, and leadership skills. Where possible, participation in existing training and education offers is recognized and credited by our Clinician Scientist Office. However, we are also increasingly active in developing our own courses and offering them to our fellows. Moreover, we cooperate with proven trainers and experts, core facilities and other cooperation partners to offer high-quality tailor-made training opportunities.

In 2024 we could further diversify our training portfolio: in cooperation with the European School of Management and Technology (ESMT) we have piloted an »Entrepreneurship Module« for (Junior) (Digital) Clinician Scientist. And, more recently, we have expanded our training offers in the field of leadership development, introducing new offers addressing topics such as lateral leadership, constructive and sensitive conflict resolution and understanding power structures/dynamics in scientific institutions. We are also increasingly integrating patient and stakeholder perspectives across various formats ensuring that the training remains relevant and grounded in »real-world needs«. All these initiatives aim to equip Clinician Scientists with the diverse skills needed to thrive in academic medicine and beyond.

Finally, during the last two years we have made significant progress towards providing a formalized, regularly updated Transcript of Records for our fellows, enabling them to better track their mandatory credit points for the curriculum and document their corresponding achievements throughout the program duration.

9. Dragun, Duška and Huber, Nathalie: »Schluss mit »Feierabendforschung««. In: Berliner Ärzte, 12/2017, pp.29-31. http://www.berliner-aerzte.net/pdf/bae1712_029.pdf

Ten Years BIA Mobility Fund & Acquisition and Distribution of »Internationalization Grants«

Our BIA Mobility Fund is a vital resource for early-career researchers as they take crucial steps toward professional development and independence. They often rely on external support to access international platforms where they can present their research findings, build professional networks, and foster collaborations that shape the trajectory of their further careers. By enabling participation in international conferences, specialized training programs and work visits, our mobility fund aims at not only providing financial resources but also the encouragement to step into the international scientific community. It allows our fellows to apply for €750 per year for active participation in research and/or career relevant conferences, attending advanced workshops, or collaborating on-site with international partners.

We are proud to announce that our BIA mobility fund celebrates its ten-year anniversary this year! Over the years, this funding instrument has proven to be a valuable

tool to allow (Junior) (Digital) Clinician Scientists to showcase and discuss their project results nationally and internationally and to build up lasting professional networks connections – crucial elements in establishing scientific credibility and independence. Although there was a marked, temporary decrease in the number of applications to the fund during the corona pandemic, the demand for the fund has risen again. To date, we have supported over 300 (!) fellows through the BIA mobility fund, enabling them to attend conferences and institutions by work-visits across the globe (see figure 6).

In 2024, our BIA mobility fund could be extended through the support of the Stiftung Charité by also offering a number of »internationalization grants«, each doted with up to 5.000 Euro. These grants aim at intensifying international cooperation and to work together on joint projects »side by side« – either outbound (scientists in the partner lab abroad) or inbound (visits by external partners to the home lab). The first calls for the »internationalization grants« were launched and the first six grants were awarded to (D)CSP Fellows – the target countries of the first recipients are the US, Vietnam, France, and Belgium and Australia.

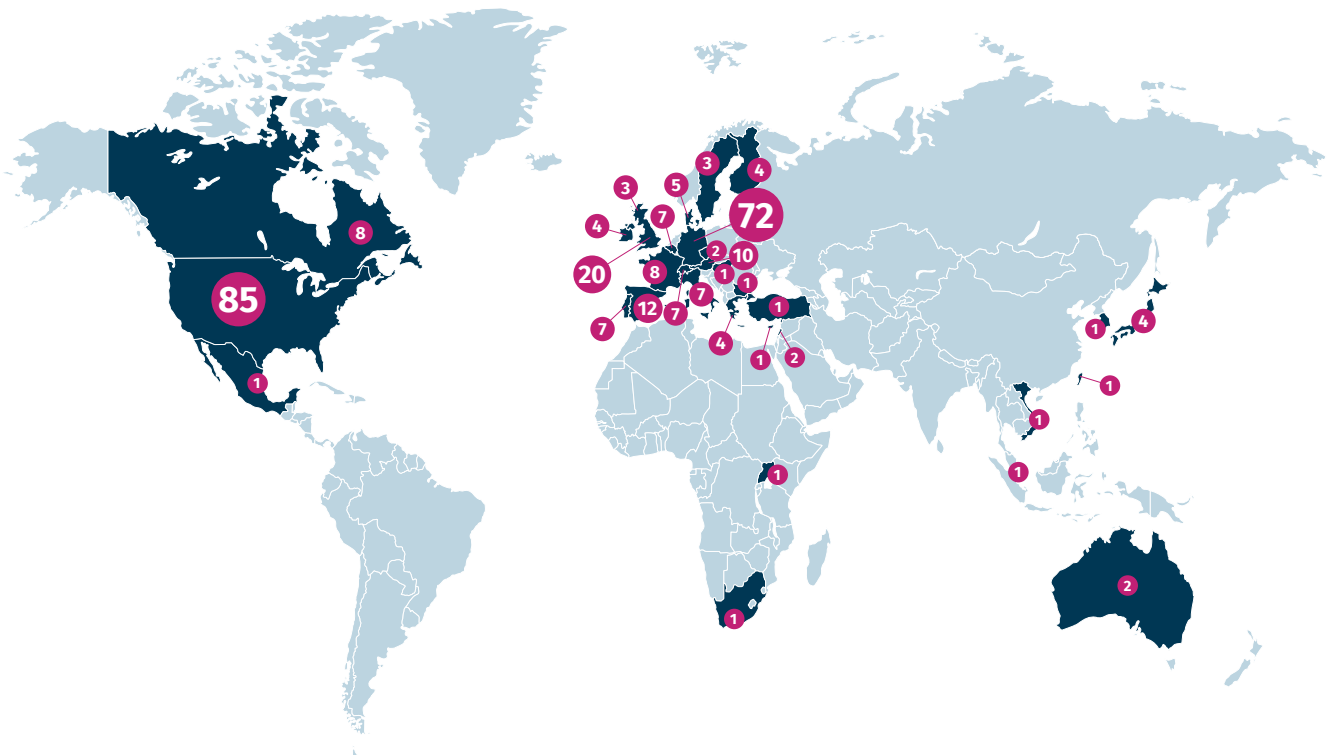


Figure 6: Global distribution of BIA Mobility Grants since 2015.

Clinical and academic achievements of program fellows and alumni

Our fellows and alumni show excellent clinical and academic career progression: 51% of our alumni became specialists, 27% completed their habilitation, and 30% secured leading positions (Source: BIA internal data, reference date 06/2025).

Our alumni are not only successful Clinician Scientists themselves, but they also become the mentors and supervisors of a new generation of fellows, further ingraining the Clinician Scientist culture. A significant proportion of our fellows obtain professorships – some of them already during their (J)(D)CSP funding. Overall, we can currently pride ourselves on 31 professors among our fellows and alumni (17 W2 professorships and 14 W3 professorships) (see table 2).

Position/Qualification	Count (Fellows & Alumni)
Medical specialists	256
Clinical Leadership Positions (Senior Physician and above)	66
Group Leaders	53
»Habilitations«	138
ERC Grants	6
W2 Professorships	17
W3 Professorships	14

Table 2: Career progression outcomes of (Ad)(J)(D)CSP fellows and alumni. N=504.

High Return on Investment: Promoting Clinician Scientists Pays Off

Another impressive program outcome result is the cumulative amount of third-party funding raised by alumni and (Junior) (Digital) (Advanced) Clinician Scientists of 106 million Euros (see figure 7). This roughly corresponds to a »return on investment« of two to one underlining once again the effectiveness of the program and the excellence of its fellows.

CSP Alumni Network & Career Tracking

We are delighted that our BIH Charité Clinician Scientist community already boasts over 350 alumni and is constantly growing! For this reason, alumni work is becoming more and more important and is considered as a central component of our program activities. In 2024, we intensified the communication with our alumni and we launched a biannual alumni newsletter, providing for example information on news from the Clinician Scientist office, events, and alumni success stories. Moreover, we created a BIH Charité Clinician Scientist Alumni Network. This network aims to create a platform where alumni can share their personal experiences and expertise, their scientific and clinical knowledge and career recommendations and, thus, benefit from each other. For this purpose, we use the Charité Alumni Portal and have implemented a specific »Clinician Scientist Program alumni group« on this platform. This Alumni Portal acts as communication tool, e.g. for the alumni newsletter and for posting information on relevant grants, events, and news from BIH or BIA. If you are an (upcoming) alumna/alumnus and not registered yet – take advantage of this opportunity now and browse to the following website: www.alumniportal.charite.de/login. In addition, a logo was developed for the alumni network to make membership in the alumni network visible, and a flyer about alumni work was published (see figure 8).

As you may have noticed since our Program Book 2023, we have grown so much that we are only listing the profiles of our current fellows. Instead of alumni profile pages, we provide an index with all our alumni over

the different funding lines so far, and an link to previous online editions of this book, where you can find their profiles in more detail. In addition, all our alumni are visible on our website through the »fellows and alumni finder«.¹⁰ We warmly encourage you to explore this online tool!

In order to systematically monitor the careers of our alumni we have been developing a career tracking tool that can be used in meta-research analysis. It will allow the analysis of correlations between the careers of young scientists at Charité and the study of correlations leading to successful careers in academic medicine. It will also be a valuable tool to determine when and why young scientists most often leave academic careers and what alternative career paths they take. With this deeper understanding of patterns for Clinician Scientist career trajectories we can further refine and optimize our funding lines and support measures on the one hand. On the other hand we can make an evidence-based, meaningful contribution to the current debate about tailored and attractive target positions for Clinician Scientists (see Na et al. 2024).¹¹ We are more than happy that we can

now contribute our Berlin experience to the nationwide ECLIPSE project. This is a collaborative project between us, the EKFS and the MFT for the first Germany-wide online cross-sectional survey of CSP fellows and alumni. In addition to the online survey, in-depth interviews and the establishment of a central database are planned. After the completion of the ECLIPSE project, the database is planned to be continued as a »National Clinician Scientist Cohort« (NCSC) by the BIA. The NCSC will serve as an empirical basis for local and national longitudinal and further cross-sectional studies and provide prospective, evidence-based recommendations for action to (science) policymakers and potential funding bodies.

10. www.bihealth.org/en/translation/innovation-enabler/academy/fellows-and-alumni/translate-to-englisch-alumni

11. Na, Il-Kang, Meij, Iwan, Huber, Nathalie: »Clinician Scientist – quo vadis? Brückenbauer für Forschung, Patientenversorgung und Lehre«. In: Forschung & Lehre 10/2024.

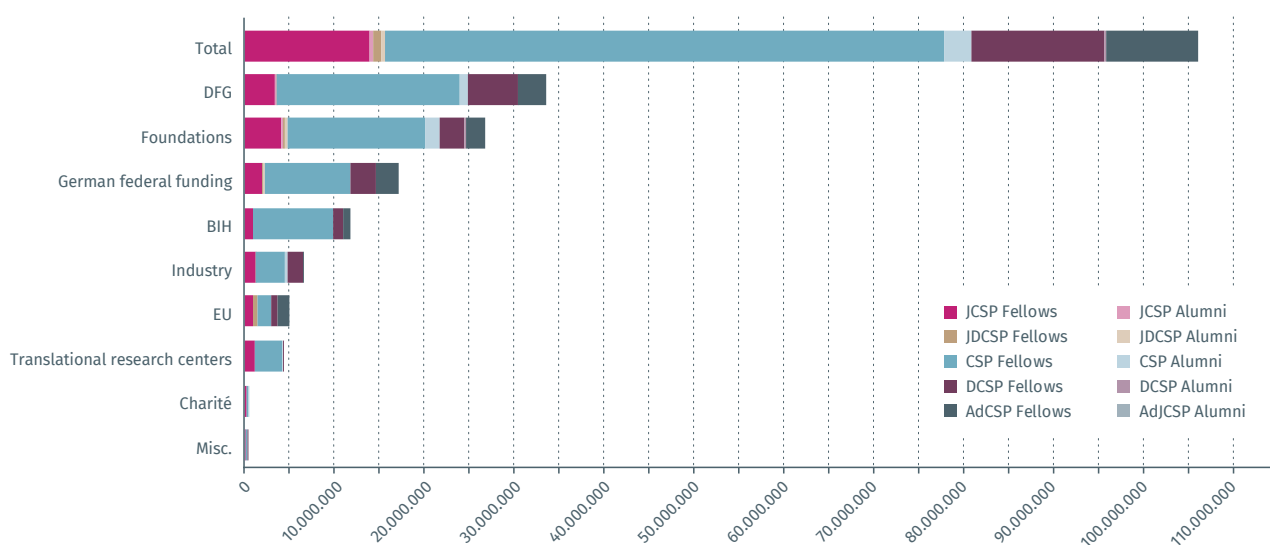


Figure 7: Third-party and intramural funding raised in € by BIH Charité (Junior) (Digital) (Advanced) Clinician Scientists up to 06/2025



Figure 8: Photograph of the certificate award ceremony 2024 and the new Alumni flyer.

Résumé and Outlook

Looking ahead, the continued development and success of our BIH Charité Clinician Scientist Program remains central to our mission of fostering translational talents. Building on the solid foundation of almost 15 years, we are committed to deepening our national and international outreach by integrating valuable input from beyond the Berlin ecosystem. Our role in the broader scientific and policy-making landscape will remain active and visible, particularly through our participation in national committees focusing on career structures and pathways for Clinician Scientists. We also see potential for further internationalization efforts and will explore new opportunities and exchange formats to further increase the reach and relevance of our program. This broader exchange of perspectives will be vital in enriching the program and ensuring its alignment with evolving scientific and clinical priorities.

An important strategic and science policy area of action will be our alumni work, which we aim to expand significantly in the coming years to build a stronger professional community, and both promote and analyze long-term impact of Clinician Scientist funding. Strengthening our alumni network will not only sustain a vibrant professional community with a translational mindset but also offer current fellows enhanced opportunities for mentorship and career development opportunities – for example regarding attractive target positions. At the same time, we have started with the implementation of the ECLiPSE project, a key initiative for a nationwide data collection on Clinician Scientists that will not only bring

valuable new insights into the career trajectories of Clinician Scientists but also enable an even more evidence-based refinement of program structures, as the first meta-analyses come to fruition.

Securing long-term sustainability is a continuous goal, and we will persist in our efforts to acquire third-party funding for the program and its funding lines in addition to the base funding by Charité and BIH. We are particularly hopeful that our above-mentioned Advanced Clinician Scientist application will successfully take the final hurdle, and we can secure funding for it. This will allow us to support further experienced fellows and strengthen the bridge between research and clinical leadership.

All our efforts are united by one vision: to build a resilient, dynamic, and impactful program that empowers Clinician Scientists, benefits patients and elevates the role of translational research within medicine.

Finally, we extend our sincere thanks to all those who make this work possible: our dedicated supporters and funders, the program directors and their deputies, the Berlin Chamber of Physicians, the members of the (Digital) Clinician Scientist Boards, and the members of the newly implemented CSP Steering Committee. Their unrestricted commitment to promoting Clinician Scientists, collaborative spirit, and extraordinary engagement continue to shape and strengthen our program's future.

The background of the entire page is a repeating pattern of organic, cell-like shapes. These shapes are light blue and white, with thin, darker blue lines separating them, creating a textured, biological feel.

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- › Nuclear Medicine
- › Pediatric Oncology
- › Differentiated Thyroid Cancer

Whole-Body ¹³¹I Imaging in Children with Thyroid Cancer with Undetectable Thyroglobulin. Benefit or Relict?

In Germany, pediatric patients with differentiated thyroid cancer (DTC) are treated with thyroidectomy followed by adjuvant ¹³¹I radioiodine (RAI) treatment with an activity of 25 to 50 MBq per kg body weight in carcinomas larger than 1 cm according to current guidelines (1). If 6 months after RAI treatment ultrasound and an undetectable tumor marker thyroglobulin (TG) under thyroid hormone replacement indicate absence of remnant persistent disease, the child undergoes diagnostic whole-body ¹³¹I imaging (DxWBS) with 5 MBq/kg. This DxWBS is accompanied by measurement of the TSH-stimulated (»true«) TG level. In a 10-year-old boy with 35 kg, DxWBS results in an estimated effective dose of 107 mSv, which is equivalent to approx. 50 years of environmental radiation exposure in Berlin (2).

One might consider measuring the stimulated TG level to confirm absence of disease without administering ¹³¹I to perform additional DxWBS. This would obviate the

inpatient stay and radiation exposure but is currently not recommended because sufficient evidence to omit the DxWBS is missing.

The aim of my project is to systematically evaluate the value of DxWBS in addition to stimulated TG levels and ultrasound of the neck for determining the presence or absence of tumor disease after RAI therapy that would require further treatment. The working hypothesis is that the diagnostic accuracy of stimulated TG to detect persistent disease that would require therapy is non-inferior to DxWBS. That would mean that DxWBS is not beneficial in children with DTC and undetectable levels of stimulated TG after initial RAI treatment. To thoroughly test this hypothesis, we aim to perform two retrospective studies and subsequently design a prospective confirmatory multicenter trial.

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- › Deep Brain Stimulation
- › Parkinson's Disease
- › Patterned Stimulation

Translating Burst Cycling Deep Brain Stimulation to Patients with Parkinson's Disease

Subthalamic (STN) deep brain stimulation (DBS) is an efficient treatment option for patients with advanced Parkinson's disease (PD), improving motor symptoms such as tremor, bradykinesia, and rigidity. However, conventional DBS (cDBS) has limitations, including inconsistent effects on disabling axial motor symptoms like gait disturbances. Moreover, stimulation effects are only short-lived, and symptoms re-emerge shortly after stimulation is turned off. Preclinical studies in a PD mouse model have demonstrated that a specific burst cycling DBS (bDBS) paradigm can induce long-lasting motor improvements, especially on gait, potentially through mechanisms of synaptic plasticity. Prolonged stimulation effects would contribute to higher energy-efficiency, prolonged battery life as well as reduced stimulation-induced side effects. This study aims to evaluate the therapeutic effects of a newly identified bDBS paradigm on motor symptoms in 20 PD patients treated with STN DBS. We will compare the efficacy and longevity of bDBS versus cDBS in improving bradykinesia, rigidity, and gait performance with and without dopaminergic medication.

The primary outcomes will include reductions in Unified Parkinson's Disease Rating Scale (UPDRS-III) scores, objective measures of motor symptoms, including gait performance and prolonged therapeutic effects post-stimulation. Electrophysiological biomarkers, such as beta oscillations, will be analyzed using local field potentials (LFPs) recorded from the DBS leads during and after stimulation. This study seeks to provide clinical insights into the potential of STN bDBS to offer prolonged and improved stimulation effects on PD motor symptoms, paving the way for novel avenues for optimizing DBS treatment strategies in PD patients.

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› Nephrology
› Immunology
› Rheumatology

Role of TREM2+ Macrophages in Lupus Nephritis

Systemic lupus erythematosus (SLE) affects primarily women of childbearing age, and, as a chronic disease without definitive treatment, poses a significant socio-economic burden. An important cause of morbidity and mortality in SLE is lupus nephritis (LN). LN is an immune complex-mediated glomerulonephritis: immune complexes deposit in the glomerulus and can lead to an inflammatory cascade that results in organ damage and loss of kidney function. Even though autoantibodies are required in the pathogenesis of the disease, autoantibodies alone are not sufficient for the development of autoimmune organ damage. A significant number of patients with autoantibodies develop mild or no nephritis while others develop a rapidly progressing nephritis leading to dialysis, suggesting the existence of yet unidentified factors that amplify organ damage. Using the well-established NZB/W F1 lupus mouse model and whole kidney scRNA-seq, we identified clusters of macrophages in LN feature a phenotype marked by the expression of the macrophage-specific receptor Trem2 (Biniaris et al, Nature, 2024). This was of high interest

because Trem2 has been previously associated with disease-associated macrophages (DAM) in the liver, lung, or brain and blocking or stimulating Trem2 has been found to regulate disease outcomes. Expression of Trem2 by kidney DAM had not been previously described and the role of Trem2 in lupus nephritis remains unknown. Bioinformatic analysis of our dataset predicted that activated intermediate parietal epithelial cells could interact with DAM and promote a pro-fibrotic program of a subset of parietal epithelial cells. This finding is reminiscent of liver Trem2+ macrophages being part of a »fibrotic niche«, as well as of a pulmonary subset of Trem2+ macrophages associated with fibrosis. However, Trem2+ macrophages do not necessarily have a detrimental role in disease processes: in the context of non-alcoholic fatty liver disease, Trem2+ macrophages were involved in the removal of lipids from the liver and loss of Trem2 led to a pro-inflammatory response, which actually aggravated fibrosis. The role of Trem2+ macrophages in SLE or in LN has not been investigated.

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- › Cardiology
- › Palliative Care
- › Unmet Needs
- › Patient and Stakeholder Engagement

Leveraging Choice Experiments for the Patient-Centered Design of a Palliative Care Service For Persons with Heart Failure

Persons with heart failure experience severe symptoms, poor quality of life, and almost 50% die within 5 years after initial diagnosis. Yet, there is no established model for providing palliative care to persons with heart failure. Palliative care can include numerous elements (e.g., physical symptom management, psychological support, advance care planning, caregiver support) and can be provided in different ways (e.g., specialist consult during hospitalization, nurse-practitioner-based clinic visits, telehealth services). In order to design evidence-based palliative care services with maximum reach and value for HF patients, care delivery models must be tailored to the needs and preferences of patients. Thus, rigorous and innovative research to better understand the palliative care needs of persons with heart failure is needed.

Discrete choice experiments (DCE) offer a novel and sophisticated quantitative method to study the preferences of HF patients regarding palliative care services. Grounded in behavioral economic theory, the DCE methodology breaks down a hypothetical service (in this case, a hypothetical palliative service for HF patients) into key

modifiable attributes (e.g., location, providers involved, services provided) which have different levels (e.g., location: at the bedside; in an outpatient clinic; in the patient's home). During the choice experiment, study participants are repeatedly presented with two different combinations of service attributes and asked to choose between the two. Based on their responses, the relative importance of the various modifiable service attributes can be quantitatively estimated using advanced statistical computation. DCE allows us to rigorously quantify the needs and preferences of patients regarding a hypothetical palliative care service, calculate trade-offs between service attributes, and elicit patients' preferences about the components of a palliative care service.

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- › Monogenic kidney disease
- › Renal virus delivery
- › Base editing



Geschwister Mangelndorff Stiftung

Characterization of Adeno Associated Virus Delivery for Renal Genome Editing

The number of patients with CKD is on the rise and represents a global health burden. To date, >600 monogenic causes of CKD are known. While being individually rare diseases (<1 out of 2000 individuals affected), taken together, they are significant contributors to both adult and pediatric disease etiology. Mendelian mutations account for up to 50% of childhood-onset and 10–34% of adult CKD cases. Dialysis and transplantation are the only treatment options for end-stage kidney disease (ESKD), but neither is curative.

CRISPR/Cas systems have emerged as a promising genome-editing technology for the treatment of various monogenic diseases in mice, non-human primates, and humans, but its potential for monogenic kidney diseases remains unexplored.

For successful in vivo genome editing, the genetic material must be delivered to the tissue and cell type of interest. Adeno-associated virus (AAV) has emerged as a safe and effective delivery vehicle for in vivo genome editing and AAV-based therapies have already been approved.

However, for the kidney overall transduction efficiencies via the intravenous route are low. Recent publications indicate that local administration of AAVs into renal artery, vein or pelvis may be beneficial, but systematic analyses remain limited.

Therefore, the aim of this project is a quantitative evaluation of renal transduction efficiency of different recombinant AAV serotypes in vivo. The AAVs will be injected into the tail vein, the renal artery, renal vein or renal pelvis of 5-week-old Ai14 reporter mice leading to expression of a fluorescence protein in case of successful transduction. To evaluate overall renal transduction efficacy as well as specific transduction in distinct renal cell types, I will use flow cytometry, immunofluorescence and confocal microscopy. Here, I will perform co-stainings with antibodies defining distinct renal cell types.

Overall, this project aims to identify an effective combination of AAV serotype and injection route for renal transduction to provide a foundation for future in vivo genome editing experiments.

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- > Autoinflammation
- > Pediatric rheumatology
- > Immunology
- > Autophagy

Analysis of Autophagy in Inflammatory Diseases and its Potential as a Therapeutic Target

Autoinflammatory disorders represent a group of monogenic diseases that cause systemic inflammation and are typically driven by a well-defined group of cytokines, e.g. interleukin (IL) 1-beta or type I interferons (IFN). They are rare but often mirror features of more common inflammatory diseases: E.g. patients with Aicardi-Goutières syndrome (AGS) often mimic symptoms of systemic lupus erythematosus (SLE) or dermatomyositis (DM), and share the upregulation in type I interferon signaling as an underlying immunological phenotype. Despite recent advances in the understanding of the driving pathomechanisms as well as the advent of targeted therapies, a large number of these patients remains refractory to treatment. A more thorough understanding of the processes contributing to the inflammatory phenotype is therefore needed to improve quality of life and reduce morbidity and mortality in patients suffering from inflammatory diseases.

Autophagy is a multifaceted process that ensures cellular homeostasis. The regulation of autophagy and inflammation is closely intertwined: Stimulation of pattern recognition receptors in the context of infection activates autophagy. Autophagy itself is part of the microbial

defense and in turn induces genes involved in the anti-viral immune response, causing an increase in type I IFN production. On the other hand, loss of autophagy-related genes causes a hyperinflammatory phenotype in myeloid cells.

We therefore propose that dysregulation of the autophagic pathway is contributing to the inflammatory phenotype of patients with inflammatory diseases.

In this project, we aim to verify this hypothesis in a cohort of more than 200 patients. We are analyzing autophagy at the transcriptional level in the patients' peripheral blood mononuclear cells using single-cell RNA sequencing. Informed by the results of these experiments, we are examining autophagic activity in cells from patients with inflammatory diseases with transcriptional changes in autophagy. Finally, we seek to determine the contribution of autophagy to the inflammatory phenotype of the underlying diseases.

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› Genetics and Genomics of
Cardiovascular Diseases

Heart Failure with Preserved EF – Single-Nucleus RNA Sequencing to Uncover Molecular Signatures & Cellular Networks

Heart failure with preserved ejection fraction (HFpEF) accounts for more than half of all heart failure cases and continues to rise in prevalence, predominantly affecting older individuals and women. It is associated with a high burden of comorbidities, and in contrast to heart failure with reduced ejection fraction, established pharmacological therapies, such as beta-blockers or angiotensin receptor blockers, have failed to improve clinical outcomes. As a result, current management is limited to treating comorbidities, highlighting the need for therapeutic strategies targeting disease mechanisms and cardiac remodeling.

Previous research has been limited by bulk RNA sequencing, which averages gene expression across a mixed cell population, masking variations and rare cell populations crucial for understanding complex diseases. Recently, single-nucleus RNA sequencing (snRNA seq) has emerged as a revolutionary technology. Unlike bulk RNA sequencing, snRNA seq provides a detailed view of gene activity at the individual cell level, enabling the identification of rare populations and subtle gene expression variations.

This project aims to elucidate the cellular and molecular basis of HFpEF, a complex condition with limited therapeutic options. We combine single-nucleus RNA sequencing, spatial transcriptomics, and CellChat to achieve unprecedented resolution of gene expression in human HFpEF samples. Single-nucleus transcriptomics reveals cellular heterogeneity, identifying subpopulations comprising as little as 1% of the main population. CellChat maps communication networks between cells, exposing interactions otherwise hidden. Spatial transcriptomics adds a crucial spatial dimension, revealing how these interactions are organized. Together, this multidimensional approach provides a comprehensive understanding of the cellular microenvironment and pathophysiological mechanisms in HFpEF. The rarity and exceptional value of our human endomyocardial biopsies further highlight the groundbreaking nature of our work.

Our goal is to define HFpEF as a distinct molecular disease entity by identifying cell type-specific transcriptional signatures and pathological pathways. Ultimately, we aim to create a comprehensive cell atlas of the human heart in this condition, advancing the understanding and potential treatment of HFpEF.

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Fields of Research

› Hematology
› Aging

Long-Term Molecular Effects of Tyrosine Kinase Inhibitors in CML-Patients

Tyrosine kinase inhibitors (TKI) have revolutionized the treatment of chronic myeloid leukemia (CML). CML has become a disease that can be controlled in almost all patients and thus treatment tolerability and late effects of CML treatment are major concerns of patients, physicians, and the socio-economic system. Analyses of late/side effects of long-term TKI treatment are currently limited to observations from clinical trials and comprise cardiovascular, metabolic, neuropsychological and musculoskeletal disease and resemble health problems associated with aging. To systematically investigate if TKI therapies can lead to premature aging in CML patients we seek to quantify selected molecular hallmarks of aging. To that end we will characterize epigenetic changes using comprehensive methylation analyses in CML patients under TKI-therapy and in TFR; assess (immune)-senescence and inflammaging through immune-profiling in CML patients under TKI therapy and in TFR; and create a comprehensive clinical patient registry for integrative analysis of molecular and clinical data.

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Fields of Research

- › Stroke
- › Intracerebral Hemorrhage
- › Cardiovascular Diseases

Effects of PCSK9 and its Inhibition on Ischemic Stroke Susceptibility

In Germany, around 300,000 people suffer a localized cerebral perfusion deficiency every year. As a result of a stroke, 30% of patients die. Elevated serum levels of low-density lipoprotein cholesterol (LDL-c), for whose regulation PCSK9 (proprotein convertase subtilisin/kexin type 9) plays a key role, is an important, modifiable risk factor for cardiovascular disease. For the prevention of cardiovascular diseases, such as ischemic stroke, the drug-based reduction of elevated LDL-c serum levels using monoclonal antibodies against PCSK9 has proven to be an effective therapy.

In contrast to the known preventive function of PCSK9 inhibition for ischemic stroke, effects on stroke susceptibility, i.e. the outcome after suffering a stroke, are not yet known. Since 2016, we have been investigating these effects of PCSK9 and its inhibition. In a mouse model that closely mimics the human risk population for cardiovascular disease, we are analyzing whether and how PCSK9 and its inhibition influence the outcome after an experimental ischemic stroke.

Furthermore, we investigate the pathomechanisms underlying the phenotype and use a clinical cohort of

stroke patients to investigate whether the effect observed in the mouse model can be replicated in humans.

Preclinical investigation of the underlying pathomechanisms: At different time points after induction of experimental ischemic stroke, we investigate potential pathomechanisms by which PCSK9 or its inhibition might influence stroke outcome. We focus on a) cerebral blood flow and microthrombi, b) blood-brain barrier function and c) inflammatory processes and the immune response as well as oxidative stress. For deeper mechanistic insights with high spatial resolution at the cellular and transcriptional level, spatial transcriptomics analyses are also performed.

Translational studies in a clinical stroke cohort: To assess the translational potential of our preclinical data, we are investigating a cohort of patients with ischemic stroke. Serum samples from the cohort are used to measure PCSK9 levels and correlate them with functional outcome, cardiovascular endpoints and ischemic stroke lesion size.

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Fields of Research

› Migraine Pathophysiology
› Cardiovascular Risk in Migraine

Effects of Oxytocin Receptor Antagonization in Migraine: A Randomized, Double-Blind, Placebo-Controlled Human Provocation Study

This study investigates the complex relationship between hormones, particularly oxytocin, and migraine, a condition affecting approximately 15% of the global population, predominantly women. Hormonal fluctuations, notably during the premenstrual period, have been implicated in triggering migraine attacks, with a historical focus on estrogen. However, recent attention has shifted to alternative hormones, with oxytocin as a prime candidate. Oxytocin's multifaceted functions, including analgesic effects and modulation of the trigeminovascular system, position it as a potential protective factor in migraine pathophysiology. To explore the role of oxytocin in migraine pathophysiology, the placebo-controlled, double-blind, randomized human provocation study employs an oxytocin receptor antagonist as the provocation substance to assess the effects of oxytocin inhibition as a potential migraine trigger. We hypothesize that an inhibited effect of oxytocin due to receptor antagonization will lead to migraine-like attacks in women

with migraine. Beyond investigating oxytocin receptor antagonization as a trigger, the study examines the impact of oxytocin receptor blockade on vascular parameters and migraine biomarkers, such as calcitonin gene-related peptide (CGRP). Male migraine patients serve as a control group to evaluate sex-specific effects, while age-matched healthy women without migraine provide a second control group to determine whether underlying migraine pathophysiology influences the outcomes of oxytocin receptor antagonism.

Overall, this study aims to enhance the understanding of hormone-dependent mechanisms in migraine pathophysiology and to explore novel therapeutic strategies targeting the oxytocin pathway.

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- › Palliative Psychiatry
- › Advance Directives
- › Medical Ethics

»Palliative Care« for Treatment-Resistant Depression: a Mixed-Methods Study on the Perspectives of Patients, Their Family Members, and Healthcare Providers

Historically, palliative care has been directed towards individuals with life-threatening somatic illnesses like cancer. In contrast, despite the frequently persistent and potentially life-threatening character of psychiatric disorders, an explicit palliative care approach does not yet exist in the field of psychiatry. In recent times, there have been suggestions to explore such an approach for patients diagnosed with severe and persistent mental illness. However, a palliative care approach in psychiatry has sparked debates. While some believe that integrating a palliative strategy into the realm of psychiatry holds promise for enhancing the quality of care for individuals coping with severe and persistent mental illness by focusing on alleviating the suffering, others fear that such an approach is faced with challenges, including difficulties with decision-making capacity and prognostication in severe and persistent mental illness. To date, there has been limited understanding of the perspectives held by patients, family members, and healthcare pro-

viders regarding a palliative care approach in psychiatry, particularly for severe and persistent depressive disorder. This research project utilizes both qualitative and quantitative methods to analyse attitudes towards a palliative care approach in psychiatry.

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- › Neurophysiology
- › Naturalistic Monitoring

Naturalistic Monitoring of Dyskinesia to Adapt Closed-Loop Deep Brain

This project aims to utilize technological developments in home monitoring to advance the understanding and treatment of dyskinesia in Parkinson's disease. People with Parkinson's disease are typically treated with levodopa to improve their movement. As a side effect with disease progression, most patients develop involuntary abnormal movements, called levodopa induced dyskinesia. Dyskinesia are often an inclusion criteria for device-aided therapies, such as deep brain stimulation (DBS). In this project, we aim to decode dyskinesia presence and severity, based on neurophysiological biomarkers recorded with sensing-enabled DBS systems. We utilize smartphone-based ecological patient reported outcomes (EMA) und wearable movements sensors to monitor Parkinson patients at home. These data are combined with the neurophysiological biomarkers, recorded at home. The development of dyskinesia specific biomarkers in a naturalistic, at home, environment, will enable us to automatically decode dyskinesia in the

future. This again, will help future closed-loop DBS systems to tailor the amount of stimulation applied to the individual and real-time clinical state of the patient.

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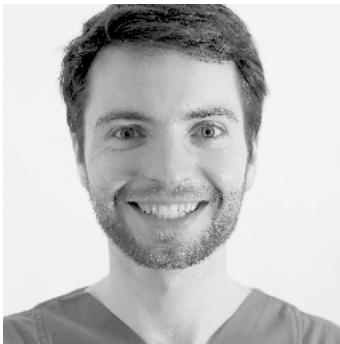
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- › Computational Biology

Repressive Chromatin on Extrachromosomal DNA

In recent years, extrachromosomal DNA (ecDNA) has emerged as a significant contributor to cancer progression across various cancer types. ecDNAs are small, circular chromatin structures that carry oncogenes, with dozens of copies typically present in each cancer cell, leading to oncogene amplification and overexpression. Recently, we and others have characterized the chromatin landscape of ecDNA. However, the role of repressive chromatin domains on ecDNA remains poorly understood. This project aims to combine computational analysis with experimental approaches to investigate the prevalence and functional significance of these domains.

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› Metabolic Imaging

Characterising Metabolic and Biomechanical Properties of HCC Using Novel Imaging Methods

Hepatocellular carcinoma (HCC) is the most common primary liver cancer and the third leading cause of cancer-related deaths worldwide. Over 80% of HCC cases arise in fibrotic livers, where increased deposition of fibrous collagens and fibronectin creates a rigid tumour environment, impeding the differentiation of primary hepatocytes and promoting proliferation. Additionally, angiogenesis-induced alterations in vascular and interstitial fluid pressure influence force-sensitive signalling mechanisms that drive HCC progression. Metabolic derangement, characterised by increased glucose uptake and aerobic glycolysis, is a hallmark of cancer and is commonly observed in HCC.

We propose a mechanometabolic interplay during HCC progression, where structural changes in HCC and the tumour microenvironment (TME) affect tumour metabolism through mechano-transduction, converting mechanical cues into biochemical outputs. These structural and metabolic changes are dynamic indicators of cancer formation and progression. Currently, no imaging technique can simultaneously visualise the structural and metabolic characteristics of HCC and its environment

in vivo, which is crucial for early diagnosis and predicting treatment responses.

To address this gap, we plan to use advanced multiparametric PET-MR imaging methodologies. While FDG-PET focuses on early glycolysis stages, we will introduce non-invasive MRI methods such as chemical exchange saturation transfer (CEST) and MR-spectroscopy (MRS) to evaluate metabolic processes in both the tumour and liver. CEST will provide insights into the metabolic flow of glycogen, glutamate, or glucose, and MRS will assess the acidity of the tumour and its microenvironment. We will also investigate the mechanical properties of the tumour and its ECM in vivo using multifrequency magnetic resonance elastography (mMRE).

Our goal is to create a comprehensive mechanical and metabolic profile of the tumour and its microenvironment through a longitudinal, multiparametric, quantitative imaging approach. This strategy aims to facilitate integration into routine clinical practice to offer a more accurate detection method for tumour recurrence and early treatment response.

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- › Osteoarthritis
- › Regenerative Medicine
- › Orthopaedics
- › Basic Science

Vascular Phenotyping of Osteoarthritis

Osteoarthritis (OA) is a chronic degenerative joint disease and remains one of the most significant challenges in modern medicine. Despite its classification as a »serious disease« by the FDA, no disease-modifying osteoarthritis drugs (DMOADs) have been successfully developed. One key reason is the heterogeneous nature of OA, which encompasses multiple clinical phenotypes and molecular endotypes rather than a single, uniform disease. Emerging evidence suggests that vascular invasion plays a crucial role in OA progression, particularly through the pathological proliferation of type H vessels, which are linked to pain fiber infiltration, inflammation, and cartilage degeneration.

This study aims to elucidate the mechanisms by which cartilage loses its natural resistance to vascular invasion and to identify OA patient subpopulations who may benefit from anti-angiogenic therapy. We hypothesize the existence of a »vascular« OA endotype characterized by aggressive vessel infiltration and rapid disease progression. To investigate this, we will conduct an ex vivo study using osteochondral samples from joint replacement surgeries, correlating vascular density with clinical data,

histological cartilage degradation, and molecular markers.

Our work packages include (i) 3D vessel structure imaging via immunohistochemistry, (ii) spatial transcriptional imaging using mRNA probes for angiogenesis- and neurogenesis-related markers, (iii) comprehensive clinical phenotyping, and (iv) transcriptomic and epigenomic profiling of cartilage and subchondral bone. Furthermore, we will analyze samples from patients who underwent transarterial periarticular embolization (TAPE), a pain-relieving, symptomatic OA treatment targeting pathological vessels, to assess differences between responders and non-responders.

This project has high translational potential, leveraging state-of-the-art imaging and molecular profiling to advance personalized OA therapies. By refining OA classification based on vascular characteristics, our findings could be used to design the first successful DMOAD and improve patient selection for anti-angiogenic treatments.

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- › Osteology
- › Experimental Traumatology

Neuro-Osteogenic Interaction after Neurotrauma in Polytrauma – Systemic and Local Effects Guiding Regeneration?

Trauma is one of the leading causes of death and disability worldwide, especially in younger people. Among polytraumatized patients, traumatic brain injury (TBI) and traumatic spinal cord injury (SCI) are associated with a drastic rise in complications and mortality, and even if survived frequently cause long-term disability. The molecular mechanisms of the whole-body pathophysiology in the acute posttraumatic phase are not well understood. Consequently, targeted therapies to modulate posttraumatic systemic disruptions of homeostasis and prevent complications such as sepsis, cardiovascular dysregulations, multi-organ failure, reduce mortality and optimize the overall outcome are lacking.

Furthermore, the molecular base of accompanying skeletal phenomena, such as heterotopic ossifications and enhanced fracture healing in the context of TBI and TSCI on the one hand and reduced overall bone quality of intact bone after neurotrauma on the other, stay elusive.

With a murine polytrauma model combining TBI, SCI, femoral fracture and their combinations in an intensive care-setting we aim in cooperation with colleagues from the departments of neurosurgery, anesthesiology and the BCRT/JWI to explore their pathophysiological interactions in the acute phase post trauma using multimodal, multi-scale analysis towards mechanistical studies of the systemic and osseous phenomena in polytrauma.

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- › Cell Therapies

Preclinical Development of Chimeric Autoantibody Receptor (CAAR) T Cell Therapy for Myelin-Oligodendrocyte Glycoprotein Antibody-Associated Diseases (MOGAD)

Myelin-Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) is an inflammatory disorder of the central nervous system (CNS) characterized by attacks of immune-mediated demyelination predominantly targeting the optic nerves, brain, and spinal cord, leading to optic neuritis, encephalitis and myelitis. The MOG IgG antibodies are directly pathogenic by facilitating complement deposition, antibody-dependent cell-mediated cytotoxicity, and antibody-dependent cellular. In MOGAD irreversible attack-related disability accumulates with each attack. Since well-investigated, pathomechanism-based options are not established, strategies for attack prevention treatment are based on broad immunosuppression and experiences from other autoimmune diseases. These include prednisolone, azathioprine and rituximab, which have considerable disadvantages. Overall, up to 50 % of patients remain refractory or must stop treatment due to side effects including risk of opportunistic infections. This highlights the need for a disease-specific and highly effective therapy in MOGAD.

Recently, the principle of chimeric antigen receptor (CAR) T cells, which were developed for the treatment of hematologic malignancies, was utilized to engineer chimeric

autoantibody receptor (CAAR) T cells for the treatment of autoantibody-mediated disorders. In this very exciting approach, the T cell is equipped with a T cell receptor that presents the antigenic target region of the autoantibody on its surface. Thereby the T cell receptor can specifically bind to those B cells carrying the disease-associated pathogenic antibody as a surface receptor. The CAAR T cell will engage in an immunological synapse with the pathogenic B cell and specifically lyse only the disease driving B cell. Our group has recently proven the efficiency of CAAR T cells in depleting N-methyl-D-aspartate receptor (NMDAR) specific B cells for treatment of the most common form of autoimmune.

In my project I aim to develop efficient and specific MOG CAAR T cells that selectively deplete MOG antigen-specific B cells. MOG CAAR T cells have the potential to function as a living drug, offering a long-term, curative solution with just a single application for a severe demyelinating disease. This project will significantly contribute to novel therapeutic approaches for autoimmune neurological disorders, particularly by advancing our understanding of CAAR T cell design and functionality for small or membrane-proximal antigens.

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- › Tumor Biology
- › Spatial Proteomics



High-Plex Spatial Proteomics to Explore the Metastatic Bone Marrow Niche in Patients with Neuroblastoma (High-Plex NB)

High-Plex NB is a project aiming to characterize the mechanisms of bone marrow metastasis in high-risk neuroblastoma. The bone marrow microenvironment fosters tumor dormancy and subsequent relapse in many solid tumors. Neuroblastoma is a highly malignant pediatric tumor arising in the adrenal gland. It proves fatal in >50% of patients with high-risk disease. At diagnosis, bone marrow infiltration by disseminated tumor cells is detected in 90% of patients with high-risk disease. Despite successful eradication of the primary tumor through multimodal therapy patients often die due to bone marrow metastasis, where small populations of disseminated tumor cells can persist as minimal residual disease later give rise to relapse.

While bone marrow-residing mesenchymal stromal cells and the MIF-CXCR4 signaling axis are implicated in neuroblastoma invasion, the mechanisms by which neuroblastoma cells home to the bone marrow and form metastasis remain poorly understood. The lack of spatially resolved data still currently hampers a deeper understanding of the cellular interactions between disseminated neuroblastoma cells and the metastatic bone marrow niche.

High-Plex NB aims to characterize the metastatic bone marrow niche in matched formalin-fixed paraffin-embedded bone marrow and primary tumor samples from patients with high-risk neuroblastoma with varying degrees of bone marrow infiltration and therapy response. As such we aim to investigate the mechanisms underlying neuroblastoma bone marrow infiltration and therapy resistance. We will employ targeted high-plex immunofluorescence chip cytometry technology with custom panel design to characterize all cell types residing in the bone marrow niche including invading neuroblastoma cells.

Combining tumor cell intrinsic and extrinsic parameters and their interplay is a prerequisite for a basic understanding of metastatic spread, which is ultimately the leading cause of death from cancer. We hope to establish novel biomarkers to monitor minimal residual disease, stratify patient risk and assess therapy response. High-Plex NB could also identify novel drug targets to ultimately interrupt the invasion-metastasis cycle.

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- › Induced Pluripotent Stem Cells Derived Sensory Neurons
- › Mitochondria

Paclitaxel-Induced Mitochondrial Alterations in iPSC-DSN as Potential Predictive Marker and Therapeutic Target in CIPN

Chemotherapy-induced peripheral neuropathy (CIPN) is a prevalent and often irreversible adverse effect of cytotoxic treatments, characterized by sensory loss, altered sensations, and neuropathic pain. Paclitaxel, a widely used chemotherapeutic, is strongly associated with CIPN. Up to date, predictive markers of CIPN as well as targeted treatments are missing.

Our research group has successfully developed induced pluripotent stem cell-derived sensory neurons (iPSC-DSN) as a novel human in vitro model for studying chemotherapy-induced neurotoxicity. A recent multi-omics analysis of paclitaxel-treated iPSC-DSN from a patient with severe CIPN revealed differentially expressed genes related to mitochondrial function and transport and preliminary experiments depicted mitochondrial as well as lysosomal alterations.

This project aims to analyze mitochondrial and lysosomal parameters in iPSC-DSN of patients with and without CIPN (1) at baseline as potential predictive marker, (2) after paclitaxel treatment, and (3) correlate clinical severity of CIPN. I will investigate mitochondrial morphology and motility as well as markers of mitochondrial functionality and mitophagy. With the results, I hope to add to understanding pathomechanisms of CIPN that may result in targeted therapies and help to identify patients at risk of CIPN.

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› T cell Immunology
› MR Imaging

Immune Activation and Target Epitopes in Patients with Fatigue Syndromes Following COVID-19

This research project investigates persistent SARS-CoV-2-specific immune responses in post-COVID-19 syndrome (PCS) patients, particularly those meeting myalgic encephalomyelitis/chronic fatigue syndrome (ME-CFS) criteria. It compares these responses to COVID-19 convalescents without PCS. PCS, occurring within three months post-infection and lasting at least two months, affects daily life and lacks alternative explanations. Symptoms include fatigue, headache, myalgia, and neurologic deficits. Potential causes involve autoimmunity, viral reactivation, or persistent viral reservoirs. Recent studies suggest viral persistence as a PCS driver, with viral components detected in various organs, triggering ongoing immune activation. The study examines three groups: PCS patients meeting ME-CFS criteria, PCS patients not meeting these criteria, and COVID-19 convalescents without PCS. Samples come from established cohorts. The primary objective is to assess recently activated SARS-CoV-2-specific T cells, including their phe-

notype, functionality, and exhaustion status, as well as antibody epitope patterns. The main hypothesis suggests PCS patients will show higher proportions of activated T cells with impaired functionality and reduced receptor avidity. Methods include flow cytometry for T cell analysis, stimulating PBMCs with SARS-CoV-2 peptide pools. High-content imaging will profile exhaustion markers, while peptide microarrays will assess B cell epitopes. The study aims to provide insights into PCS pathomechanisms, potentially leading to prognostic factors, diagnostic markers, and effective treatments.

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- › Deep Brain Stimulation
- › Neuroimaging

MR Morphological Characteristics in PD Patients with Heterozygous GBA Mutation

Heterozygous Glycocerebrosidase (GBA) mutation is the most frequent genetic risk factor to develop Parkinson's disease (PD). GBA mutation carriers tend to have a manifestation of the disease at a younger age and a more aggressive disease progression with earlier cognitive decline and higher rate of neuropsychiatric comorbidities. One recent study reported that GBA positive-PD patients seem to suffer from a more severe cognitive decline especially after implantation of deep brain stimulation and should therefore be carefully selected for this therapy.

I want to use magnetic resonance imaging (MRI) derived voxel based morphometry (VBM) to detect cerebral volumetric differences between GBA positive and negative PD patients at the timepoint of the DBS evaluation in regions known to play a role in cognition. Furthermore I want to do follow ups on cognitive testing 2 years after DBS implantation to see whether there is a more severe

cognitive decline in GBA positive patients. The aim is to correlate cognitive data and volumetric analysis and find radiologic biomarkers for a future cognitive decline under DBS therapy to optimize selection for DBS implantation especially for patients with GBA mutation.

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- › Clinical research
- › Preclinical Tumor Models
- › Combination Therapies of Radiation & Targeted Therapies

Breaking Radioresistance in Soft Tissue Sarcomas Through Novel Combination Therapies: Development of a Preclinical Pipeline

Soft tissue sarcomas (STS) are rare, malignant tumors of mesenchymal origin. Standard treatment for high-grade STS includes resection and radiotherapy, but all subtypes are uniformly treated despite their radioresistance. Subtype-specific therapies for unresectable or metastatic STS show promise in enhancing radiotherapy (radiosensitization). However, there is a lack of data on effective combination therapies with radiosensitizers to improve STS outcomes. This project aims to establish a translational pipeline for personalized STS therapy using targeted therapies to overcome radioresistance.

Hypotheses: Intertumoral heterogeneity in radioresistance can be reproducibly quantified in a 3D model. Targeted compounds can overcome radioresistance, with interactions quantifiable in a 3D model.

Study Design and Methods: Conducted at Charité – Universitätsmedizin Berlin with collaborations from various institutes, this study will use human STS cell lines. These cells will be cultured in 3D using extracellular matrices. Stability will be verified through microscopy, immunohistochemistry, immunofluorescence, DNA sequencing, and qPCR.

Radiation will be applied under controlled conditions, and clonogenic survival assays will measure radiosensitivity and combination therapy efficacy. Additional assays, including CellTiter-Glo® and DNA damage response assays, will further characterize therapy effects.

Hypothesis Testing: Hypothesis 1: Radioresistance will be assessed in three STS cell lines through photon irradiation at 0, 2, 4, 6, and 8 Gy, followed by clonogenic survival assays.

Hypothesis 2: Radiosensitization with targeted compounds (Pazopanib, Olaparib, Xevinapant) will be evaluated with 2 Gy irradiation and varying compound concentrations. Dose enhancement ratios (DER) will be calculated and compared using Wilcoxon rank-sum tests.

Innovation Potential: This project aims to develop a screening platform for personalized combination therapies of radiosensitizers and radiotherapy, potentially improving tumor control and reducing toxicity in STS. Identifying combination partners can lead to mechanistic studies, biomarker development, and clinical trials.

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- › Geriatric Psychiatry
- › Psychosis

Neurocognition in Patients with Non-Affective Psychosis in the Second Half of Life (NAP-2)

Patients in the second half of life suffering from schizophrenia and related diseases are rarely represented in studies, predominantly because of the patients' age and weak reliability. As a consequence, knowledge of the critical characteristics of this disease in the second half of life is lacking, especially regarding influencing factors on a favorable course of disease.

Neurocognitive deficits can be sequelae of non-affective psychosis, negatively influence the course of disease and impair participation in society. Impairment of neurocognitive abilities occurs early, frequently and most likely stable. However, data on elderly patients in comparison with healthy controls are lacking, which makes it unclear whether the cognitive deterioration is age-appropriate. It can be assumed that there are disease- and treatment-related factors with a negative impact on neurocognition. Risk factors for an above-average neurocognitive deterioration may be an unfavorable cardiovascular risk profile, a long-term therapy with antipsychotics, consumption of alcohol or nicotine, missing physical exercise, alterations in the brain volume and

psychosocial variables such as socioeconomic status or social integration.

The presented study aims as the first of its type at investigating how differences in neurocognition between patients with non-affective psychosis and healthy controls in the second half of life evolve and which biological and psychosocial influencing factors are crucial. Therefore, a cross-sectional observational study including four age cohorts (in patients and healthy controls, aged 40 years or older) and a retrospective questioning is planned.

If the age-related neurocognitive deterioration is stronger in patients with non-affective psychosis, risk factors that are linked to the neurocognitive deficits could be identified. Hence, the planned study may reveal potential modifiable risk factors and form the basis of therapeutic interventions, health policy and social measures in order to diminish the course of disease of patients with non-affective psychosis.

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Fields of Research

- › Ultrasound
- › Elastography
- › Dispersion

Clinical Translation of Time-Harmonic Ultrasoundelastohgraphy and Dispersion

In recent years, ultrasound elastography has been established as a component of multiparametric ultrasound (mpUS), but scientific studies on the various organ systems have provided varying degrees of evidence. In particular, shear wave elastography of the liver has become widely established. The main limitations in the use of shear wave elastography in the liver can be addressed by the novel time harmonic elastography (THE) developed at Charité. However, the question of dispersion, which can be precisely explored using THE via stimulation with different vibration frequencies of the examination bed, is almost completely unanswered in the scientific literature. The differentiation of subtle pathological conditions in large numbers of cases is necessary in order to establish the dispersion of liver stiffness as a clinical marker for cardiac congestion or tumor characteristics, for example. The dispersion would provide previously inaccessible or unexplored information about the tissue to be examined in the context of »viscosity«.

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Fields of Research

- › Trauma Surgery
- › Regenerative Medicine
- › Biomaterials

Development of Regenerative Hybrid Scaffolds for Critically-Sized Bone Defects

Critical bone defects often occur as a result of severe trauma, tumor operations and insufficient fracture healing. The reconstruction requires interoperative bone replacement material. In 2021, bone replacement materials were transplanted for a total of 97,579 bone defects per year in Germany, most of them from autologous bone.

However, this method is associated with high donor morbidity. Complications such as bleeding and hematomas, permanent nerve damage, prolonged pain, wound infections and temporary loss of sensation occur in 15-25% of cases. The procedure also prolongs the operating time and subsequent hospital stay. This results in a clinical need for regenerative synthetic bone substitutes to replace autologous bone grafting. However, the synthetic bone replacement materials currently available are biologically far inferior to autologous transplants in terms of tissue healing. In my project, me and the team of the Experimental Trauma Surgery Lab at Campus Benjamin

Franklin are researching resorbable bone implants made of organic polymers that can be used to bridge bone defects and promote the formation of new bone.

To this end, we combine 3D printing technology and the synthesis of various resorbable biomaterials.

In the course of the project, we are investigating the mechanical strength of the 3D printed constructs on the one hand and the osteointegration capability of various biomaterials on the other in order to determine an optimal combination of implant architecture and biomaterial composition. The aim is to provide surgeons with an off-the-shelf implant for bone replacement that allows them to flexibly bridge different bone defects intraoperatively without the requirement of autologous grafting or having to perform a revision operation to remove the material afterwards.

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- › Neurology
- › Chronic Pain
- › Pain Mechanisms

Cortical Mechanisms of Chemotherapy-Induced Neuropathic Pain: Predisposition, Therapeutic Options and Preventive Measure

Chronic pain remains a major medical challenge, particularly in oncology, as chemotherapy-induced peripheral neuropathy (CIPN) is a frequent and debilitating side effect. CIPN affects up to 50% of patients receiving chemotherapy, leading to spontaneous pain, hyperalgesia, and allodynia, often necessitating reductions in chemotherapy dosage. Despite similar treatment regimens, only a subset of patients develops chronic pain, highlighting the need to identify underlying mechanisms and predictive factors. Additionally, the role of psychological factors in pain susceptibility and treatment response is still unclear.

While peripheral nerve damage in CIPN is well-characterized, central mechanisms contributing to CIPN-related pain are poorly understood. Chronic pain involves not only sensory processing but also cognitive and emotional dimensions, altered cortical activity and connectivity, and neuroinflammation. These mechanisms are well-established in other chronic pain conditions but are not well understood in CIPN.

To address these questions, this research will utilize a well-established CIPN mouse model to assess cortical

activity, neuroinflammatory markers, and behavioral changes over acute and chronic phases. MRI will be used to analyze volumetric changes, functional and structural connectivity. Optogenetic circuit manipulation will help delineate key pathways, particularly those involving the Periaqueductal Gray, which is central to pain modulation and treatment response. The study will also examine the impact of opioidergic and serotonergic pathways on CIPN symptoms, with potential implications for optimizing existing treatments like duloxetine and opioids. Furthermore, this project will explore chronic stress as a predisposing factor for severe CIPN. To investigate preventive strategies, the neuroprotective potential of lithium will be assessed, building on prior findings that lithium may prevent CIPN symptoms through peripheral mechanisms, while its potential cortical effects remain unexplored.

By integrating behavioral, neuroimaging, and molecular approaches, this study aims to uncover the central mechanisms associated with CIPN and advance personalized treatment strategies for patients.

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- › Neurology
- › Cardiology
- › Immunology

Central Control of Stress Networks in Stroke-Heart Syndrome: An Innovative Target for Therapeutic Interventions

In Germany, over 270,000 individuals experience an ischemic stroke each year. Globally, stroke ranks as the second leading cause of death and a major contributor to adult disability. Within the first year, 25-30% of stroke patients die, and up to 40% live with permanent impairments. Consequently, stroke imposes a substantial economic burden—annual costs in Germany alone were estimated at 17 billion euros in 2017.

Historically, stroke research focused on neuronal protection, while the brain's role in regulating peripheral organs was largely overlooked. Only recently have the systemic effects of disrupted central regulation gained attention. One example is »stroke-heart syndrome«, describing severe cardiac complications following ischemic stroke. Within days, 10-20% of patients develop acute myocardial damage—even without prior heart disease—and those with cardiovascular comorbidities face a higher risk of recurrent events.

Early cardiac complications double or triple short-term mortality. Long-term, one-third of deaths among stroke

survivors are due to cardiac causes. Despite secondary prevention, current therapies fail to address the underlying mechanisms of stroke-heart syndrome. The brain-heart axis remains poorly understood.

This project will investigate early brain-mediated cascades that drive cardiac damage. Using iDISCO 3D Clear-Map histology, optogenetics, chemogenetics, and immune-specific interventions, the study will map stroke-induced brain activity and define how central circuits influence peripheral cardiac outcomes via immune, sympathetic, and HPA-axis pathways.

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- › Tumor Microenvironment
- › Immunotherapy
- › CAR T cells
- › Neuroblastoma



Unraveling Interplay Between MYCN and Cellular Components in the Immunosuppressive High-Risk Neuroblastoma Microenvironment to Enhance Immunotherapy Success

Neuroblastoma is the most common extracranial solid tumor in children. Half of neuroblastomas at high risk for relapse harbor amplifications of the MYCN oncogene, a key player in tumor development, progression and metastasis and correlating with poor prognosis and therapy resistance. Cell-based immunotherapies offer a promising new approach against solid tumors. Initial success has been observed in treating relapsed high-risk neuroblastoma using chimeric antigen receptor (CAR) T cells targeting a neuroblastoma-specific antigen (GD2). However CAR T cell efficacy was not sustained against neuroblastoma because their immunosuppressive microenvironment impedes CAR T cell persistence and function. Cellular components within the microenvironment, namely tumor-associated macrophages and cancer-associated fibroblasts, contribute with intertwined function to the complex immunosuppressive tumor state. A recent study indicates MYCN amplification may potentially strengthen neuroblastoma immune evasion. The mechanism involved, and whether it acts through macrophages and fibroblasts in the tumor microenvironment remains unclear.

Our research aims to uncover how oncogenic MYCN levels influence the presence, behavior, and function of macrophages and fibroblasts in the immunosuppressive neuroblastoma microenvironment and how these components hinder immunotherapy. I will test whether (1) the microenvironment in neuroblastomas with/without MYCN amplifications shows distinct differences in the composition and distribution of distinct macrophage, lymphocyte and fibroblast subpopulations; (2) crosstalk dependent on or driven by oncogenic MYCN levels in neuroblastoma cells and involving tumor-associated macrophages and fibroblasts creates an immunosuppressive microenvironment hindering CAR T cell effectiveness; (3) targeted strategies to disrupt macrophage and fibroblast interactions in the tumor microenvironment enhance CAR T cell efficacy. Mechanisms underlying immunosuppression will be revealed by combining comprehensive analyses of patient tumor samples using multiplex immunohistochemistry, gene expression profiles and functional in vitro assays. Findings will be used to design and test new strategies to improve CAR T cell efficacy by disrupting the immunosuppressive network.

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- › Pediatric Pulmonology
- › Primary Ciliary Dyskinesia
- › Genotype/Phenotype Correlations



Characterization of Patient-Derived Nasal Epithelial Cultures to Unravel Disease Mechanisms in Primary Ciliary Dyskinesia

Primary Ciliary Dyskinesia (PCD) is a highly underdiagnosed rare genetic (autosomal recessive and in rare cases X-linked or autosomal dominant) disease that is poorly understood and cannot be cured. PCD affects 1 in 7500 people and is caused by mutations in more than 50 genes important for ciliary function and assembly. Ciliary dysfunction leads to impaired mucociliary clearance in the airways, resulting in chronic progressive mucobstructive lung disease. Cilia are also important (i) to establish left-right asymmetry during embryogenesis, (ii) in the brain, and (iii) in the reproductive system. Symptomatic treatment with inhaled hypertonic saline, physiotherapy to mobilize mucus from the airways, and azithromycin to modulate chronic bacterial infection and inflammation are the only available therapies. There is an unmet research need: Understanding of disease mechanisms and improving diagnostics to identify targets for causative treatment options. We established next generation sequencing (PCD panel and whole exome) in collaboration with the Institute of Human Genetics. However, many mutations are (ultra)rare and their functional consequences remain unknown. In 30–45% of patients with

PCD, the genetic defect cannot be identified. Thus, functional and biochemical studies are important to (i) improve diagnostics, (ii) unravel disease mechanisms, (iii) identify mutation-specific therapeutic targets, and (iv) serve as assays for preclinical testing of novel therapeutic strategies. With a multi-method approach using patient-derived and mutation-specific nasal epithelial cells cultured at air liquid interface my project aims to functionally characterize PCD-causing mutations with unknown molecular and functional consequences. Patient-derived nasal epithelial cells will be taken non-invasively from nasal brushings by utilizing our newly established conditionally reprogrammed cell technology.

I will use this approach for functional and biochemical characterization of PCD mutations with and without known molecular consequences. In addition, this project will determine gender-specific effects on the functional phenotype of PCD mutations and determine effects of in vitro treatment with β -agonists and/or read-through agents on ciliary dysfunction.

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- › Pediatric movement disorders
- › Neurodevelopment
- › iPSC-derived neurons
- › Single-nucleus multiome analysis



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The Role of Dopamine in Allan-Herndon-Dudley Syndrome (AHDS)

The Allan-Herndon-Dudley syndrome (AHDS) is an ultra-rare disease caused by a defective thyroid hormone transport (SLC16A2 mutation, MCT8 deficiency) into the central nervous system. This leads pre- and postnatally to a disturbed T3-dependent gene expression of multiple target genes and clinically to a complex syndrome with developmental delay, movement disorders and high patient distress. We recently conducted a stakeholder engagement of affected families to establish patient-centered therapy goals. Various therapeutic strategies currently exist to overcome the transport defect, but these have not yet achieved an efficient improvement of patient-oriented treatment wishes. Thus, there is still an urgent need for therapy development. In cooperation with European neuro-pediatricians, we phenotyped 27 patients and described clinical signs of dopamine deficiency for the first time. In all our patients, we found correspondingly reduced dopamine degradation products in the cerebrospinal fluid, which is why we initiated

levodopa/carbidopa substitution. After two months up to one year, the patients showed promising clinical improvement in patient-centered treatment goals such as better target movements. I now want to test the hypothesis that dopamine plays a crucial role in the pathogenesis of AHDS (new disease mechanism of a known disorder). To this end, (1) I differentiate dopaminergic neurons from patient and control induced pluripotent stem cells and perform neurotransmitter analyses to investigate the influence of MCT8 deficiency on dopamine metabolism and (2) I perform multiome analyses with human fetal and postnatal brain tissue (NeuroBio-Bank, NIH) using single-nucleus ATAC and RNA sequencing to better understand the relationship of thyroid hormone and dopamine metabolism, to identify target cells and a time window for therapies.

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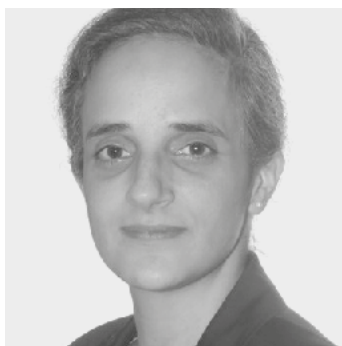
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The background of the entire page is composed of a dense, overlapping pattern of organic, cell-like shapes. These shapes are rendered in two shades of blue: a very light, almost white blue, and a slightly darker, muted blue. The shapes vary in size and orientation, creating a textured, fluid appearance that resembles a microscopic view of tissue or a modern, abstract pattern.

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Fields of Research

- › Myocardial Fibrosis
- › Heart Failure
- › Cardiovascular Imaging
- › Congenital Heart Disease



Mechanisms of Ventricular Interdependence – an Integrative Approach Towards Understanding Right Ventricular Failure

Right ventricular failure (RVF) is a high-risk factor for morbidity and mortality in patients with congenital heart disease, but also secondary to left heart failure. However, research on heart failure predominantly centers around the left ventricle, while the right ventricle (RV) has long been neglected. The knowledge of its determinants and variables to define RVF is relatively limited, as are suitable approaches to measure RV function and dysfunction in order to predict RVF at an early stage. Biomechanical whole organ and tissue patterns of adaptation and maladaptation to different types of adverse RV loading are yet not well understood.

The fundamental gap in knowledge about the underlying mechanisms that lead from function to dysfunction and failure of the RV interferes with the large and unmet clinical need for approaches that prevent and revert RVF. Accordingly, the main objective of the project is to improve the principal understanding of RVF through an

integrated investigation of organ and tissue function, with a specific focus on the role of myocardial fibrosis and the interplay between the RV and the left ventricle. Multi-dimensional imaging and clinical data of healthy individuals and patients with cardiovascular disease will be analyzed and combined using machine learning and phenomapping methods. With our approach, we aim to identify networks for mechanism-based stratification and de novo endophenotyping as well as RV specific biomarkers as diagnostic and prognostic measures and potential therapeutic targets. The project will thus contribute to set the prerequisites for the future development of patient-tailored diagnostic and treatment strategies that may ultimately improve outcomes in patients with RVF.

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- › Inflammatory Bowel Disease
- › Intestinal Organoids
- › Epithelial Defects
- › Mucus Barrier



Goblet Cell and Mucus Barrier Dysfunction in Paediatric Ulcerative Colitis

Inflammatory bowel disease shows a rising incidence in childhood and often remains difficult to treat in paediatric patients. While immune dysregulation contributes to the pathogenesis, increasing evidence points to epithelial barrier defects as key drivers of the disease. Goblet cells are essential for mucus barrier integrity. In ulcerative colitis (UC), histology shows a depletion of goblet cells, however, transcriptomic data point towards an increased number. We previously identified ER-stress-related goblet cell dysfunction in monogenic IBD, and similar changes appear to apply to paediatric UC. We hypothesise that increased ER-stress compromises goblet cell mucin processing and secretion capacity, causing mucus barrier disruption and ultimately leading to intestinal inflammation.

We will use patient-derived colonic organoids from paediatric UC patients and controls, cultured in 3D Matrigel and microfluidic colon-chips, to investigate goblet cell

function and mucus barrier integrity. Methods include scRNA-seq analysis, flow cytometry, immunofluorescence, and colorimetric assays. Mucus structure and function will be assessed using fluorescent microspheres and bacteria under basal and ER-stress conditions. A patient registry and biobank will support sample collection, stratification, and long-term research.

This project aims to uncover ER-stress-dependent mechanisms of goblet cell dysfunction and mucus barrier breakdown in paediatric UC. Findings may contribute to the identification of novel therapeutic targets to restore mucus integrity. The organoid biobank and registry will support future IBD research.

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- › Acute Kidney Injury
- › Chronic Kidney Disease
- › Single Cell Transcriptomics/
Epigenomics

Endophenotypes of Kidney Disease Progression at the Single Cell Level

Gaps in our understanding of the transition from acute kidney injury (AKI) to chronic kidney disease (CKD) remain a major unmet medical need due to the lack of effective therapeutics for >850 million people worldwide. Although several events have been identified that lead to progression from AKI to CKD (AKI-to-CKD transition), a key bottleneck is our lack of understanding of what exactly characterizes adaptive and maladaptive repair, respectively. In the 21st century, nephrologists still group kidney disease based on clinical and histopathological classifications developed centuries ago. Single cell technologies have the potential to fundamentally improve our knowledge of kidney disease development because they capture molecular mechanisms that underlie disease drivers. This project aims to uncover cell type-specific »endophenotypes«, signatures characterizing progression (AKI-to-CKD transition), and regeneration (successful repair) alike. Leveraging state-of-the-art single cell technologies in a heterogeneous patient population mapping major etiologies of AKI, and sampling human kidney cells both invasively and non-invasively via urine, this project

aims at uncovering potentially paradigm-shifting insights into kidney disease progression that will help us target therapies based on cell states rather than preconceived disease classifications. Patient-individual tubuloids derived from urine kidney cells will serve as validation platform for in silico findings.

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- › Hematology
- › ASXL1-Mutated AML
- › Single-Cell Technologies
- › Sequencing

Deciphering Cooperating Gene Networks in ASXL1-Mutated AML for Targeted Interventions Leveraging a Custom Single-Cell Platform

This project aims to dissect the molecular mechanisms driving acute myeloid leukemia (AML) with ASXL1 mutations (ASXL1mut), which occur in up to 20% of AML cases and are linked to a particularly poor prognosis. Despite their high frequency, the role of ASXL1mut in leukemogenesis remains largely unexplored, and no targeted therapies exist, underscoring a critical unmet medical need. To address this, we previously conducted a cancer gene discovery screen in ASXL1mut mice using transposable DNA elements (transposons). Acting as mutagenic agents, these transposons randomly integrate and de-integrate across the genome, thereby activating or silencing genes in close proximity, and promoting leukemia development. By sequencing the resulting ASXL1mut AMLs, we generated a comprehensive catalogue of novel cancer genes potentially driving malignant transformation.

In this project, we aim to translate these murine findings into the human context. First, the mouse AML genes will be validated against two large-scale human ASXL1mut

AML datasets to assess their deregulation profiles. Next, high-confidence AML genes will undergo functional analysis using our newly developed state-of-the-art single-cell transposon and transcriptome sequencing platform, providing unprecedented insights into the molecular networks driving ASXL1mut AML and its clonal evolution. Additionally, we will employ high-dimensional spectral flow cytometry to link transposon alterations with phenotypic changes in our mouse ASXL1mut AMLs. Finally, the most promising AML cancer genes will undergo rigorous mechanistic characterization using CRISPR-(d) Cas9 technology, enabling precise manipulation of gene expression both in vitro and in vivo. This will allow for detailed investigation of their contribution to leukemogenesis and the preclinical development of potential therapeutic strategies or predictive biomarkers. In summary, by integrating mouse and human data with cutting-edge genomic and phenotypic tools, this project aims to pave the way for personalized therapeutic approaches, addressing the urgent need for targeted treatments in ASXL1mut AML.

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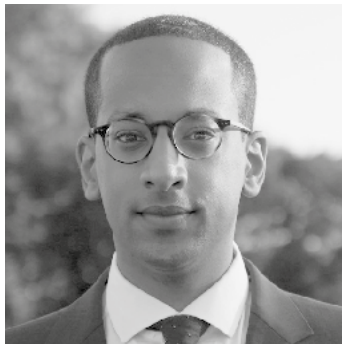
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- › Single-Cell Genomics / Transcriptomics
- › Tumor heterogeneity and microenvironment



Deciphering High-Risk Neuroblastoma Through High-Resolution Transcriptomic Profiling with Programmable Enrichment and Flow-FISH Sequencing

Rare cell types play crucial roles in tissue development, repair, and disease initiation. Although single-cell RNA sequencing (scRNA-seq) has revolutionized our ability to detect these minority populations, their low abundance poses challenges for in-depth molecular characterization. As a result, key drivers of disease may remain underexplored.

In pediatric cancers like neuroblastoma, recent single-cell studies have revealed a surprising diversity of tumor cell states, including neuron-like, mesenchymal, and proliferative subtypes. These populations interact with the tumor microenvironment in complex ways and may shift dynamically during disease progression. Some of these cell states are difficult to distinguish from normal tissue, complicating analysis and therapeutic targeting.

To overcome these limitations, we are implementing a novel method called PERFF-seq (Programmable Enrichment via RNA Flow-FISH followed by sequencing). This approach enables selective enrichment of rare cell populations based on their RNA signatures, followed by

high-throughput transcriptomic profiling. By targeting specific cell types of interest, PERFF-seq allows high-resolution analysis without the need for extremely large sample sizes.

We will apply PERFF-seq to patient-derived neuroblastoma samples to generate detailed molecular maps of tumor subpopulations that are difficult to capture using conventional methods. These findings will be validated in disease models, including patient-derived xenografts (PDX) and organoids, to functionally assess their role in tumor progression and therapy resistance. Our long-term goal is to better understand tumor heterogeneity and uncover new opportunities for precision treatment.

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- › Heart Failure
- › Metabolism
- › Myocardial Recovery

Cardiac Metabolism as a Determinant of Myocardial Recovery after LVAD Implantation

Implantation of a left ventricular assist device (LVAD; »heart pump«) represents a lifesaving treatment option for patients with end-stage heart failure. While LVADs were originally introduced as a bridging strategy to cardiac transplantation, the limited availability of donor hearts is associated with a growing number of patients requiring permanent mechanical circulatory support. Interestingly, the therapeutic response after LVAD implantation is quite heterogeneous, where some patients show myocardial recovery upon mechanical unloading (ie, improvements in cardiac function and reverse remodeling). The mechanisms of these beneficial effects remain largely unknown, and personalized approaches for patient selection and risk stratification are currently lacking.

Recent evidence suggests that alterations in myocardial metabolism may represent major determinants of the heart's ability to recover. Consequently, the outcome of patients with preserved cardiac energy production capacity might be superior compared to those with more severe derangements of myocardial metabolism. However, methods for the assessment of the complex and

dynamic metabolic network behavior remain limited in humans, and no previous study has investigated this potential link in LVAD patients.

The present project aims to better understand the role of myocardial metabolism in the therapeutic response upon LVAD implantation. To reconstruct patient-specific bioenergetic phenotypes, comprehensive molecular analyses of myocardial tissue biopsies will be leveraged to generate personalized computational models of myocardial metabolism. These »digital twins« enable large-scale simulations of the dynamic metabolic network behavior to changes in myocardial workload, differences in circulating substrate availability, and therapeutic interventions. Based on the hypothesis that patients with preserved myocardial metabolism may show a favorable response after LVAD implantation, this study will correlate the individual bioenergetic fingerprint with longitudinal outcome data. If successful, the the present project will provide important insights into the mechanisms underlying advanced heart failure and myocardial recovery alongside a novel approach for personalized risk prediction in patients undergoing LVAD implantation.

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- › Lymphoma
- › Senescence
- › Personalized Medicine

A Lymphoma-Immune Cell-Liver Multi-Organ Chip as Predictor Platform for Basic Research and Personalized Medicine

Despite intensive molecular profiling of primary patient material, lesion-based targeted therapies have not yet been able to improve standard of care in diffuse large B-cell lymphoma (DLBCL). However, therapy-induced biological switches with potential impact on treatment outcome remain elusive at the time of diagnosis because patient samples under treatment are rare and difficult to obtain. A key state switch of interest is therapy-induced senescence (TIS), an acutely stress-inducible terminal cell cycle arrest programme that acts as a delayed back-up mechanism to apoptotic cell death in response to virtually all types of anticancer agents. Senescent cells remain viable and typically exhibit a senescence-associated secretory phenotype (SASP) that affects their surrounding cells. This dynamic environment affects lymphoma-immune cell interactions, activation of trans-differentiation pathways, reprogramming, plasticity and stemness. While TIS is generally beneficial, it can occasionally cause senescent lymphoma cells to re-enter the

cell cycle and become more aggressive. To counteract this, sequential therapy has been proposed: chemotherapy followed by »senolytics«. In this context, we want to investigate the senolytic potential of different drugs. In close collaboration with colleagues from the laboratory of Prof. Sina Bartfeld (Department of Medical Biotechnology, TU Berlin), we have developed a multi-organ chip with a human lymphoma/immune cell interface and human liver spheroids to study the effects of therapy in more detail and at shorter time intervals. A microfluidic circuit connects all organ compartments via a micro-pump-driven heartbeat that mimics perfusion. Based on this multi-organ chip, we aim to investigate activity changes within the cellular immune compartment due to drug-induced altered tumour immunogenicity or drug-induced immune modulation, which collectively affect drug-induced immune-mediated cytolytic capacity.

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- › Amyloidosis
- › Short Bowel Syndrome
- › Intestinal Failure
- › Nutritional Medicine

Functional Diagnostics and Pathophysiological Investigations of Gastrointestinal Manifestations of Amyloidosis

Amyloidosis is a rare disease caused by abnormal folding of normal soluble proteins leading to fibril formation in organs and tissues throughout the body. Amyloidosis is classified based on the fibril precursor protein which also determines the clinical manifestations.

Primary hereditary transthyretin amyloidosis (hATTR), Amyloid light-chain (AL) amyloidosis and amyloid A amyloidosis (AA) lead to gastrointestinal symptoms such as obstipation, diarrhea, early satiety, and weight loss. However, the underlying pathomechanisms of these symptoms remain unknown.

The primary objective of this study is to compare disease-specific mechanisms (gastrointestinal motility, malabsorption, barrier dysfunction, inflammation, dysbiosis and permeability disorder) with healthy controls to investigate the pathophysiologic mechanisms of gastrointestinal symptoms.

The secondary objective is to evaluate objective non-invasive diagnostic tool as markers for disease severity, follow-up, and therapeutic effects. We will include approximately 96 participants in a cross-sectional and 16 patients in a longitudinal prospective observational study over 6 months. All participants will undergo a study visit including clinical, nutritional, motility, laboratory, and permeability assessment.

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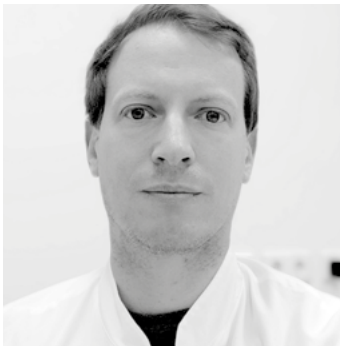
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› Functional Precision Medicine
› Hematological Malignancies

Combining Functional and Multi-Omics Approaches on Single Cell Level for Precision Medicine in Acute Myeloid Leukemia

Acute myeloid leukemia (AML) is characterized by frequent relapse, driven in part by a subpopulation of apoptosis-insensitive leukemic stem cells (LSCs). This study aims to identify and characterize these functionally distinct LSCs using an integrative approach. We apply intracellular BH3 (iBH3) profiling combined with multi-parametric spectral flow cytometry to assess apoptotic priming and protein expression at the single-cell level in genetically defined AML cell lines, patient-derived xenografts, and primary AML samples. This approach enables high-resolution discrimination of apoptotic states across heterogeneous leukemic subpopulations. Next, we molecularly characterize apoptosis-insensitive LSCs using single-cell mitochondrial DNA (mtDNA) and nuclear RNA sequencing to trace clonal evolution and identify aberrant signaling pathways associated with relapse. Finally, we assess the chemical vulnerabilities of these LSCs by screening a library of 40 FDA-approved or clinical-stage compounds, aiming to restore apoptotic sensitivity and overcome treatment resistance. Candi-

date compounds are evaluated ex vivo via dynamic iBH3 profiling and further validated in vivo using AML PDX models. This integrated strategy offers a promising avenue for the development of personalized therapeutic approaches targeting relapse-driving LSCs in AML.

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- › Molecular Genetics
- › Genome Biology
- › Connective Tissue Disorders



friede springer stiftung



Genome Sequencing of Hereditary Connective Tissue Disorders

Hereditary connective tissue disorders (HCTDs) are a heterogeneous group of rare diseases that can affect different organ systems. Hereditary thoracic aortopathies (H-TAD) comprise a subgroup that can present as isolated aortic disease or as part of a multisystemic disease such as Marfan syndrome or Ehlers-Danlos syndrome. Approximately 95% of thoracic aortic dissections occur in relatively young, previously asymptomatic patients without cardiovascular risk factors. Early genetic diagnosis is essential for individual patient management, such as the timing of preventive surgery.

The risk of developing a thoracic aortic aneurysm can often be explained by a sequence variant in one of 11 definitive causative genes. Routine diagnosis (i.e. panel sequencing) leaves more than 70% of cases unsolved. We hypothesise that genome sequencing and RNAseq will increase this diagnostic yield. To this end, 200 patients with inherited aortic and complex connective tissue diseases will be enrolled in this study over the

next two years. We hope to identify 1) new disease genes 2) new intragenic non-coding variants (e.g. deep intronic and UTR variants) and 3) extragenic regulatory variants.

To improve the interpretation of genetic variants, we will perform detailed and standardised phenotyping of the cohort and implement new prediction tools and variant callers into our software. We also plan to generate a map of regulatory elements in disease-relevant tissues for the evaluation of non-coding variants.

The identification of new causative variants will have a direct impact on the clinical care of affected individuals and their families. In addition, the discovery of new disease genes or regulatory variants will allow a better understanding of our genome and the underlying molecular pathomechanisms of thoracic aortic aneurysm and dysregulated ECM homeostasis.

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- › Emerging Zoonoses
- › Viral Pathogenesis
- › Innate Immunity

The Role of Peripheral Immune Cells to Monkeypox Virus Pathogenesis

Monkeypox (MPOX) virus led to a rapidly evolving pandemic in May 2022, with firstly over 90.000 cases reported beyond the African continent. While most infected individuals display a self-limiting disease with singular pox-like lesions, some endure systemic viral spread leading to whole body rash with risk for necrosis, organ loss and death. Since intra-host dissemination and tropism of MPOX virus are largely unexplored in humans, the cause for this clinical variability remains unknown. To elucidate the cellular tropism of MPOX virus, we exposed human peripheral blood mononuclear cells (PBMCs) from healthy donors with a currently circulating MPOX clade 2 virus isolate in absence and presence of interferon- α 2a. In kinetic experiments, we identified increasing MPOX virus DNA quantities in cell lysates, but apparently not supernatants, that peaked five to six days post exposure, suggesting susceptibility of PBMCs to infection. IFN- α 2a treatment markedly reduced MPOX virus DNA quantities, suggesting that infection is sensitive to type I interferons. Using scRNA-sequencing of MPOX virus-infected PBMCs,

we are currently identifying viral RNA-positive immune cell subsets and characterizing the corresponding gene expression profile at the single cell level. Finally, coinfections with MPOX virus and HIV-1 have been widely reported during the MPOX virus pandemic, as both virus infections affect the same risk group of men who have sex with men (MSM). Clinical observations reported more severe MPOX virus infections in immunocompromised patients compared to immunocompetent individuals. Therefore, we aim to investigate possible interrelations between MPOX virus- and HIV-1-infection at the level of viral replication and infectivity.

Our results have the potential to illuminate aspects of intra-host propagation of MPOX virus that may involve a lymphohematogenic route for replication, and will widen our knowledge on poxvirus pathogenesis, therefore contributing to the global preparedness in times of frequently emerging zoonoses.

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- › Epidemiology

Effects of Dopamine Modulation on Motivational and Motor Function in Major Depression Characterized by Low-Grade Inflammation

Major depressive disorder (MDD) is a leading cause of disability worldwide and has many adverse mental and somatic health consequences including cardiometabolic diseases. However, about one third of patients do not achieve full remission from depression even after multiple antidepressant treatments – potentially due to its clinical heterogeneity. Chronic low-grade inflammation is present in more than a quarter of patients with MDD, is associated with treatment resistance, and may represent the underlying substrate linking depression and cardiometabolic diseases.

A large body of evidence on depression heterogeneity point to an »immunometabolic« subtype characterized by the clustering of immunometabolic dysregulations with atypical behavioral symptoms related to energy homeostasis. Motivational and motor impairments reflected by symptoms of anhedonia and psychomotor retardation in MDD are closely related to alterations in

energy homeostasis, are associated with increased inflammation, and may be a direct consequence of the impact of inflammatory cytokines on mesolimbic dopamine (DA) signaling.

In the proposed project, we will examine the effect of DA stimulation on motivation and motor function in patients with MDD and healthy controls and the role of inflammation using a double-blind, randomized, placebo-controlled, cross-over design. If successful, our study would provide crucial evidence that pharmacologic strategies that increase DA may effectively treat inflammation-related symptoms of anhedonia and psychomotor retardation in MDD. Similar pharmacological strategies may have clinical utility in other neuropsychiatric populations with increased inflammation as well.

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› Translational Psychiatry
› Immunopsychiatry

Defining the Role of Clonal Hematopoiesis in Alzheimer's Disease

Somatic mutations recurrent in hematologic malignancies are detectable in 10-30% of older healthy individuals, a phenomenon called clonal hematopoiesis of indeterminate potential (CHIP). A recent study found that CHIP attenuated the risk of Alzheimer's disease (AD) by 40%, showing an enrichment of CHIP-mutated cells in the microglia fraction of the brain. However, additional studies are required to validate the findings in prospective longitudinal cohorts enriched for AD-risk and to dissect the potential protective mechanism.

Using bulk and single cell DNA sequencing we will assess the role of CHIP in AD in the prospective German Longitudinal Cognitive Impairment and Dementia (DELCODE) cohort (n=974), that includes healthy controls (HC = 233), subjective cognitive decline (SCD = 432), mild cognitive impairment (MCI = 189) and Alzheimer's disease dementia (ADD, n = 120) subjects, followed up for 8 years. We aim to: 1. Define the prevalence and landscape of CHIP mutations at different stages of the disease (HC to ADD), clarify if CHIP can predict risk of progression and identify correlations of CHIP-mutations with cognitive decline over time. 2. Define functional correlates of CHIP with biomarkers of neurodegeneration and neuroinflammation.

3. Assess the longitudinal distribution of CHIP-mutations across immune cell subsets. 4. Characterize CHIP-related functional effects in patients-derived personalized assays of microglia-like cells.

Due to the high number of cognitively unimpaired baseline participants, long clinical and neurocognitive follow-up as well as the rigorous bio-banking of the DELCODE-cohort we expect to exactly assess the putative protective effect of CHIP in AD. This will allow us to confirm or challenge the proposed protective effect of CHIP in a neurocognitive well-characterized large heterogeneous European sample. If neuroprotective properties of CHIP are confirmed, our research will provide a clear description of which mutated genes of the myeloid cells play a protective role and describe new targets for immunomodulatory preventive or therapeutic strategies in AD.

These data suggest a role for mutant, marrow-derived cells in attenuating the risk for the development of AD, possibly by counteracting a failing microglial system during aging. By dissecting the exact role of CHIP in AD we set the building stone for novel therapeutic and or preventive concepts.

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- › Rare Genetic Disorders
- › Lymphovascular Diseases
- › Lymphatic Malformations
- › Overgrowth Syndromes

A Translational Approach to Improve Healthcare of Patients with Rare Lymphovascular Diseases

Lymphovascular diseases (LVD) represent an underdiagnosed and heterogeneous group of maladies that occur sporadically or as a part of a syndromic disorder (e. g. overgrowth syndromes with vascular involvement). Due to the pathophysiological complexity of LVD, specialized, interdisciplinary care structures are required. Our project represents an innovative patient care concept to provide personalized medicine in LVD: We organize multi-centric, interdisciplinary case conferences to provide a detailed clinical characterization, essential for further phenotype-based evaluation of genetic data. Using Next Generation Sequencing we analyze the protein-coding and non-coding regions of the human genome using DNA from blood, as well as affected tissue samples. Thereby identified genetic variants are then evaluated for pathogenicity utilizing genetic databases, bioinformatic prediction tools as well as functional studies. Using this approach, both the confirmation of a known genetic disorder, as well as the identification of new candidate

genes in LVD can be achieved. Identifying disease-associated genes improves our understanding of LVD pathogenesis and is the first crucial step for the establishment of novel therapeutic approaches. Moreover, our project also aims to assess the use of already available pharmaceuticals in LVD. For this, we establish individual endothelial or fibroblast cell lines for in-vitro testing. Through this process, the efficacy of different FDA-approved drugs is tested, providing important information for the optimization of current patient therapy.

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- › Connectomics
- › Functional MRI

Evaluation of Symptom-Specific Networks in Dyskinetic Cerebral Palsy

Dyskinetic cerebral palsy (DCP) is an umbrella term for non-progressive motor disorders that result from damage to the developing brain and are characterized by a mixed dyskinetic phenotype including dystonia and choreoathetosis. While deep brain stimulation (DBS) of the Globus pallidus internus is an established treatment for isolated dystonia, current treatment options for complex acquired dystonia (i.e. DCP) remain unsatisfactory. Specifically, pallidal DBS in DCP has shown variable results in clinical trials indicating a need for better patient and target selection to improve outcome of neuromodulation strategies in this condition.

Imaging studies reveal cerebral lesions in over 60% of DCP cases, most frequently in subcortical grey matter. However, there is increasing evidence that lesions represent hubs of wider, functionally connected networks. A recent »lesion network mapping« study showed that acquired lesions causing dystonia in adults map to a common network that can be targeted for effective DBS in genetic/idiopathic forms of dystonia. In Parkinson's disease, symptom-specific networks can be differentially modulated for optimizing individual treatment outcome.

In a previous study, we have shown that lesions causing DCP are functionally connected to a network including areas involved in the pathophysiology of hyperkinetic movement disorders and highlighting the motor thalamus. However, it is still unknown to what extent specific motor symptoms are associated with different functional networks in DCP. Furthermore, currently available motor scales for DCP insufficiently account for the complexity of this movement disorder and may miss relevant effects in interventional studies. Thus, development of objective, quantitative motor assessments in DCP is needed.

The first project aims to prospectively investigate symptom-specific motor networks affected by distributed lesions in DCP. For this, movement disorder patterns will be assessed in a large cohort of children and young adults with DCP and symptom-specific functional connectivity networks will be derived from individual MRI's using lesion network mapping. The second project aims to develop objective kinematic measures in this complex movement disorder. Specifically, motor function will be quantified in a pilot prospective clinical study using marker-less 3D motion analysis. The results of both projects can potentially be used to inform and design future interventional trials of DCP.

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- › B-Cell Lymphoma
- › Immuno-Oncology

Biomarker-Informed SUMO Inhibition for Improved Lymphoma Therapy

Diffuse large B-cell lymphoma (DLBCL) is the most common type of aggressive non-Hodgkin lymphoma. Patients achieving remission after first-line immunochemotherapy have a good long-term prognosis, whereas survival is impaired at relapsed or refractory disease. Despite the striking success of immunotherapies in many tumor entities, latest trials on immune checkpoint blockade (ICB) in DLBCL have shown rather disappointing results. The poor response rate of DLBCL to ICB is partly attributed to an immune-cold tumor microenvironment (TME), which is frequently caused by genetic alterations. Within previous studies, we attributed SUMO inhibition (SUMOi) with strong immune-modulatory effects, emphasizing its potential to reactivate the TME. However, it is unclear which patient subgroups (biomarker-defined) would benefit from the addition of SUMOi to ICB and the impact of the most frequent genetic drivers of DLBCL on the TME is poorly understood. Within this project we aim to comprehensively characterize the impact of driver mutations

and the SUMO state on the TME in DLBCL patients with the ultimate goal of pinpointing biomarkers of an immune-cold TME. Furthermore, we will investigate the efficacy of SUMOi to reactivate the TME in these biomarker-defined subgroups aiming to develop biomarker-informed SUMOi-based combination therapies with ICB. In summary, this project will define biomarkers to drive forward translation of SUMOi addition to cancer immunotherapies in biomarker-defined subpopulations of DLBCL.

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- › Cancer Research
- › Non-Coding DNA
- › Pathology

Epigenetic Classifiers for the Diagnosis of Hepato-Pancreato-Biliary Tumors

The hepatopancreato-biliary (HPB) system is one of the most difficult areas of diagnosis in pathology. Multiple factors make histopathological diagnosis of HPB tumors difficult. First, because of its anatomic location, diagnostic biopsies from the HPB system are difficult to obtain. Second, the HPB system is the site of numerous benign and malign tumors that can be challenging to distinguish. Lastly, this is the site of over 13% of all metastases, as well as of 30-40% of all cancers of unknown primary. Misdiagnosis of HPB tumors can lead to over-/undertreatment of patients, translating into delayed curative interventions or overly aggressive therapies with unnecessary side effects, underlying the high unmet clinical need. Hence, a diagnostic tool that extends current approaches, is rapid, and could be used in the intraoperative setting is needed to help address the unmet medical need in HPB tumors. We hypothesize that genome-wide DNA methylation is a potential alternative for the development of molecular based classifiers that could aid the diagnosis of HPB tumors. Indeed, for brain tumors, DNA methylation-based classifiers made their way into standard diagnostic algorithms, and are now

considered desirable diagnostic criteria by the WHO. Recent technological advances make this approach very appealing. First, the development of machine learning tools makes it possible to integrate huge amount of data, such as genome-wide methylation patterns, and develop easy-to-use classifiers that can aid the diagnostic process. Second, the more rapid and less expensive nanopore sequencing was shown to be a useful tool for analyzing genome-wide DNA methylation changes. In contrast to the current gold-standard of microarray-based genome wide DNA-methylation analysis which takes several days to complete, nanopore sequencing can be completed in less than an hour, making DNA methylation classifiers a potentially useful intraoperative diagnosis aiding tool. The hypothesis is that epigenetic markers such as genome wide DNA-methylation patterns are specific for the tissue-of-origin. They can be used to build machine-learning classifiers and allow a more precise diagnosis of HPB tumors. A secondary hypothesis of this project is that more rapid and cheaper technologies can be used to analyze epigenetic changes and translate these classifiers into an intraoperative setting.

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- › Urothelial Bladder Cancer
- › Proteomics
- › Predictive Diagnostics

Individualized Medicine by Proteome-Based Ex Vivo Testing with Urothelial Cancer Signatures (IMPETUS)

Urothelial bladder cancer (UC) is a clinically challenging disease due to a wide tumor biological spectrum with rapid progression. Management of high-risk and intermediate-stage tumors requires adjuvant instillation therapy, while advanced and metastatic cancers have limited therapeutic options. After multi-level method optimization, systematic drug repurposing for a large, all-stage comprehensive cohort of 180 samples was performed based on robust effector-level proteomic signatures. With frequently reoccurring predictions across these independent samples, four new subtype-specific drug candidates of different molecular mechanisms were successfully validated in vitro. Based on a cohort of primary patient tissue, cells and in ovo xenograft tumoroids, extensive functional characterization and proteogenomic contextualization will further validate these drug candidates. Second, proteomic assays provide direct quantitative measurements of cellular effectors, which we used to identify highly robust prognostic and predictive UC subtypes. Further solidifying this approach, the application of absolute quantification techniques will be used to validate our proteomic UC subtypes.

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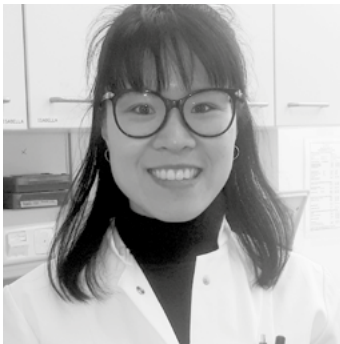
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Fields of Research

- › Acute-on-Chronic Liver Failure
- › Inflammation
- › Immunometabolism
- › Mitochondrial Function

Evaluation of Immunometabolism as a Driver of Hepatocyte Immunopathology in Acute-Onchronic Liver Failure

Patients with acute-on-chronic liver failure (ACLF) exhibit a complex immune phenotype ranging from excessive activation of the immune system to immune paralysis. The clinical consequence of this immune dysfunction is increased susceptibility to bacterial infections, which is the most frequent precipitating event of organ failures in patients who develop ACLF. The syndrome is associated with high short-term mortality, and current treatment options are limited to liver transplantation.

Whereas cirrhotic patients often present with neutropenia, patients with ACLF characteristically display increased circulating neutrophil and monocyte counts, supporting the view that the innate immune system plays a major contributory role in ACLF pathophysiology. In contrast to their circulating counterparts, information about the immune and metabolic phenotype of tissue-resident immune cells in liver and other organs such as kidney, brain or lungs of patients with ACLF is scarce.

Taking into account that patients with advanced liver cirrhosis and ACLF are often situated in the delicate and dynamic balance between exaggerated inflammatory response and immune exhaustion, it is not surprising that the search for therapeutic targets is cumbersome. Treatment of patients with ACLF thus requires individualized concepts considering the different extent of inflammation and immune dysfunction in each patient.

As a prerequisite for devising individualized treatments, this project aims at disentangling the complexity of immune phenotype and metabolic function of circulating and tissue-resident immune cell subpopulations, and to study whether immune cells are able to mediate liver immunopathology. Based on these findings, reprogramming metabolism of immune cells in order to reverse immune cell dysfunction will be investigated as a therapeutic strategy to modify the disease course of ACLF.

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- › Rare Diseases
- › Gene Therapy
- › Whole Genome Sequencing



Molecular Characterisation of Stem Cell Dynamics in Haemoglobinopathies in the Context of Gene Therapy

Stem cell transplantation remains as the only curative therapy for many rare diseases, including haemoglobinopathies such as sickle cell disease and thalassemia. Despite major advancements in recent years, severe hurdles for a successful stem cell transplantation remain, including donor availability, toxicity of therapy, graft versus host disease and rejection. Therefore, the search for a non-toxic, effective, and safe curative therapy for patients with haemoglobinopathies is ongoing. Gene therapies as functional corrective therapies are promising. But reports of malignancy such as myelodysplastic syndrome or acute myeloid leukemia after gene therapies in different trials are raising concerns, especially that the origin of those malignancies could not be identified yet. This project aims to identify causes of secondary malignancies in gene therapy compared to stem cell transplantation. By utilizing whole genome sequencing, we can differentiate causes such as insertional mutagenesis and clonal expansion of an existing premalignant clone. Further, we can identify specific genetic signatures

for driver mutations and for the exposure to alkylating agents such as busulfan used routinely in stem cell transplantation and gene therapy as part of the conditioning regimen. In this project, we will first learn from a retrospective analysis of patients in our comprehensive pediatric stem cell transplantation bio-databank. Second, we will apply the findings prospectively to patients treated with gene therapy and concomitantly build up a corresponding prospective gene therapy bio-databank for long-term observation with a focus on potential genotoxicity and safety monitoring. In a third step, this project will enable the expansion of analysis to innovative single-cell technologies and spatial transcriptomics to better characterize the stem cell dynamics on a molecular level in the context of these therapies. With this project's output, we aim to identify and validate novel biomarkers at the single-cell level for clinical monitoring of stem cell transplantation and gene therapy and make gene therapy as a promising curative therapy safer and more effective for our patients.

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Fields of Research

- › NAFLD
- › SNP
- › iPSC
- › NASH
- › MAFLD
- › MAFL
- › MASH

Generation of Liver Tissue from Induced Pluripotent Stem Cells to Investigate the Mechanism of Single Nucleotide Polymorphisms in Non-Alcoholic Fatty Liver Disease

MASH is a leading cause of end-stage liver disease (ESLD), often evolving from MAFLD. Despite the high prevalence of MAFLD, only a fraction of individuals develop severe fibrosis. The disease's complexity arises from environmental factors interacting with a polygenic susceptibility background. Studies have showed light on the genetic basis of MAFLD, revealing significant correlations with single nucleotide polymorphisms (SNPs). However, the role of SNPs in progressing from steatosis to MASH remains unclear, and creating appropriate disease models is challenging. Mouse models don't reflect the identified SNPs, and access to human samples with specific genetic backgrounds is limited. Stem cell technology has the possibility to overcome these restrictions. Human-induced pluripotent stem cells (hiPSCs) can differentiate into hepatocytes with specific genetic backgrounds, helping model MAFLD progression in human livers, potentially elucidating the mechanisms behind the disease progression.

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- › Endogenous Neuroprotection
- › Oxygen Glucose Deprivation Induced Cell Death
- › Neurodegenerative Disease

Non-Hematopoietic Erythropoietin Splice Variants as Human Endogenous Neuroprotective Agents

Erythropoietin (EPO) is a hypoxia-induced glycoprotein that inhibits apoptosis and promotes the differentiation of progenitor cells. Due to its effects on hematopoietic cells, EPO is commonly used to treat anemia. Through similar mechanisms, EPO provides neuroprotection by preventing neuronal apoptosis and promoting neurogenesis. However, these endogenous repair mechanisms are insufficient to compensate for severe damage, such as that induced by stroke. Despite its therapeutic potential in neurological diseases, the application of EPO is limited by hematopoietic side effects, including thromboembolism. In preliminary studies, we demonstrated that hypoxia-induced paracrine, but not autocrine, neuroprotection is mediated by EPO. Additionally, the detection of endogenous EPO splice variants revealed promising candidates potentially involved in autocrine neuroprotection. Notably, one specific splice variant exhibited comparable neuroprotective effects in a mouse model without inducing hematopoietic activity. In collaboration with Dr. Anna Zemella, group leader at the Fraunhofer Institute for Cell Therapy and Immunology, Potsdam, this splice variant has been produced cell-free using an innovative and highly efficient cell-free synthe-

sis approach. This method holds significant potential for developing therapeutic proteins. We are investigating the neuroprotective potential of this splice variant in a human model using iPSC-derived neurons subjected to oxygen-glucose deprivation (OGD) as an in vitro model of stroke. This iPSC model also enables us to evaluate potential effects on neuronal stem cell differentiation, thereby serving as a model for adult neurogenesis and regeneration. Furthermore, we aim to analyze the expression of this splice variant in human neurological disease. To achieve this, we employ BaseScope, a novel RNA hybridization technique, to detect single mRNA molecules and their splice variants as distinct signals at single-cell resolution in post-mortem human brain tissue. We hypothesize that this EPO splice variant is upregulated during hypoxia-induced cell death and neurodegenerative disease as part of the endogenous repair system. Moreover, we propose that it inhibits apoptosis of post-mitotic neurons and promotes neurogenesis. Should our hypothesis be confirmed, this splice variant could represent a novel neuroprotective and regenerative therapeutic strategy devoid of the hematopoietic side effects associated with conventional EPO treatment.

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- › Cardiovascular Biomarkers
- › Cardiovascular Imaging
- › Secondary Stroke Prevention

Myocardial Dysfunction and Injury Patterns in Patients with Ischemic Stroke

Cardiac complications after stroke are frequent and of medical relevance as they substantially contribute to stroke-associated disability and death both in the acute and chronic phase after acute ischemic stroke (AIS). About 20% of all stroke patients are reported to suffer a severe adverse cardiac event, such as acute coronary syndrome, heart failure or cardiac arrhythmias within the first few days after the incident stroke. Part of the spectrum of cardiac changes observed early after stroke is elevation of cardiac troponin T (hs-cTnT) which indicates presence of myocardial injury. At the same time, the rate of major adverse cardiovascular events over the course of three years is doubled if patients present this surrogate of myocardial injury in the acute phase after stroke. So far, limited information is available on the assessment of cardiac function or non-ischemic myocardial alterations in patients with AIS. State-of-the-art cardiovascular MRI (CMR) allows to precisely visualize myocardial function and injury patterns. We hypothesize that CMR will reveal myocardial dysfunction and tissue alterations in AIS patients with elevated hs-cTnT. Until now, no study pro-

vided in-depth myocardial tissue characterization by using state-of-the-art feature tracking sequences to study myocardial deformation (global longitudinal strain) together with T1 and T2 mapping sequences and late gadolinium enhancement (LGE) imaging. This approach will also allow to study diffuse myocardial fibrosis as well as edema and inflammation in addition to focal fibrosis (LGE). This project is an explorative analysis within the prospective CORONA-IS trial and the pre-specified »heart-brain-vignette« of the prospective BIH BeLOVE study. As CMR is performed at two different time points (5 days and 90 days) after AIS, we face the unique opportunity to study both the acute and chronic phase after AIS. With this approach, we wish to identify the burden of myocardial injury patterns that underly the high cardiovascular risk after AIS. Furthermore, we hope to use this data in correlation with blood-based biomarkers to improve our understanding of heart-brain-interaction as an underlying pathophysiology in the development of post-stroke cardiac dysfunction.

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- › IgG4-related Disease
- › Autoantibodies and Annexin A11
- › Pancreatic Epithelial Cell Biology

Role of the IgG4-Related Disease Autoantigen Annexin A11 in the Pathophysiology of Type 1 Autoimmune Pancreatitis

IgG4-related disease (IgG4-RD) is a systemic autoimmune condition that targets secretory organs and often mimics other disorders. The pancreas and biliary tract are frequently affected, presenting as type 1 autoimmune pancreatitis (AIP) and IgG4-related cholangitis (IRC). Recent findings identified annexin A11 as the first specific autoantigen in IgG4-RD. This project investigates the role of annexin A11 in AIP pathogenesis, hypothesizing that anti-annexin A11 IgG1 antibodies drive disease by disrupting annexin A11-mediated trafficking of membrane proteins such as ANO1, thereby impairing bicarbonate secretion in pancreatic ductal cells. IgG4 appears to mitigate IgG1-induced effects. The study uses a multifaceted approach – human tissue analysis, cell and organoid models, and in vivo mouse experiments – to characterize annexin A11 function and the pathogenic potential of autoantibodies. Findings aim to enhance understanding of AIP mechanisms, clarify IgG subclass roles, and refine diagnostic and therapeutic strategies for IgG4-RD.

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- › Spinal Cord Injury
- › Spinal Cord Regeneration
- › Neuroregeneration
- › Neurotraumatology

Promoting Neurovascular Regeneration in Spinal Cord Injury via the Cell-Specific Regulation of Ephrin-B2 Signaling

Traumatic Spinal Cord Injury (SCI) is one of the leading causes of disability in the world. A complex pathophysiology and insufficient endogenous regeneration of the spinal cord leave this condition not accessible to curative therapy. The overarching goal of our research is to facilitate endogenous regeneration to ameliorate spinal cord regeneration post SCI. Experimental research in ischemic stroke, a condition which in its pathophysiology resembles SCI, could show the guidance molecule Ephrin-B2 to be associated with a stabilization of the neurovascular unit (NVU). In SCI, the role of Ephrin-B2 is not well-known and an inhibitory role through the induction of astrogliosis is assumed. With this project, we aim to examine the cell-specific role of Ephrin-B2 on neurovascular regeneration post SCI. We hypothesize that 1st endothelial Ephrin-B2 plays a significant role in stabilizing the NVU post SCI and its specific knock-out leads to an aggravation of secondary injury in the spinal cord. 2nd Ephrin-B2 in reactive astrocytes inhibits axonal regeneration and its specific knock-out ameliorates spinal cord regen-

eration. To test these hypotheses, a cell-specific conditional knock-out mouse model is utilized (R. Adams, Münster). The primary endpoint of this project is neurological restitution, and the secondary endpoint is structural regeneration of the spinal cord. Methodically, behavioral analysis using Catwalk® automated gait analysis, magnetic resonance imaging, histological and immunohistochemical analyses, and longitudinal in vivo microscopy will be performed. As the current therapy of spinal cord injury is restricted to supportive therapies, the findings of this study will lead to an enhanced understanding of SCI pathophysiology and spinal cord regeneration with the final goal to develop translational therapies building on this preclinical work in the future. This project is performed at the neurosurgical Spinal Cord Injury laboratory at Charité Berlin in cooperation with the Fehlings Laboratory at the Krembil Neuroscience Center, University Health Network, Toronto, Canada.

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- › Neuroscience
- › Cognition
- › Spatial Navigation
- › Memory Consolidation

MASTERMIND – Mental Schemata Overcome Navigation Deficits

The ability to navigate from memory is a prerequisite for autonomy and survival. Understanding how spatial memories of navigational experiences are formed and modified will help to develop much needed tools to address deficits in memory-guided spatial navigation due to aging and memory disorders.

Recent theories support the idea that memory consolidation is a rather flexible process that can adapt to different contexts by shifting computations between the hippocampus and the neocortex as needed. One contextual factor is mental schemas, which are derived from regularities in prior knowledge and are stored as mental representations in the neocortex. New information that is congruent with a schema facilitates memory consolidation. Faster integration of congruent information occurs by accelerating a shift of computations from the medial temporal lobes to the medial prefrontal cortex. It is conceivable that similar mechanisms apply to spatial representations acquired during navigation in the real world. Therefore, spatial schemas formed by previous

navigation experiences might guide current navigation in familiar and even unfamiliar environments.

Navigational abilities decline with age, and in the most common form of dementia, Alzheimer's disease, navigation deficits occur before other symptoms. In both cases, this is due to early hippocampal degeneration, which impairs the stability of spatial memory representations. In the elderly, a preference for navigation strategies that rely less on the hippocampus appears to be a compensatory mechanism. Another strategy could be the use of spatial schemas, as context changes associated with greater neocortical involvement improve memory-guided spatial navigation in patients with hippocampal lesions.

Using virtual reality and mobile EEG, this project investigates how mental schemas affect memory-guided spatial navigation and whether spatial schemas could help to overcome deficits in memory-guided spatial navigation in older people and Alzheimer's patients.

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› Acute Myeloid Leukemia
› Tumor Immunology

Immunological Signatures of the Human Bone Marrow as Predictors for Therapy Response in AML: Analysis at the Single-Cell Level

Acute myeloid leukemia (AML) is caused by rapid expansion of myeloid blasts in the human bone marrow niche (BM). The individual course of AML is heterogeneous and depends on multiple disease- and patient-specific factors. Therefore, AML long-term survival is still limited and unsatisfactory. Although therapeutic innovation is constantly progressing, conventional chemotherapy with/without subsequent allogeneic hematopoietic stem cell transplantation (allo-HSCT) is still the mainstay of therapy for most AML patients. Over the past years, the concept of AML-induced immunosuppression has been introduced as cofactor of AML pathogenesis. Due to the lack of AML-specific cellular targets only few immunotherapies have emerged. Fortunately, the era of single cell (sc) technologies holds massive potential for in-depth AML characterization and will eventually lead to the development of new, more specific therapy targets. In this project, I combine single cell cellular indexing of transcriptome and epitome by sequencing (CITEseq) with digital deconvolution and spatial methods to detangle the functional status of AML and immune cells in the human bone marrow. In detail, I am interested in the following questions:

I. How does conventional AML induction chemotherapy influence mechanisms of immunosuppression and cell-cell interactions in the human BM niche and are these changes reversible? II. Are specific immunological signatures at baseline associated with relapse or durable remission? III. Is there a characteristic spatial distribution amongst surrogates of these signatures in the human BM? To address these questions, I use cutting-edge single cell methods on two different study cohorts: I. a longitudinal patient cohort under induction chemotherapy and II. a retrospective patient cohort of whom 50% have shown stable long-term remission and 50% have relapsed over time. The latter patients will be assessed at baseline only and were selected due to their homogeneous mutational profile (NMP1+/FLT3-) and the absence of allo-HSCT induced immunological bias. The results of both cohorts will be validated in larger publicly available data sets using digital deconvolution methods. In the long run, this approach is likely to optimize response prediction and shape personalized AML therapy, in particular with regard to choice of optimal consolidation therapy, and development of new targets for AML immunotherapy.

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- › Immunology
- › Aging
- › Senescence & Senolytics

Evaluation of an Anti-Inflammatory Therapy for MINOCA in a Translational Pig Model

Myocardial infarction with non-obstructive coronary arteries (MINOCA) accounts for up to 10% of myocardial infarctions, disproportionately affecting younger women. A predominant cause is cardiac microembolization (CME) due to plaque erosion, which triggers an intense inflammatory response rather than significant vascular obstruction. However, the lack of pathophysiological understanding has hindered the development of targeted therapies.

Using a novel translational porcine model mimicking MINOCA, it was possible to identify leukotriene signaling—particularly elevated LTA₄H and LTB₄—as key drivers of systemic inflammation and cytokine release following MINOCA. The aim of this project is now to evaluate anti-inflammatory therapies targeting leukotriene and cytokine pathways to attenuate CME-induced myocardial damage. Therefore, animals undergoing MINOCA will be subjected to repurposed, FDA-approved, anti-inflammatory drugs and infarct size, cardiac function as well as the inflammatory immune response will be assessed in an acute and chronic setting.

The project leverages an established collaboration between DHZC and ETH Zürich and could enable the identification of the first targeted therapy for MINOCA as a basis for future clinical studies.

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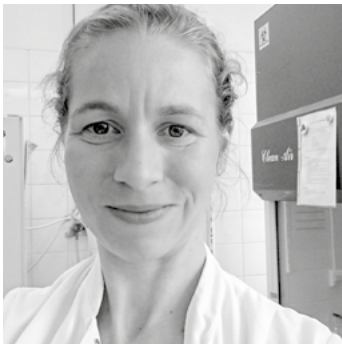
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› Obesity
› Immunology

OncObesity: Influence of Obesity and Metabolic Disorders on the Pathogenesis of Oncological Diseases – an Immunological Approach

Obesity is a growing global health challenge and an independent risk factor for at least 13 types of cancer. The underlying mechanisms remain incompletely understood but likely involve immunological alterations within adipose tissue (AT) as well as systemic metabolic dysregulation. Among the immune cell populations affected by obesity, mucosa-associated invariant T (MAIT) cells – innate-like T cells responsive to microbial metabolites and inflammatory cytokines – exhibit obesity-related dysfunctions that may impair tumour surveillance and promote cancer progression.

This research project investigates how metabolic factors and cancer influence MAIT cell localisation, phenotype, and function, with particular focus on how obesity enhances inflammation and weakens anti-tumour immunity. The aim is to elucidate the metabolic impact on immune-mediated tumour defence, identify predictive markers for early intervention, and characterise AT-resident MAIT cells in a surgical oncology setting.

To this end, patients undergoing abdominal surgery – including bariatric and oncologic procedures – are enrolled in a prospective clinical study. Immune cells isolated from AT and peripheral blood mononuclear cells (PBMCs) will undergo ex vivo profiling via flow cytometry and single-cell RNA sequencing (scRNA-seq). In parallel, in vitro co-culture models incorporating (pre-)adipocytes and cancer cell lines will be employed to assess MAIT cell responses under defined metabolic conditions. Gene expression, cytokine secretion, and cytotoxic activity will be analysed in correlation with clinical parameters such as BMI, cancer type, and patient outcomes.

The translational potential of this project lies in identifying immunological biomarkers predictive of cancer risk and treatment response in metabolically compromised individuals. Moreover, the results may reveal novel therapeutic targets and inform the development of perioperative immunomodulatory strategies tailored to obese cancer patients.

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- › Tissue Engineering
- › Decellularization
- › Recellularization
- › Cell Isolation
- › 3D-Printing

Neo|PancreasPrint – 3D Printed Perfusable Islet-Hosting Tissue Based on Biocompatible Ink Derived from Human Decellularized Pancreatic Tissue

T1DM mostly appears at a young age and involves the autoimmune destruction of insulin-secreting β -cells. The main therapy remains regular substitutions with exogenous subcutaneous synthetic insulin. Despite modern insulin schemas, new drugs and insulin pumps, patients with critical metabolic lability still exist. These open-loop systems require glucose monitoring four to six times per day, leading to extreme reliance on patient compliance. Indeed, achieving the required tight glycaemic control is critical. Hence, closed loop glycaemic control has been developed, in which continuous glucose monitoring is coupled to an automated insulin delivery. However, these devices still require user input and are unable to achieve the level of glycaemic control of islets. One alternative is transplantation of the whole, vascularized pancreas. However, this procedure remains a demanding surgery, associated with complications such as bleeding, thrombosis and transplant pancreatitis. Islet transplantation into the liver has emerged as a minimally invasive alternative devoid of these high surgical risks. Unfortunately, more than 60% of the infused islet mass does not survive and is destroyed. Reasons are poor engraftment and reconstruction of islet niche, failed vascularization and

progressive attrition due to high workload on each islet and risk of immune rejection. Advances to address these issues are being observed in three different fields, such as encapsulation strategies, xenotransplantation and regenerative medicine. The study aims to use human decellularized pancreas-based biocompatible ink to construct the Neo|PancreasPrint, a novel 3D printed, perfusable islet-containing tissue that is vascularized, biocompatible and anastomosable. We believe that, for an optimal islet functionality in vivo, it is important to perfect the ex vivo preconditioning, as it is the only way to incite islet engraftment, while minimizing their workload and stress. In a first step, we propose implementing the Neo|PancreasPrint as a platform to study human islet pathophysiology in relation to organo-typical extracellular matrix-based biocompatible ink - ink compatible to be seeded with cells of choice after the printing process. Secondly, we suggest using the Neo|PancreasPrint as the first human pancreas-based perfusable, anastomosable 3D printed islet-containing endocrine tissue that could prospectively serve the role of an endocrine neo-organ for transplantation.

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- › Bone Metabolism
- › Osteoimmunology
- › Periprosthetic Joint Infections
- › PD-1/PD-L1

Role of PD-1/PD-L1 in the Altered Bone Metabolism in Periprosthetic Joint Infections and its Potential for a Targeted Salvage Therapy

Periprosthetic joint infections (PJI) are a severe complication affecting 1-5 % of primary total knee and hip arthroplasties, despite advances in antibiotics and aseptic techniques. Due to biofilm formation, PJIs are highly resistant to antibiotics and often require complete prosthesis removal and reimplantation. After PJI-related revision, prosthesis failure rates rise significantly – about 20 % due to recurrent PJI and 25 % due to aseptic loosening. The mechanisms behind this increased long-term failure are not well understood. We hypothesize these failures are linked to a dysregulated immune response, leading to increased osteoclastogenesis and impaired bone regeneration.

In a previous study, we observed significant inflammation in the bone. A follow-up study revealed a reduction in bone stock and regenerative capacity that persists despite treatment. This inflammatory upregulation mobilizes monocytes in peripheral blood and increases osteoclast numbers and activity in bone. Prolonged cytokine expression may alter the local immune response, generating myeloid-derived suppressor cells (MDSCs) that hinder clearance of bacterial components. Both mono-

cytes and MDSCs can differentiate into osteoclasts, with preliminary results suggesting their involvement in PJI.

This project aims to clarify the mechanisms of PJI-related prosthesis loosening and evaluate a potential therapeutic target to improve bone health and reduce prosthesis failure rates. We will explore targeting the programmed cell death 1 (PD-1) receptor as a novel therapeutic strategy, investigating its role in septic activation of MDSCs and the potential utility of PD-1 inhibitors to restore bone metabolism. We will assess efficacy and safety in vitro and in vivo. This study will provide essential evidence for the potential use of anti-PD-1 therapy in patients with PJI.

PD-1 inhibition presents an innovative approach for PJI treatment. PD-1 inhibitors have shown safety in systemic infections and may enhance bone health by suppressing excessive osteoclast generation while improving immunocompetence. In summary, targeting PD-1 could offer a new strategy to improve outcomes for PJI patients, a population for whom drug-based therapies are currently lacking.

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- › Monogenic Kidney Disease
- › Genome Editing
- › Base Editing

Genome Editing as a Treatment for Monogenic Atypical Hemolytic Uremic Syndrome

Atypical hemolytic uremic syndrome (aHUS) is a very rare, life-threatening thrombotic microangiopathy mainly caused by uncontrolled complement activation. It is associated with thrombocytopenia, acute kidney injury, and hemolytic anemia. Approximately 50-70% of aHUS patients have an underlying monogenic cause. Variants in the gene encoding complement factor H (CFH) are the most common cause of aHUS in 20-30% of the cases and lead to an overall severe disease course, with high risk for relapses and rapid progression to end stage renal disease (ESRD). Once ESRD is reached, patients require either dialysis or transplantation, which represent an enormous health-care burden worldwide. Eculizumab and novel complement inhibitors such as Ravulizumab have entered clinical applications. It is highly effective for treating acute episodes and preventing relapses but still bears major side effects. In addition, it needs to be administered frequently as a life-long treatment. Thus, a »single-shot« »once-and-done« treatment via CRISPR-based genome editing could be a promising novel treatment strategy for patients suffering from aHUS due to CFH variants. CRISPR base editing combines high efficacy and easy programmability without requiring dou-

ble-strand breaks (DSB) or donor templates. Recent in-vivo applications underscore its potential. We will target different CFH variants by in in-vitro base editing in HEK293T reporter cells using different combinations of guide RNAs and base editors. Targeted amplicon sequencing will assess base editing efficiency. The results will be validated in iPSC-derived hepatocytes after introducing the patient variant. Adeno associated virus (AAV) has successfully been used as a vehicle to express base editors in-vivo. Due to the large size of base editors, split inteins must be employed to distribute the payload of base editor and gRNA on two AAVs. Thus, I will test different splice-inteins and splicing positions to generate a split-intein base editor that sufficiently edits the CFH variants. In the long run, we plan to apply base editing in-vivo to explore if pathogenic variant correction can prevent, halt or reverse disease progression. Overall, this project aims to use innovative CRISPR base editing tools that have been successfully applied to correct other genes, to now correct CFH variants causing aHUS.

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- › Neuromuscular Diseases
- › Proteomics
- › Myositis

Molecular Characterisation of Pathomechanisms in Acquired and Hereditary Myopathies

Idiopathic inflammatory myopathies (IIMs) form a group of autoimmune diseases affecting the skeletal muscle and, to a variable extent, other organ systems (e.g. skin, lungs, heart muscle). While research has shown that the pathogenesis of IIMs is heterogeneous, this fact is not yet reflected by the current classification systems. As a consequence, in many cases the treatment of IIMs follows an »one fits all« approach that does not reflect the complex molecular differences of IIMs. Pilot studies have shown that some subtypes of IIM might even be misclassified in the current diagnostic classifications. In this project, we will perform state-of-the-art untargeted proteomics in a large cohort of patients with IIM to explore the molecular pathogenesis of the different diseases. This data will then be used in the development of a modern, evidence-based classification system (»Myositis 3.0«) that may hold important implications for future therapy strategies (e.g. targeted therapies) in the context of IIMs.

A second focus of my research is the characterisation of mitochondrial dysfunction in the context of IIMs and hereditary myopathies. Mitochondria may show abnormalities caused by primary genetic defects (e.g., mtDNA mutations and deletions), inflammatory processes (e.g., in Inclusion body myositis), and/or by post-translational/-transcriptional mechanisms (e.g. sequestration of transcription factors, e.g. in Myotonic dystrophy type 2). This interplay and the particular role of mitochondria in the pathophysiology of the individual diseases is only incompletely understood. Unraveling the role of mitochondria, however, may open new opportunities for targeted treatment approaches.

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- › Urinalysis

Urine-Derived Regenerative Tubular Epithelial Cells (regTEC): Biomarker and Therapeutic Target in Acute Kidney Injury

Acute kidney injury (AKI) is a major health problem implying significant morbidity and mortality. The care for AKI patients is merely supportive and our understanding of the pathophysiology is limited. One key to improved treatment of kidney disease is new diagnostic methods that allow assessment of individual regeneration potential or risk of progress to chronic kidney disease after AKI. Recently, dedifferentiated kidney tubular epithelial cells (TECs) have been revealed to be essential in injury and repair processes in AKI.

Usually, urine is devoid of cells. But after AKI, patient's urine contains kidney TECs with regenerative potential (regTECs). These cells, so far only detected by single-cell RNA sequencing, have a molecular makeup which is sufficiently preserved in urine to infer data informative about expression patterns in a patient's kidney without biopsies. My project specifically promotes the rapid translation of these findings to a clinically relevant application in three elementary steps:

- 1) The dynamics and prerequisites of the damage-associated occurrence of regTECs in urine will be elicited in a temporally resolved manner with single-cell RNA sequencing and validated separately in kidney tissue.
- 2) The regTEC detection in urine will be made scalable using simplified methodology (flow cytometry), thus enabling the examination of larger cohorts and clinical use of urinary regTECs as a cellular biomarker for AKI recovery.
- 3) Using cell sorting, regTECs will be isolated from urine and made accessible for the evaluation of their regenerative potential in vitro.

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- › Intracranial Hypertension
- › Neuroimaging

Multimodal Predictive Markers in Idiopathic Intracranial Hypertension

Idiopathic Intracranial Hypertension (IIH) is a rare, chronic condition that primarily affects obese women of child-bearing age and is characterized by an increase in intracranial pressure. The etiology of the disease remains unclear. The main symptoms of IIH are therapy-resistant headaches and visual disturbances. About one-third of patients develop permanent visual field defects and reduced visual acuity. While mild cases may respond to drug therapy aimed at lowering intracranial pressure, progressive cases with worsening vision require repeated lumbar punctures or neurosurgical interventions, such as the placement of a ventriculoperitoneal shunt.

Despite the significant increase in IIH incidence, there are currently no early predictors of disease progression. However, early identification of at-risk patients is crucial to prevent visual impairment. Distinct subpopulations and familial forms of IIH, which are associated with more severe clinical courses, have been described. Therefore, the evaluation of predictive markers requires a thorough characterization of the studied patients, which has so far been lacking due to the rarity of the disease.

Since 2019, Charité offers a specialized outpatient clinic for patients with IIH. Between 2004 and 2024, more than 1,000 patients have been treated at Charité, representing one of the largest patient cohorts internationally.

With this interdisciplinary initiative, we aim to address the previously unmet medical need for predictive markers in IIH, thereby improving both the diagnostic accuracy and prognosis for people affected by IIH, as well as enhancing our understanding of the disease. To achieve this, we will collaborate with partners within and outside Charité to investigate the significance of multimodal diagnostic and predictive markers in IIH. Our approach includes comprehensive genetic diagnostics via whole genome sequencing, biochemical biomarkers, and magnetic resonance imaging. Cooperating institutions include the Institute of Medical Genetics and Human Genetics, the Department of Neuroradiology, and the Clinical Research Unit (CRU) at the Experimental and Clinical Research Center (ECRC) at Charité.

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› Autoimmune Encephalitis
› Neuroimmunology
› Humoral Immune Responses

Cross-Reactivity of Antiviral Antibodies Against Neuronal Autoantigens as Cause of Autoimmune Encephalitis

Encephalitis is a rare but serious neurological condition in which inflammation of the brain frequently causes irreversible neurological dysfunction and death. Few years ago, it was shown that these disorders can be mediated by antineuronal autoantibodies, most commonly by those that target the NMDA receptor. Some of the affected patients had previously undergone viral brain infections. However, it has so far remained unclear whether these infections might be causatively related to the autoantibody formation and the antineuronal autoimmunity.

This research project will investigate whether antineuronal autoimmunity can occur through specific cross-reactivity of antiviral antibodies or as an unspecific epiphenomenon. Cerebrospinal fluid and serum biosamples from anti-NMDA receptor encephalitis patients have been collected via the German autoimmune encephalitis network (GENERATE). They will be analyzed using VirScan, a recently developed bacteriophage display technology to derive reactivity profiles of antibody binding against all human-pathogenic viruses at the resolution of single

viral peptides. These investigations will be performed in a rotation to the lab of VirScan co-founder Benjamin Larman at the Johns Hopkins University School of Medicine. Afterwards, the binding epitope data will be used to isolate monoclonal antibodies against selected viral peptides from stored cells of autoimmune encephalitis patients with the history of viral encephalitis to further investigate antineuronal cross-binding on the level of monoclonal antibodies.

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- › Ischemic Stroke
- › Magnetic Resonance Imaging
- › Computer Tomography

Prediction of Long-Term Outcome in Acute Ischemic Stroke Patients Using Lesion-Network Mapping

Despite the development of successful acute treatments for ischemic stroke, stroke remains a leading cause of lasting disability and permanent functional dependence worldwide. Not only can an ischemic stroke lead to severe motor deficits impeding mobility, there is growing evidence that stroke can lead to more complex and long-term symptoms including chronic depression and progressive cognitive decline; all of which have a substantial impact on the patients' rehabilitation success and quality of life.

Due to the frequency and complex course of these post-stroke symptoms, understanding the underlying mechanisms is essential to develop accurate prediction models to identify patients at risk to ultimately introduce targeted treatments. A relatively new methodology, called lesion network mapping (LNM) using the so-called human connectome offers a new and accessible way to evaluate anatomical as well as functional connectivity across lesions using magnetic resonance (MR) sequences acquired in routine clinical diagnostics. The consider-

ation of lesion functional connectivity has the potential to substantially increase the accuracy of our outcome prediction models in the field of stroke and subsequently allow us to optimize individualized treatment options for stroke patients.

Therefore, the current project will pool MR and clinical data of >1500 ischemic stroke patients from several independent large, well-characterized, prospective stroke cohorts to 1) identify the functional networks associated with early motor recovery, as well as the development of post-stroke depression and cognitive impairment – using LNM with the connectome. Furthermore, 2) we aim to validate these findings using predictive modelling in a large independent cohort of stroke patients with long-term follow-up. Our main hypothesis is that with a systematic approach using large datasets (including clinical & preclinical data), we can apply LNM to predict post-stroke outcome, including motor recovery, functional dependence, as well as post-stroke depression and cognitive decline.

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- › Headache
- › Stroke
- › Reversible Cerebral Vasoconstriction Syndrome
- › Takotsubo Syndrome

Acute Neuro-Cardiac Stress Syndromes – Identification of Clinical and Proteomic Clusters by Machine Learning Methods

Reversible cerebral vasoconstriction syndrome (RCVS) and Takotsubo syndrome (TTS) are potentially life-threatening stress-associated conditions. RCVS is a neurovascular headache disorder leading to stroke in up to one-third of cases. TTS as a cardiomyopathy is associated with serious complications such as cardiogenic shock in 20%. Although both conditions are increasingly recognized, their pathophysiology and clinical heterogeneity, ranging from benign to fatal forms, are incompletely understood.

Current evidence supports the hypothesis that RCVS and TTS belong to the same spectrum of stress-associated syndromes, with numerous clinical and pathophysiological similarities between the two syndromes, and cases of concurrent or consecutive occurrence of RCVS and TTS. However, the overlap of RCVS and TTS has not been systematically explored.

A more comprehensive understanding of different clinical phenotypes and their prognosis could improve diagnosis and clinical management. To this aim, it is necessary to identify clinical predictors and diagnostic markers for

(1) identification of patients at risk for complications, and (2) the occurrence of combined neurocardiac stress syndromes.

In this project, we will use machine learning methods to analyze clinical data and proteomic profiles of deeply phenotyped patients with RCVS to identify clinical and proteomic clusters with prognostic relevance, and to develop algorithms for classification into these clusters. In a second step, within an interdisciplinary multi-centric collaboration, we will compare these clusters between patients with RCVS and TTS.

The findings of this project will be relevant to the pathophysiological understanding of the spectrum of stress-associated neuro-cardiac diseases, their diagnosis, prognosis, and the identification of potential targets for treatment and prevention.

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- › Immunology
- › Inflammatory Bowel Disease
- › Penetrating Crohn's Disease

Deciphering Molecular Mechanisms of Fistula Formation in Crohn's Disease for Targeted Interventions

Crohn's disease (CD) is a chronic inflammatory bowel disease characterized by a heterogeneous disease course, often leading to fibrosis and fistula formation, significantly impairing patients' quality of life. Despite advances in targeted therapies, a comprehensive understanding of the molecular mechanisms driving these phenotypes remains elusive, preventing effective personalized treatments. This study aims to elucidate the key mechanisms of epithelial-stromal-immune cell cross-talk promoting epithelial-mesenchymal transition (EMT) and extracellular matrix (ECM) remodeling in CD, thereby facilitating the development of a novel molecular taxonomy for precise therapeutic interventions. Utilizing an established cohort from the IBDome project, we will integrate bulk-RNAseq, spatial transcriptomics, interstitial microenvironment profiling, and imaging mass cytometry (IMC) data to identify molecular signatures distinguishing the B1 (non-stricturing/non-penetrating), B2 (stricturing), and B3 (stricturing/penetrating) CD phe-

notypes. Additionally, an assembloid model of CD-associated fistulas will be developed to validate key pathways and investigate potential therapeutic targets. By addressing the unmet need for phenotype-specific treatments in CD, this project holds substantial translational potential for improving clinical outcomes and advancing personalized medicine.

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- › Valvular Heart Disease
- › Interventional Therapy
- › Echocardiography

New Advances in Tricuspid Regurgitation

Tricuspid regurgitation (TR) is one of the most common structural heart diseases and is associated with high morbidity and mortality (Singh et al., Am J Cardiol, 1999; Nath et al., JACC, 2004). In recent years, various interventional therapies for TR, such as percutaneous annuloplasty and transcatheter edge-to-edge repair, have been developed (Praz et al., EuroIntervention, 2021). Particularly, older multimorbid patients who were previously managed symptomatically may benefit from minimally invasive therapies. The accurate quantification of TR using echocardiography is a prerequisite for determining the treatment indication. However, previous echocardiographic measurements of the regurgitation orifice area (EROA) and volume (RegVol) based on the »PISA« method are prone to errors due to the tricuspid valve anatomy and underestimate the TR severity by approximately 50 %. An angle-corrected formula for calculating EROA may address this diagnostic gap (Tomaselli et al., Eur Heart J Cardiovasc Imaging, 2022).

In the current project, titled »New advances in tricuspid regurgitation«, we aim to evaluate (i) the prevalence and prognosis of TR in different heart failure phenotypes, (ii)

the outcome of patients after different types of interventional TR therapy, and (iii) the prognostic value of angle-corrected quantification of TR.

To accomplish this, we prospectively enrolled 1000 patients with heart failure who underwent echocardiographic examination to quantify TR. Follow-up telephone visits are conducted at three, six, twelve, and twenty-four months. Additionally, patients who undergo interventional TR therapy are enrolled in the »Berlin Registry of Right Heart Interventions«. Follow-up examinations, including echocardiography, electrocardiograms (ECG), laboratory measurements, and symptom assessments, are performed at two, twelve, and twenty-four months. Offline echocardiographic analysis enables further TR assessment using the angle-corrected formula for calculating EROA, allowing evaluation of the prognostic value of the corrected TR grading in both cohorts.

The overall objective of this project is to enhance the diagnosis and treatment of TR patients.

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- › Liver Transplantation
- › Organ Recovery
- › Aging

Alleviation of Senescence Induced Ischemia-Reperfusion in Liver Grafts of Elderly Donors [SenEx]

Ischemia-reperfusion injury (IRI) is a major complication in liver transplantation, leading to significant cellular damage and impacting graft function and survival. Reactive oxygen species (ROS) are central to this damage, with their overproduction during reperfusion causing oxidative stress and cellular injury. The role of NAD⁺, a critical coenzyme involved in redox reactions and energy metabolism, has emerged as a key factor in regulating ROS levels and mitigating cell damage. This study aims to explore the mechanisms by which NAD⁺ influences ROS production and to evaluate potential therapeutic interventions to enhance liver graft outcomes.

This ongoing study employs a comprehensive approach combining molecular pathway analysis with an advanced small animal perfusion system. The molecular analysis focuses on how NAD⁺ depletion impacts autophagy and ROS production. In parallel, an experimental perfusion system is used to simulate ischemia-reperfusion con-

ditions in isolated liver grafts. This system includes continuous flow and oxygenation to mimic physiological conditions and allows for the assessment of NAD⁺ supplementation and other interventions on ROS levels and cell viability.

Preliminary findings indicate that NAD⁺ depletion, commonly observed in aging and stress conditions, significantly reduces autophagy, leading to increased ROS production which contributes to cell necrosis. Early results suggest that NAD⁺ plays a crucial role in mitigating ROS-induced cell damage during liver transplantation. Therapeutic strategies aimed at preserving or enhancing NAD⁺ levels could improve graft function and patient outcomes post-transplant.

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- › Muscular Dystrophy
- › Protein Aggregation
- › Muscle Ageing

Therapeutic Strategies in Myofibrillar Myopathies – a Model for Protein Aggregation Diseases?

Myofibrillar myopathies are a clinically and genetically heterogeneous group of neuromuscular diseases often leading to progressive skeletal myopathy and cardiomyopathy. The exact molecular pathogenesis of myofibrillar myopathy remains to be fully understood, though most mutations generate myofibrillar structural changes with abnormal intracellular aggregates of the intermediate filament desmin and other proteins.

These protein aggregates have been likened to aggregates and amyloids found in Alzheimer and Parkinson diseases, systemic amyloidosis, amyotrophic lateral sclerosis (ALS), Huntington disease and type II diabetes, but also share properties with prions, as found in Creutzfeldt-Jakob disease. Protein misfolding and the amyloid phenomenon are increasingly being recognized as a major disease driving mechanism, yet no therapies exist to date to cure the associated diseases.

I created and characterized a CRISPR-Cas9 engineered rat with an exchange of arginine to proline at position 349 (R349P) of the desmin gene; orthologous to the most prevalent desmin mutation in humans. This is the first rat model of myofibrillar myopathy. The rats exhibit both skeletal and cardiac myopathies with sarcoplasmic aggregates and signs of injury and remodeling, as well as ventricular arrhythmias, a disorganization of the cardiac tissue architecture and fibrosis.

This Clinician Scientist project explores several therapeutic approaches targeted against protein aggregation in a rat model of desminopathy, the most common myofibrillar myopathy, to improve muscle function.

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- › Implementation Research
- › Mixed-Methods Research

Improving Community-Based TB Care in Resource-Restricted Settings

The shortage of facility-based healthcare workers is a pressing global issue, especially in low- and middle-income countries. This problem is expected to further worsen due to factors such as population growth and the increasing prevalence of chronic diseases in the years to come, posing a significant threat to the progress of achieving universal health coverage. Madagascar, a low-income country facing substantial challenges in delivering healthcare services to its population, contends with a significant burden of Tuberculosis (TB). Despite the ongoing efforts of the Malagasy national TB program (NTP) and non-governmental organizations to offer free TB care to all, TB incidence continues to be high. One critical bottleneck lies in the absence of a well-structured Community Health Worker (CHW) strategy within the NTP activity plan, impeding evidence-based decision-making, the coordination of prevention and control initiatives, and the enhancement of program effectiveness. Community Health Workers (CHWs) are community members who receive training to deliver health promotion, education, and basic care services. They play a vital role in various contexts by acting as a bridge between communities and the formal healthcare system.

In the battle against TB, CHWs can actively engage in tasks such as case finding, contact tracing, and treatment follow-up. Our project aims to design, implement, and assess the effectiveness of an evidence-based strategy aimed at empowering CHWs to provide TB care services in Madagascar. This initiative will involve a comprehensive mixed-methods research approach, engaging CHWs and key stakeholders responsible for TB care policies and CHWs management. The study comprises several components, including a mixed-methods formative research phase, strategy workshops guided by design-thinking principles, the implementation of the new strategy in a pilot area, and an impact assessment of the developed strategy's influence on the availability of TB care services, using both quasi-experimental and qualitative methods. All activities will be conducted collaboratively with Madagascar's NTP and local civil society organizations, with the potential to scale up the intervention if proven effective. In essence, this project aligns with global efforts to combat TB and improve healthcare access for marginalized populations.

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Fields of Research

- › Neurology
- › Immunology
- › Oncology

Immune Checkpoint Inhibitor-Induced Peripheral Nervous System Toxicity – Mechanisms, Prevention, Prediction

Immunotherapies have revolutionized cancer treatment. However, 1 in 15 patients develops severe neurological adverse events for which targeted treatment options and predictive markers are missing. Within my research project, I establish the first animal model of neurological immune-related adverse events (irAE-n) and analyze patient-derived immune cells driving irAE-n to identify the underlying disease mechanisms and test new therapeutic approaches. In addition, I investigate whether genetic markers or specific antibodies in the peripheral blood may be used to predict neurological adverse events. My overall goal is to prevent neurological side effects and thereby improve the quality of life of patients with cancer.

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- › Cholestatic Liver Diseases Mainly
Primary Sclerosin Cholangitis
- › Microbiology
- › Gastrointestinal Oncology

Multimomics and Functional Characterization of the Mutational Landscape in Small Intestinal Neuroendocrine Tumors

The intestinal epithelium is characterized by rapid self-renewal, driven by crypt-base stem cells that express the R-spondin receptor Lgr5. Stem cell turnover and differentiation into various intestinal cell types have been recapitulated using organoids and, recently, also growth factor conditions to culture hormone-producing enteroendocrine cells (EECs) have been revealed. Characterized by a high level of complexity, EECs can dedifferentiate and form intestinal neuroendocrine neoplasms (NENs), a set of rare but lethal tumors with a concerning increase in incidence. Small intestine neuroendocrine tumors (siNETs) in most cases already present with metastases at the time of initial diagnosis. Their grading is mainly based on the Ki-67 proliferation index, which determines therapeutic procedure and ultimately patient survival. At present, the development of more effective therapies and prognostic markers has been limited partly by a lack of studies aiming to understand the biology of these rare tumors. In addition to small patient numbers, the

absence of in vitro and in vivo models that accurately represent these tumors constitute a limitation in this field. To gain a deeper understanding of siNET biology, our group has launched a multimomics project, in which whole genome sequencing (WGS) and transcriptome analysis has been performed. Our preliminary data hint towards an important role of specific mutations. By using organoid culture system technologies, this study aims to understand the functional impact of specific mutations in siNETs and to explore their role for EEC survival, proliferation and dissemination.

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- › Uveal Melanoma
- › Cancer Immunotherapy
- › Cancer Immunomodulation

Identification of Targets and Biomarkers for Innovative Uveal Melanoma Immunotherapy

Uveal melanoma (UM) is an aggressive intraocular tumor that in terms of biology and clinical behavior is markedly distinct from its cutaneous counterparts. Despite highly effective eye-preserving radiation therapies such as proton beam therapy (PBT), systemic dissemination occurs in ~50% of patients through early micrometastases. There is no established (neo)adjuvant therapy to prevent metastatic spread. Although recent immunotherapeutic advancements have been made, prognosis of metastatic UM remains dismal due to a lack of effective therapies for the majority of patients. Various factors have been identified that lead to low immunogenicity and the development of an immunosuppressive tumor microenvironment, however there is a high need for a better understanding of the complex immune-evading mechanisms as a prerequisite for novel therapeutic strategies in UM. Together with the Helmholtz-Zentrum Berlin, the Charité is one of the largest PBT centers worldwide. In this project we will characterize the cellular ecosystem

in UM and investigate the immune modulating effects of PBT on the tumor microenvironment and decipher mechanisms of disease progression and immune escape. For this, we will perform single-cell multi-omic mapping of the UM tumor immune contexture using high-dimensional spectral flow cytometry complemented by single-cell transcriptomic and proteomic characterization and spatial profiling in a comparative approach of PBT-pretreated UM tumors (with secondary surgical resection) and treatment-naïve UM tumors (obtained through primary enucleation). With this research, we aim to identify immunomodulatory targets and corresponding predictive biomarker candidates for therapeutical interventions in the neoadjuvant/adjuvant setting to develop novel therapeutic strategies to reduce the risk of metastases.

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› Migraine

› Headache

› Sex Hormones

Migraine across the Female Lifespan: Influence of Sex Hormones on Calcitonin Gene-Related Peptide (CGRP) in Tear Fluid and Plasma

Migraine prevalence in women is three times higher than in men. Hormonal fluctuations play a crucial role in the generation of migraine attacks. According to the estrogen-withdrawal-hypothesis, a drop in estrogen concentrations can trigger migraine episodes. In line with this hypothesis, migraine frequency and severity are higher during the perimenstrual phase of the menstrual cycle but also in the perimenopausal period before hormonal stabilization at an older age. The pathophysiological mechanisms leading from hormonal changes to the development of migraine attacks are still widely unknown. The neuropeptide Calcitonin Gene-Related Peptide (CGRP) has a key role in migraine initiation. During a migraine attack, CGRP is released from trigeminal afferents and triggers an inflammatory response. Preliminary preclinical research suggests that sex hormones fluctuations can lead to activation of the trigeminovascular system and release of CGRP, which may explain the high prevalence of migraine in women of childbearing age. One-third of all women of childbearing potential in Europe take oral contraception, most commonly combined oral contraceptives (COC). The regular intake of a COC leads to suppression of the physiological hormonal fluctuations. The effect of COC on migraine is highly variable, with

migraine attacks occurring most frequently during the seven-day hormone-free interval. The exact association between COC intake and CGRP release remains to be determined. From a methodological angle, the accurate measurement of CGRP in peripheral blood samples is challenging. CGRP has a short half-life time and is subject to dilution effects after release. The measurement of CGRP in tear fluid has recently been proposed as a non-invasive and more direct method due its spatial proximity to the trigeminal nerve. This research project aims to elucidate the complex relationship between sex hormones and CGRP in different hormonal states across the female lifespan (regular menstrual cycle, COC, postmenopause). Key research questions are: 1) How do different hormone profiles affect CGRP release? 2) Does the impact of sex hormones on CGRP differ between patients with migraine and healthy controls? 3) To what extent is CGRP measurement in tear fluid feasible for clinical practice?

The findings of this project could help to explain the changes in migraine frequency in the course of life of women and specifically during the menstrual cycle and under contraceptive treatment.

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- › Neurology
- › Omics
- › Stem cells
- › Sensory Neurons

Modelling Chemotherapy Induced Neurotoxicity with Patient-Specific Induced Pluripotent Stem Cell-Derived Sensory Neurons

Chemotherapy-induced polyneuropathy (CIPN) affects about two thirds of patients treated with neurotoxic chemotherapy, frequently leading to dose reduction, early treatment termination and potentially irreversible neurologic sequelae in a growing number of long-term survivors of malignant diseases. Despite recent advances in cancer immune therapy, classical antineoplastic therapies remain pivotal in the standard of care of various solid malignancies with millions of treated patients annually. The lack of predictive biomarkers, preventive or effective symptomatic treatments make CIPN a significant unmet medical need. To define the individual molecular mechanisms leading to neurodegeneration, we combine a clinical study with a patient-specific neuronal disease model, multi-omics and bioinformatic analyses: We derived iPSC from patients in a clinical cohort CICARO (NCT#02753036), where breast and ovarian cancer patients receiving antineoplastic therapy underwent comprehensive neurological phenotyping for the development of neuropathy, including the assessment of sensory loss or pain, clinical electrophysiology and serum

biomarkers. Building on this cohort, we developed a fully human and patient-specific in-vitro model of neurotoxicity using iPSC-derived sensory neurons from patients who either developed CIPN or were spared from side effects. In cooperation with the Core Facilities Stem Cells, Proteomics, Metabolomics, Genomics and Bioinformatics, we combine unbiased RNA sequencing, deep proteome and lipidome analyses to define predictive biomarkers as well as neurodegenerative and neuroinflammatory cascades of neurotoxicity that partly explain clinical symptoms of sensory loss or pain in the patient. To confirm robustness of the molecular pathways, results are confirmed by cross-validation in cultivated primary human neuronal tissue derived from the Charité Brain Bank. The adoption of these models to other diseases and symptoms of the peripheral nervous system, such as axonal polyneuropathies and chronic neuropathic pain, will enable the development of novel predictive biomarkers and new neuroprotective treatment strategies.

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- › Rare Disease
- › Pediatric Liver Disease
- › Multi-Omics
- › Translational Medicine



friede springer stiftung



Dissecting and Intercepting the Course of Biliary Atresia – 4D Inflammation Phenotyping and Validation for Immunotherapy

Biliary atresia (BA) is a rare, severe liver disease in neonates characterized by fibro-inflammatory obstruction of the bile ducts, leading to cholestasis, fibrosis, and, if untreated, end-stage liver disease. The only treatment option, a surgical removal of the extrahepatic bile ducts (»Kasai« portoenterostomy) has limited success, with most patients necessitating liver transplantation within the first two years of life. The overall pathogenesis of BA remains poorly understood; however, dysregulated immune responses involving various cell subsets have been described, suggesting that immunomodulatory treatments hold promising therapeutic potential. This project aims to (1) elucidate the fibro-inflammatory landscape of BA, (2) identify immune signatures as predictors of clinical outcomes and (3) ultimately validate targeted immunomodulatory therapies.

Integrated analysis of spatial transcriptomics and multiplex immunofluorescence (mpIF) will be applied to characterize the liver immune landscape from infants with BA and cholestatic controls. Paraffin embedded liver specimens will be obtained from BA patients with

different clinical outcome after »Kasai« (liver transplantation vs. native-liver survival) and at different time points to enable 4D mapping of the liver immune environment. After identification of relevant immune targets, immunomodulatory therapies will be validated in a rhesus rotavirus (RRV)-induced mouse model of BA, as well as in human cholangiocyte and multicellular liver organoid models.

BA is the leading indication for pediatric liver transplantation, highlighting the urgent need for effective treatment options. By unraveling the underlying immune interactions in BA, this project aims to discover a novel immunomodulatory treatment that could intercept the inflammatory bile duct obstruction, prevent fibrotic remodeling and instead enable liver regeneration, thus finally restoring liver function. Additionally, insights gained from BA pathogenesis will help to understand general mechanisms of liver fibrosis and regeneration and potentially improve treatment approaches in many other chronic liver diseases.

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- › Molecular Genetics
- › Genome Biology
- › Rare Diseases
- › Limb Malformations

Start Codon Variants and Their Molecular Consequences for Genetic Diseases

The establishment of next generation sequencing (NGS)-based testing methods in molecular genetic diagnostics has revolutionized genetic diagnostics over the last decade. Although this has solved many unexplained cases of genetic diseases and numerous new disease genes have been discovered, around 2/3 of all cases remain unsolved. A central problem of NGS-based molecular genetic diagnostics is the identification of the one actual causative variant among many harmless deviations in the genome. The interpretation of stop codon variants, for example (occurrence of a premature stop codon through stop-gain or frameshift variants and thus premature termination of protein biosynthesis or degradation of the defective mRNA through nonsense mediated decay, and thus complete loss of function (LOF)) is now diagnostically simple and has been very well investigated scientifically. Start codon variants (i.e. mutations of the start codon ATG) could have similar effects, but have been studied sparsely, so that their interpretation is still difficult and they are therefore probably also underdiagnosed. This is also due to the greater number of possible molecular effects of the loss of an ATG start

codon and their difficult predictability. For example, a complete loss of function can occur if no alternative start codon is found or used. However, translation may also fall back on alternative start codons that lie within the original reading frame. These can then lead to residual proteins with different degrees of N-terminal truncation, which may be stable or unstable and show reduced function or a gain of function. Therefore, the aim of this project is to characterize the molecular effects of start codon variants in detail and to analyse their respective clinical-genetic significance. In addition, a bioinformatic program is to be established with which these effects and thus the clinical significance of the respective variants can be predicted bioinformatically. As a starting point, start codon variants already identified in preliminary work will be further investigated experimentally. In addition, this project will draw on the large amount of existing data and samples available in the institute of genetics. This allows further cases with start codon variants to be systematically identified and characterized.

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Fields of Research

- › Deep Brain Stimulation
- › Synaptic Plasticity
- › Human Single Cell Research

Unlocking the Synaptic Potential of Deep Brain Stimulation in Parkinsons Disease

Deep Brain Stimulation (DBS) of the subthalamic nucleus (STN) provides symptomatic relief in Parkinsons Disease (PD), but symptoms reoccur once stimulation is turned off. Understanding the DBS mechanism of action at the single neuron level may be critical to seize the full potential of DBS and achieve long-lasting effects beyond the stimulation period. The innate neuronal plasticity of the stimulated circuit can only be revealed upon stimulation. Thus, further refinements of DBS paradigms are in dire need of characterisations of on-stimulation circuit read-outs. Evoked resonant neural activity (ERNA), an evoked potential signature elicited shortly after DBS pulses has been shown to predict therapeutic efficacy. In recent work, we have developed a multimodal strategy to decode dynamic synaptic circuit engagements that lead to the emergence of ERNA (Steiner et al. 2024; Nature Communications). In the proposed project we test the plasticity of STN efferents (outgoing neuronal projections), which may be critical for the neurocircuit activation (and circuit plasticity) that is reflected by ERNA.

In rodents, novel patterns of DBS have been shown to outperform conventional forms of DBS when they target the individual plasticity of STN efferents and consider single-neuron responses (Spix et al., Science, 2021). To date, STN efferent plasticity has not been directly recorded in humans. The projects outlined by the application at hand aim to fill this gap, thereby providing human single neuron evidence for the DBS mechanism of action.

We are aiming to establish a translational research pipeline that builds on 1) mechanistic interrogation of synaptic plasticity of STN efferents in PD patients at single neuron resolution during DBS implantation surgery and 2) establishing simultaneous testing of circuit-plasticity as indexed by evoked potential signatures (ERNA-amplification). Ultimately, we aim to design physiology-guided stimulation paradigms that consider dynamic plastic properties of the engaged synaptic circuits.

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- › Cystic Fibrosis
- › Pediatric Lung Disease
- › Gut-Lung-Liver Crosstalk
- › Multi-Omics



Risk Factors and Novel Therapeutic Targets for Early Cystic Fibrosis Disease Through Deep Phenotyping and Multi-Omics Approaches

Cystic fibrosis (CF) is a rare, genetic multisystem disease affecting the lung, but also liver, pancreas and intestines shortly after birth. Since causal therapies with CFTR modulators have little effect on important extra-pulmonary manifestations, these organ affections are still relevant and have become more crucial for long-term outcomes. Knowledge explaining the divergent inter-individual organ involvement and the large known heterogeneity in disease severity remains limited. Furthermore, little is known about organ crosstalk and how dysfunction of one organ affects other organs. To examine the organ crosstalk and identify (predictive) biomarkers with an integrated analysis, we have set up a comprehensive prospective, longitudinal, observational, multicenter TRACK-CF study cohort. It includes young children with CF undergoing regular study visits and allows deep phenotyping of early extra-pulmonary and pulmonary morphological and functional changes (i.a. by ultrasound, MRI, spirometry, multiple-breath washout) from diagnosis onwards. Parallel collection of biomaterials in a bio-

bank provides a unique opportunity to search for disease-promoting pathways and therapeutic targets by multi-omics approaches. Our objective is to determine individual extra-pulmonary disease severity and its risk factors in early CF disease. We aim to disclose interactions between intestinal and liver manifestations and lung disease and the treatment effects of CFTR modulators in children with CF using airway and stool microbiome and serum metabolomics and proteomics. We believe that longitudinal deep phenotyping of individuals in the TRACK-CF cohort coupled with regular biomaterial sampling of affected localizations will offer new insights into the development, organ crosstalk and response to treatment in children with CF.

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- › Pathophysiology of Pneumonia
- › Ventilator-Associated Pneumonia
- › COVID-19

Immune Cell and Microbiota Analyses to Identify Risk Factors and Biomarkers for Ventilator-Associated Pneumonia

Approximately 10% of patients admitted to the intensive care unit (ICU) who are newly ventilated will develop ventilator-associated pneumonia (VAP); a condition associated with prolonged hospital stay and high morbidity and mortality. The pathophysiology of VAP is not well understood, but likely involves microbiota perturbations and related impairment of colonization resistance and antibacterial defense. Current diagnostic criteria for VAP have low specificity and sensitivity leading to undertreatment and adverse outcomes or overtreatment, associated with microbiota perturbations and emergence of multi-drug-resistant pathogens. More clearly molecularly defined diagnostic criteria may provide substantial improvements over current diagnostic strategies. Moreover, there is an urgent medical need for the discovery of novel prophylactic strategies.

Our main hypothesis is that we will be able to detect alterations in the molecular signatures of respiratory

and peripheral blood immune cells, the microbiome, and the metabolome, preceding VAP. Our secondary hypothesis is that these signatures, across these distinct cellular and molecular compartments, are further interlinked and may co-evolve over the clinical course.

To test these hypotheses, we conduct a longitudinal, observational clinical cohort study enrolling patients recently admitted to the intensive care unit. We retrospectively identify patients developing VAP (cases) and those who don't (controls). Biological samples of cases and controls will be comparatively analyzed by single cell multi-omics, metagenome sequencing, metabolomics, and integrative computational methods. Thus, we aim to gain new insights and hypotheses about the pathophysiology and risk factors of VAP and identify potential new biomarkers and targets for novel prophylactic strategies or diagnostic tests.

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- › Secondary Immunodeficiency
- › Stem Cell Transplantation

joséCARRERAS
LEUKÄMIE-STIFTUNG

Shaping the BCR Repertoire: Donor and Recipient Interplay in Complications after Allogeneic Stem Cell Transplantation

Allogeneic stem cell transplantation (ASCT) is the only curative treatment option for many hematologic malignancies. Effective immune reconstitution is crucial to achieving both a graft-versus-leukemia effect and a functional immune defense. B cells, as key players in adaptive immunity, contribute to both physiological and pathological processes post-transplant. Recent studies highlight the role of impaired B cell reconstitution in the pathophysiology of graft-versus-host disease (GvHD), with evidence suggesting that the coexistence of patient- and donor-derived B cells is associated with GvHD occurrence.

Our preliminary work demonstrated that while the majority of post-transplant B cells originate from the donor, the B cell receptor (BCR) repertoire reverts to a pre-transplant, patient-like profile, suggesting a shaping of the receptor repertoire by the patient's antigen environment. This project aims to characterize B cell reconstitution

longitudinally in donor-recipient pairs undergoing ASCT, investigating chimerism, IGHV gene usage, and BCR phylogenetics. By correlating measurable B cell dynamics with clinical outcomes such as GvHD, this study seeks to deepen our understanding of immune reconstitution post-ASCT.

The insights gained from this large-scale investigation are expected to significantly enhance our understanding of immune dynamics post-ASCT. By closing existing knowledge gaps and uncovering the mechanisms underlying B cell reconstitution, the project may identify critical targets for intervention, in the long term enabling the development of individualized treatment strategies.

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Fields of Research

- › T cell Therapy
- › Neurologic Autoimmunity
- › Drug Development

T Cell Engager as Targeted Therapy for MuSK-Associated Myasthenia Gravis

Myasthenia Gravis (MG) is a chronic neuromuscular disease caused by antibodies against the neuromuscular endplate. A particularly severe subtype is caused by antibodies against muscle-specific kinase (MuSK). MuSK is essential for the integrity of the neuromuscular endplate and in particular for the functional transmission of excitation. MuSK antibodies disrupt this process, which leads to exercise-induced muscle weakness in affected patients. This mainly affects the respiratory muscles and the throat muscles required for swallowing and speaking, which is why MuSK patients have a high risk of myasthenic crises with respiratory depression requiring intensive care.

The aim of this project is to develop a T cell engager based on BiTE technology that specifically mediates the depletion of the disease-causing B cells. We have previously pursued a similar approach using so-called chimeric autoantibody receptors (CAARs). These CAARs

were integrated into the genome of autologous T cells by a viral vector, which, despite their impressive therapeutic potential, has several disadvantages. The production of CAAR T cells is very time-consuming and cost-intensive and is associated with high regulatory requirements. In contrast to CA(A)R T cells, T cell engagers are significantly cheaper to produce and are also much safer for patients. Given our extensive experience with CAAR technology, we expect to make rapid progress in the development of this new generation of T cell therapy. Ultimately, we aim to translate this technology to other neurologic diseases and beyond.

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- › H. Pylori
- › Gastrointestinal Inflammation
- › Gastrointestinal Oncogenesis
- › Cell Biology

Unleashing Mutated Cells – How H. Pylori Infection Drives Gastric Carcinogenesis

Gastric cancer (GC) is one of the most common and deadly cancers worldwide. Although the main risk factor is infection with the chronic gastric pathogen *Helicobacter pylori*, the mechanisms through which it promotes transformation remain unresolved. Human GC is epidemiologically associated with mutations in genes such as APC or CDH1 and GC tumors are enriched for mutations in genes such as TP53 and KRAS. These changes promote GC development in a stepwise fashion, allowing cells to accumulate mutations and gain an advantage over healthy cells. However, a dominant genetic pathway has not yet been identified. Modelling GC in mice has only recently become possible with the identification of *Anxa10* as a stomach-specific marker that can be used as a promoter. This revealed that individually the above-mentioned GC-associated mutations are not sufficient to induce gastric lesions in uninfected mice but that a combination of at least three is required. Most recently, a large biobank study revealed that *H. pylori*-infected

individuals with germline mutations in BRCA genes are at high risk for developing GC. In the context of infection-induced DNA damage, loss of BRCA may lead to disruption of genetic integrity. Yet, the signaling events and genetic pathways as well as their interplay during GC development upon infection have not been explored experimentally. I hypothesize that the highly organized, adaptive response of the gastric epithelium to infection may be maladaptive if GC-associated mutations (e.g. BRCA, APC, CDH1) or mutations typically found in tumors (e.g. TP53, KRAS) are present. The interaction with *H. pylori* may lead to an increased mutational rate (with accelerated growth) or to a competition advantage e.g. via generation of less differentiated cells that escape the normal mechanisms of tissue control leading to malignancy. I want to study the interaction of *H. pylori* with gastric mutagenesis and competition advantage in cells harboring single mutations. I propose the existence of infection-associated oncogenic routes.

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- › Schizophrenia Spectrum Disorders
- › Psychosis
- › Negative Symptoms
- › Clinical Interventions

Oxytocin-Augmented Group Psychotherapy for Patients with Schizophrenia

The effectiveness of current treatment options for socio-cognitive deficits and negative symptoms (NS) in schizophrenia spectrum disorders (SSD) remains limited. The cause of NS is thought to be an interference between the mesocorticolimbic dopamine system for social reward expectancy and the network for socioemotional processes. Oxytocin (OXT) may enhance functional connectivity between these neuronal networks. Lower plasma OXT levels correlate negatively with NS severity and deficits in social cognition in SSD. It has been shown that intranasal OXT administration improves social cognition, including empathy, in healthy subjects but in SSD results are inconsistent. According to the social salience hypothesis, the effect of OXT varies depending on the social context and individual factors. Also, OXT-mediated effects on psychopathology, NS, and empathy may depend on genetic variants of OXT receptors (OXTR). In a pilot study, we demonstrated a reduction in NS by OXT administration in a positive social context in SSD. We also demonstrated that NS and other symptoms improved after mindfulness-based group psychotherapy (MBGT). The aim of this study in individuals with SSD is to examine the effect of combining OXT administration with MBGT

on NS, empathy, affect, and stress. The main hypothesis to be tested is that the use of OXT compared to placebo prior to MBGT in patients with SSD will result in a greater reduction in NS. The research design is based on an experimental, triple-blind, randomized, placebo-controlled trial. The manualized MBGT sessions are led by two psychotherapists over four weeks. Four sessions take place once a week in a group of six patients. The effects of OXT peak after 30–80 minutes for optimal reinforcement of social behavior. Patients receive 24 I.U. of OXT or placebo intranasally 30 minutes prior to each therapy session. Plasma OXT levels will be determined by radioimmunoassay. To exclude gender bias, both women and men will join mixed-sex groups controlled for hormones. Change in NS as the primary endpoint will be measured with validated interviews (Positive and Negative Syndrome Scale, PANSS) and psychometric questionnaires (Self-Evaluation of Negative Symptoms, SNS). Variables, including plasma OXT levels, will be measured at baseline and post-intervention and the role of genetic variations of the OXTR genes for the NS will be looked at exploratively.

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- › ICU Acquired Weakness
- › Skeletal Muscle

The Role of Circadian Rhythm Disruption in Critical Illness Myopathy

Circadian rhythms adjust behavior and physiological processes to the 24-hour(h)-light-dark cycle according to the Earth's rotation. Circadian rhythms can be disrupted in septic intensive care unit (ICU) patients. In skeletal muscle, clock genes control several downstream processes, involved in the regulation of muscle mass, function, and glucose metabolism. Pathomechanisms in these downstream processes, have been identified by our research group in Critical Illness Myopathy (CIM). CIM remains an unresolved clinical problem that places a high burden on individuals and the health care system. For the first time, my hypothesis-generating project investigates whether circadian disruption contributes to CIM. In a first reverse translational approach, we will investigate circadian patterns, based on high-frequency 24-h vital-sign and glucose data in ICU patients with CIM compared to ICU patients without CIM. From bedside to bench, we will compare circadian rhythms in gene expression and metabolism in the skeletal muscle in the mouse

model of sepsis-induced muscle atrophy and in controls. Circadian disruption may be a key mechanism for the described CIM pathophysiology downstream of the circadian feedback loop. The identification of novel pathways is of great importance for the development of preventive strategies, which are currently unavailable. The future perspective of the project will be the translation by individualized circadian interventions, such as overnight fasting and dynamic light therapy as innovative systems approaches to prevent CIM and improve outcomes. Circadian medicine is an emerging new field with high interdisciplinary relevance because circadian rhythms influence almost all physiological functions, and are essential for health.

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- › Psychophysiological Measurement
- › Addictive disorders

Craving Assessment in Patients with Alcohol Use Disorder Using Virtual Reality Exposure

Virtual Reality (VR)- based therapies as a major component of digital mental health applications have received increasing scientific attention for diagnosis and treatment of alcohol use disorders (AUD). AUD cause a substantial burden of disease with worldwide 3 million deaths per year and high relapse rates. Alcohol craving is a major predictor for relapse and a main diagnostic criterion. Craving is associated with psychological and physiological responses and can be induced by presenting alcohol cues (»cue-exposure«). Cue-exposure therapy (CET) is an effective strategy in Cognitive Behavioral Therapy (CBT) to reduce craving, but due to high organizational, timely and financial efforts it has not yet been established in clinical routine. Our research group aims to develop an innovative Virtual Reality-based cue exposure therapy for AUD. This study will examine if the therapy software induces a transient change in craving levels and if this is measurable via subjective and physiological parameters. Data on severity of AUD and craving, comor-

bidities, demographics, side effects and the feeling of presence in VR will be additionally collected. Patients will use a 3D VR headset to immerse themselves into three different situations (neutral vs. target situations) while heart rate, heart rate variability, pupillometry and electrodermal activity will be measured continuously. This project contributes to the development and careful adjustment of the software and is the first step to clinically validate an innovative VR tool that we plan to use for an effective VR treatment for AUD.

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- › Big Data
- › Machine Learning

Development of an Automated Nephro Candidate Score (N-CS) for Chronic Kidney Disease of Unknown Etiology

Chronic kidney disease (CKD) is an increasingly prevalent global health issue. While high blood pressure and diabetes mellitus are the leading causes, a significant number of patients suffer from monogenic diseases. Despite advances in next-generation sequencing, the underlying genetic cause remains unknown in many cases, as some patients lack pathogenic variants in known disease-associated genes. Expanding the search to all genes not previously linked to CKD yields an overwhelming number of potentially disease-causing variants. However, due to limited resources, it is impossible to investigate each one in depth. To pinpoint the most promising candidates, researchers rely on various databases (e.g., population databases, RNA sequencing databases, animal models). Yet, this process is time-consuming, non-scalable, and highly dependent on individual expertise. My project aims to address these challenges by developing the Nephro Candidate Score (N-CS) – a tool designed to systematically, objectively, and efficiently prioritize genetic

variants in a standardized manner. By integrating diverse data sources and incorporating machine learning, the N-CS mitigates subjective biases in variant evaluation and uncovers patterns that may go unnoticed by human analysis. To maximize accessibility and collaboration, the N-CS will be available as both a web tool and a command-line tool, facilitating standardized research efforts across multiple institutions. Standardization is especially crucial for rare diseases, where collective data sharing can drive significant breakthroughs. Ultimately, this project seeks to improve treatment and genetic counseling for CKD patients by identifying novel candidate genes, paving the way for better diagnostics and targeted therapies.

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- › Digital Medicine
- › Neuroimaging
- › Software Development

Advancing Automated Deep Brain Stimulation Programming by Encompassing Stimulation-Induced Side-Effects

Deep Brain Stimulation (DBS) has demonstrated superior efficacy compared to best medical treatment in advanced Parkinson's Disease (PD) patients and those with early motor complications. To maximize the therapeutic potential of DBS, precise customization of stimulation parameters is crucial, as these parameters need to be tailored according to individual patient anatomy, electrode placement, and symptom profile. Currently, this programming process follows a laborious and iterative trial-and-error approach, involving multiple in- and outpatient visits over several months or even years. Highly skilled medical personnel are required to adjust stimulation parameters based on observed therapeutic effects and potential stimulation-induced side-effects. This protracted process places a considerable burden on both patients and health-care providers.

To address these challenges, I have developed and validated StimFit, an open-source software that leverages a data-driven algorithm to suggest optimal stimulation parameters based on individual electrode location. In a recent clinical trial involving 35 PD patients, StimFit demonstrated non-inferiority compared to the standard-

of-care settings derived from traditional clinical optimization. This marked the first time a data-driven model was applied to suggest optimal stimulation parameters in a randomized, double-blind trial, providing strong evidence that data-driven algorithms could assist future programming strategies. This may not only save time and resources but might pave the way for more complex neural interfaces that would be impossible to program using conventional clinical trial-and-error procedures.

Distinct technical opportunities to further improve StimFit predictions and ultimately increase the therapeutic benefit of the treatment have emerged from our previous work. Therefore, the central aim of my current project is to further improve the therapeutic benefit of the software by establishing a fine-grained model to predict stimulation-induced side-effects, which can occur due to inadvertent co-stimulation of adjacent neural tracts. Within the scope of the JDCSP I am developing and validating a data-driven model to predict stimulation-induced side-effects based on neuroimaging data. This model will then be integrated into the StimFit software to improve the therapeutic benefit and safety of DBS settings suggested by the software.

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- › Vascular Neurology
- › Neuroinfectious Diseases

A Camera-Based Remote Sensing System for Stroke Detection

In Germany, every year about 90.000 patients undergo cardio-thoracic surgery. In these patients, a major perioperative complication is ischemic stroke, with an incidence of ~2% on average but up to 5% in certain types of procedures. Large vessel occlusion-related acute ischemic stroke (LVO-AIS) is particularly devastating but can be effectively treated by early mechanical thrombectomy if recognized immediately. In the postoperative setting, however, rapid recognition of relevant neurological deficits is complicated by reduced vigilance of the patient due to prolonged weaning from sedation. Thus, detection of perioperative LVO-AIS is regularly delayed, leading to poor neurologic outcome and often lifelong disability or death.

We, thus, aim to develop a novel, automated, bedside camera-based clinical surveillance system to detect typical LVO-AIS-related motion deficits (MD), leveraging machine learning-based motion analysis. This interdis-

ciplinary project, situated at the intersection of vascular neurology, cardiology, cardiothoracic surgery, and computational medicine, is conducted in collaboration with the German Heart Center Berlin (DHZC) and the Institute for Machine Learning at the Eidgenössische Technische Hochschule Zürich (ETH Zürich).

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- › Image Analysis
- › Biophysics
- › Cancer Research

Development of a Microscopic Magnetic Resonance Elastography for Non-Invasive Characterization of Gliomas Since

Multifrequency Magnetic Resonance Elastography (MRE) is an MRI-based technique for the non-invasive, quantitative assessment of tissue viscoelasticity, generating elastograms that map mechanical properties across tissue slices. While MRE is clinically established for evaluating chronic liver disease, its application holds significant potential in cancer diagnosis and therapy, being the only non-invasive method for in vivo assessment of viscoelastic tissue properties in intracranial neoplasms. These properties are known to influence the tumor micro-environment, invasive growth, metastasis, and therapy resistance. However, the application of MRE in neuro-oncology remains limited due to constraints in spatial resolution.

This project aims to extend the clinical utility of MRE to the brain, particularly for glioma assessment, by enabling high-resolution imaging. Gliomas exhibit considerable inter- and intratumoral heterogeneity and grow in an infiltrative manner, necessitating high-resolution viscoelastic mapping to capture relevant biophysical variations and to better understand oncogenesis. To achieve this, the project incorporates advanced post-processing

techniques, specifically Principal Component Analysis-based denoising, to enhance the signal-to-noise ratio and enable high-resolution elastogram acquisition without increasing scan time. Simultaneously, the project focuses on optimizing MRI sequence parameters to ensure an effective balance between image quality and acquisition efficiency. Additionally, a novel inversion algorithm, incorporating physics-informed deep learning models and tailored for high-resolution brain MRE, is being developed.

For broader clinical application, optimizing spatial resolution and image quality without prolonging acquisition times is crucial. By combining imaging physics, computational image analysis, and neuro-oncological insight, this interdisciplinary project seeks to establish high-resolution MRE as a powerful tool for non-invasive, in vivo characterization of gliomas.

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- › Multi-Omics
- › Lymphoma Biology
- › Machine Learning
- › Novel Study Design

DFG Deutsche
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Anticipation, Prediction and Optimization of the Therapeutic Outcome of Lymphoma Patients Using Digital Model Analysis

Diffuse large B-cell lymphoma (DLBCL) is the most common non-Hodgkin's lymphoma disease worldwide. The standard therapy R-CHOP has a high cure rate in the first-line setting, but relapsed cases have poor survival despite extensive treatment options. To date, there are no clinically relevant risk group biomarkers that can predict therapy efficacy or determine improving therapy additions. Previous studies used static data to try to make predictions and do not take into account changes in tumor tissue that are triggered under therapeutic pressure.

My research is dedicated to the dynamic change of lymphoma disease under therapy in order to enable a better, individualized therapy selection and control in the future. My interdisciplinary project in close cooperation with Prof. Dr. Roland Eils at the BIH Center for Digital Health aims to develop new prediction parameters through a unique and innovative multimodal evaluation approach of combining clinical data, basic tumor biology research and current bioinformatics analysis methods.

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- › Food Allergy
- › Food Provocation

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Genome Wide Association Study to Identify Patients at Risk of Severe Anaphylaxis Using Single Nucleotide Polymorphisms

Anaphylaxis is a potentially life-threatening type I hypersensitivity reaction, with an acute onset, that requires immediate therapy with adrenaline. In order to identify patients with a higher risk of severe anaphylaxis (and therefore provide personalized treatment recommendations), reliable biomarkers are sought for.

Genome-wide association studies (GWAS) are increasingly used to identify associations between single nucleotide polymorphisms (SNPs) and clinical phenotypes. With the costs of full genome sequencing decreasing and abundance of databases containing whole-genome sequencing data, the technical tools for GWAS are becoming increasingly more accessible. There is an unmet need to identify patients at higher risk of developing severe anaphylaxis in order to provide personalized care recommendations and reduce unnecessary healthcare costs. Based on our recent publication, identifying differences in miRNA profiles of patients prone to anaphylaxis, it is

likely that GWAS will reveal differences in SNPs associated with risk of severe reactions.

The project will contribute to our understanding of anaphylaxis processes and may help reveal underlying biological processes that could contribute to anaphylaxis pathomechanisms. Ultimately, if the resulting models could explain who might be predisposed to severe anaphylaxis, this project has a real potential for changing current clinical guidelines on the management of anaphylaxis, and therefore has deep consequences in optimizing personalized medicine approach and reducing healthcare costs.

The project will be developed according to open science and reproducible science principles. The analysis writing process will be version-controlled, and the data will be shared publicly according to Open Data principles and DFG Guidelines on the Handling of Research Data.

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- › Pediatrics
- › Cholestatic Liver Diseases
- › Newborn Screening

DiaperID – Digital Stool Colour Screening For Early Detection of Cholestatic Liver Diseases in Newborns

With DiaperID we develop a digital screening tool for neonatal cholestatic liver diseases. The key for timely diagnosis and treatment of these diseases lies in the detection of pale or discoloured stools. Our approach is an image recognition algorithm based on artificial intelligence that can differentiate between conspicuous and normal stools based on their colour. To make this screening test for rare diseases widely applicable, we are developing a smartphone app that parents can use to analyse their children's stool colour themselves. The results of utilisation and user experience of the first application studies will help us on our road to make DiaperID available to all parents independent of their socio-economic status.

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- › MRI-Directed Biopsy Pathway
- › Risk-Adapted Screening

STIFTUNG CHARITÉ

Shaping Risk-Adapted Prostate Cancer Screening Using Liquid and AI-Based Imaging Biomarkers

Prostate cancer (PCa) is the second most common cancer in men worldwide, with incidence expected to double by 2040. While multiparametric MRI has become guideline-approved for primary PCa diagnosis via the »MRI pathway«, its effectiveness remains limited by inadequate pre-MRI risk stratification, highlighting an unmet clinical need.

My previous research focused on developing explainable artificial intelligence (xAI) models for interactive AI-assisted decision-making in liver and prostate cancer diagnosis. Using a retrospective sample of 1,224 men, we developed a VGG-Net-based convolutional neural network to detect and classify PCa lesions at MRI, achieving high accuracy in internal and external test samples. The system provided visual and textual explanations using PI-RADS features with 80% accuracy, confirmed by expert readers. Additionally, that zone-specific PSA density calculation doubled specificity and reduced false positives by 25% while maintaining 93% sensitivity for clinically significant PCa. In prospective studies, saliva-derived polygenic risk scores (PRS) correlated with MRI findings while PSA values did not, indicating potential to reduce unnecessary MRI examinations.

My project aims to develop a holistic approach for early PCa diagnosis, incorporating radiological, clinical, and genetic data from German multi-center patient cohorts. We will train a deep learning system using the German National Cohort to identify imaging, clinical, and genetic biomarkers for improved patient risk stratification through hierarchical Bayesian Networks. Also, we will build a comprehensive xAI tool for PCa detection combining liquid and imaging biomarkers in the prospective multicenter PROSTAIT study. This study will validate and refine xAI-assisted prostate MRI algorithms to enable risk-adapted informed decision-making using PRS.

This project addresses the European Union Council's call for risk-adapted PCa screening strategies. The multi-modal prediction model using nationwide big data analysis will enable personalized patient monitoring, while the PROSTAIT study represents the first German investigation combining PRS with AI-supported MRI interpretation. By integrating population-specific risk stratifiers with explainable AI tools, this research will pave the way for risk-adapted patient management in PCa diagnosis.

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- › Multiomics Analysis
- › Geriatric Anesthesiology

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Multomics Analysis of Postoperative Neurocognitive Disorders in Older Patients

Postoperative delirium (POD) and postoperative neurocognitive disorder (NCD) are common and severe complications after surgery and are associated with increased morbidity, mortality and loss of autonomy. Both POD and NCD can be regarded as complex diseases, as their development is multifactorial, and only hypotheses are currently available regarding their etiology. It is believed, that no single hypothesis can adequately explain the causal relationships of POD and NCD, and that only pathway interactions can describe the complex phenomena. A systematic approach combining genomic, transcriptomic, proteomic and environmental data using pathway analyses in a patient population has not yet been described. Therefore, the aim of this project is to describe biological pathways involved in the development of POD and NCD using multi-omics analysis in a hypothesis-generating approach.

This project is part of the multicenter prospective observational study BioCog – »Biomarker Development for Postoperative Cognitive Impairment in the Elderly« (ClinicalTrials.gov ID: NCT02265263). 1032 patients ≥65 years of age undergoing elective surgery were included. Primary endpoints are the occurrence of POD and NCD. Blood samples were obtained from patients preoperatively, on the first postoperative day, and three months after surgery.

Genomic, transcriptomic, as well as miRNA profiling data were generated using microarray analysis. In addition, proteomic data on selected parameters are available. These data will be analyzed under consideration of the clinical database in a multi-omics approach.

A particular benefit of a multi-omics approach is the possibility of integral (longitudinal) analysis, since data beyond the gene level can also be considered. Another crucial advantage of this project is that omics data from the patient collective of interest are available, that regulatory elements can be taken into account by means of miRNA profiling, and that the clinical database of the study provides comprehensive information on environmental factors. In addition, repeated sampling enables the consideration of temporal factors related to primary endpoints. All DNA, RNA and plasma samples were stored in a biobank, so that further investigations (e.g. methylation patterns, de novo sequencing) are possible.

Finally, biological pathways of POD and NCD are to be established. These should provide new hypotheses for follow-up studies on the prevention and treatment of POD and NCD.

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- › Immunology
- › Single-cell genomics



Mapping Single-Cell Dynamics in the Regression and Recurrence of Liver Fibrosis in Metabolic Dysfunction-Associated Steatohepatitis

Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterised by the excess accumulation of liver lipids and the presence of cardiometabolic risk factors, most notably obesity and insulin resistance. MASLD can progress to steatohepatitis (MASH) and fibrosis, the latter being the main determinant of patient outcomes, including liver cirrhosis, liver failure and hepatocellular carcinoma. It is well established that liver fibrosis can regress upon cessation of the causative hepatotoxic injury. Furthermore, affected individuals often go through cycles of weight loss and weight regain that may affect the severity and progression of disease. However, the mechanisms of fibrosis regression and the impact of recurrent body weight changes in MASLD and MASH are only incompletely understood.

We aim to close this gap in our current understanding by applying single-cell Multiome sequencing and spatial transcriptomics to mouse models of liver fibrosis regres-

sion and MASH recurrence as well as in patient biopsies. This will enable us to provide an in-depth characterisation of the cellular landscape and the mechanisms underlying the regression and recurrence of MASH fibrosis and to describe the specific functional states of involved cell populations. A better characterisation of these mechanisms will help to identify novel therapeutic targets for treating liver fibrosis, and biomarkers for predicting and monitoring treatment responses.

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- › Image-Guided Radiotherapy
- › Data-Driven RT Workflows

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Development of a Novel Digital Class Solution for Data-Driven Adaptive Radiation Therapy

Traditional radiotherapy (RT) plans are static and do not adapt to daily patient-specific anatomical changes, variations in tumour volume or treatment-related toxicity. In contrast, Adaptive Radiation Therapy (ART) allows real-time or off-line adjustments to treatment plans based on changes in patient anatomy, tumour dynamics or response as captured by functional imaging. Despite its transformative potential, the use of ART in clinical practice is currently limited or non-existent. This is partly due to the lack of advanced software tools for monitoring changes in tumours or organs at risk, as well as for tracking patient toxicity, and the absence of standardised protocols and effective workflows. In this study, we aim to develop a digital class solution to integrate multiple data sets, such as anatomical and functional imaging, patient feedback, toxicity and tumour response, into a novel ART workflow to facilitate knowledge generation for real-time data-driven RT. This prospective proof-of-concept study will include three ART solutions: 1) Online

ART: Real-time, on-couch treatment adaptation for small cell lung cancer and esophageal cancer patients, integrating daily CBCT, electronic patient-reported outcome measures, and electronic physician-reported toxicity data; 2) Offline ART: Weekly diffusion-weighted MRI-guided response-adaptive RT for patients with head and neck cancer, targeting resistant tumour regions; 3) One-stop shop: Planning and delivering RT in a single session for palliative treatment of bone metastases, streamlining workflow by eliminating multiple sessions. As a result, this digital class solution can overcome current limitations in ART delivery by developing a comprehensive, digital, data-driven workflow. The long-term goal of this project is to provide a digital platform for adaptive strategies based on predictive models of response and toxicity. Successful implementation of this technology could significantly advance radiotherapy.

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- › Digital Health
- › Psychocardiology
- › Health Related Quality of Life



Digital Collaborative Care – Expanding Access to Effective Treatments

Coronary artery disease (CAD) is one of the most common causes for mortality. About 30% of CAD patients also suffer from depression.

For these patients, Conventional Collaborative Care (CCC) is a highly effective improving cardiovascular risk factors (CVRf), medication adherence, depression scores, and health-related quality of life (HRQoL): Patients present to nurses every 2-3 weeks for three major tasks: (1) Symptom monitoring, (2) Supervision of self-care goals, i.e., better medication adherence, cessation of smoking, more exercise, healthy diet, and (3) Offering support to achieve these goals. The effects of these interventions accumulate, as CVRF and depression share several therapeutic interventions, e.g., physical exercise. However, CCC is hardly available, labor-intensive, and regional proximity is required. In the absence of CCC, we usually refer these patients to outpatient psychotherapy which is just as well short in supply. Therefore, in order to give patients access to treatment, we believe CCC needs to be translated into the digital environment. To do so, we propose the combination of two existing tools: Just-in-Time Adaptive Interventions (JITAI) and Patient-Reported Outcome Measures (PROM).

JITAI could improve cardiovascular risk factors such as physical activity, smoking, diabetes management, diet, and mental health. Effectiveness in depression is still inconclusive and requires further research and development, which we propose in this project by linking PROM to JITAI. PROM measurement has shown to significantly reduce mortality, emergency ward visits, hospital admissions, and to improve patients' HRQoL in cancer patients.

We will use routine PROM assessments as ecological momentary assessment (EMA) on which a JITAI is dependent. Additionally, we plan to assess CVRF, e.g., smoking, exercise, and diet. So, for example, if PROM anxiety scores are high, a relaxation technique can be offered. Or, when exercise is low, a walk route can be proposed. This integrated assessment will give physicians additional signals: for example, low exercise may be explained by high PROM pain interference scores.

The goal is to develop a digital JITAI based that improves patients' depression and CVRF. We will evaluate the JITAI in a pilot randomized-controlled trial. To detect medium effect sizes (Cohen's $d=0,55$) with a power of 80% and $\alpha=0,05$, we need $N=104$ participants ($N=52$ per group).

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- › Hyperinflammatory Syndromes

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Telemedicine for Quality Improvement in Diagnosis and Therapy of Hemophagocytic Lymphohistiocytosis

Hemophagocytic Lymphohistiocytosis (HLH) is a rare though life-threatening hyperinflammatory syndrome in which uncontrolled immune activation leads to excessive cytokine release – the so-called cytokine storm. Due to its clinical overlap with sepsis, HLH remains frequently undetected. Mortality rates are high, particularly among critically ill patients (58%). In a previous systematic review, we could demonstrate that a multitude of therapeutic strategies are applied in HLH patients while current recommendations remain inadequately implemented. Here, telemedicine offers a unique opportunity for research while at the same time improving diagnosis and therapy of HLH. Within this project, we will establish a reference center for national and international consultation requests. In that we concentrate digitally all HLH cases in one center, we will generate a sufficient number of cases to perform robust data analysis. Diagnosis and therapy recommendations are based on current guidelines. For therapy evaluation, daily telemedical visits are carried out from the day of the first consultation. Serious and refractory cases are assessed interdisciplinary by a Charité expert panel.

Moreover, there is an unmet need of targeted therapies. One reason lies in the poorly investigated pathophysiology of HLH. Therefore, we will establish and expand a biobank of cytokine profiles to enable safe diagnosis and causal treatment.

Collecting data of a rare disease prospectively will allow us to create a machine learning algorithm which will ultimately support deep disease characterization. The database will be used to develop an app for physicians as a diagnostic and treatment tool. Simultaneously, data will be transmitted via the app for scientific evaluation.

Our aim is to reduce in hospital mortality through rigorous adherence to current guidelines. Moreover, we will assess treatment efficacy and develop an HLH severity score to improve classification of HLH patients. This project is the first international study in HLH patients using telemedicine.

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- › Rare diseases
- › Digital health
- › Remote patient monitoring
- › Digital biomarkers

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Digital Monitoring for Predicting the Course of Rare Diseases

Patients with rare diseases require highly individualised therapy by specialists, which is often only available to a limited extent. Myasthenia gravis, a chronic autoimmune disease leading to exercise-induced muscle weakness, is in many ways a model disease for rare diseases:

Fluctuations in the course of the disease can lead to severe crisis-like deteriorations if timely intervention is not provided, including adjustment of immunosuppressive therapy.

Through the development of MyaLink, the digital platform for monitoring vital signs and PROMs (patient reported outcome measures) of patients with myasthenia, real-world data can be collected, enabling new insights into disease progression and therapy response. The platform enables communication between doctor and patient and the sharing of data, which can concretely improve care. These effects are to be measured in studies within the

framework of the digital clinician scientist programme. In addition, machine learning algorithms will be developed that can detect potential deteriorations. The implementation of such digital biomarkers in MyaLink can then bring direct benefits to patient care by supporting doctors in decision-making and automatically identifying patients at risk of crisis.

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- › Single Cell Genomics

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Longitudinal Dissection of Glioblastoma Evolution at the Single Cell Level

Glioblastoma is the most common malignant CNS-intrinsic tumor and presents a leading cause of cancer-related death. To date, primary untreated tumors have been well molecularly characterized at the bulk and single cell level, yet glioblastomas inevitably recur despite standard therapy consisting of resection, irradiation, and alkylating chemotherapy, and the cellular and molecular processes underlying their evolution and therapy resistance remain incompletely understood. Previous studies have pointed towards a potential role of non-genetic mechanisms in driving glioblastoma evolution, which encompass adaptive changes in tumor cell states as well as non-malignant microenvironmental factors, that together cooperate to promote tumor recurrence, but cannot be adequately resolved at the bulk level.

In this collaborative project between multiple departments at the Charité, the BIH, and the MDC-BIMSB, we propose to systematically analyze the evolutionary land-

scape of tumor and microenvironmental cell states in paired clinical samples of human glioblastoma, profiled by single nucleus RNA-sequencing (snRNA-seq) at diagnosis and recurrence following standard therapy. We expect that this work will enable an improved pathophysiological understanding by uncovering the non-genetic dynamics and heterogeneity that underlie glioblastoma evolution, and, via multimodal integration with clinical, molecular, and histological data, guide the improvement of diagnostic and therapeutic avenues.

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- › Clinical Decision Support Systems

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Intelligent Antimicrobial Therapy Reviewing and Optimizing System (IATROS) – Development and Validation of an Intelligent Antibiotic Cockpit for Nosocomial Infections in Visceral Surgery

Nosocomial pneumonia and urinary tract infections are among the most common hospital-acquired infections in visceral surgery patients with incidence rates reaching up to 20% and 4%, respectively. Inaccurate use of antimicrobial therapy increases patient morbidity and contributes to local and cumulative global antibiotic resistance which is already considered a leading medical challenge of the 21st century by the World Health Organization. The goal of the Intelligent Antimicrobial Therapy Reviewing and Optimizing System (IATROS) is to develop and validate an automatic, intelligent solution for guideline-based real-time review and monitoring (S3 and S2k guidelines) of antibiotic therapies after surgical major resections using nosocomial pneumonia and urinary tract infections as proof of concept. This will include real-time support for diagnostic and therapeutic follow-up steps, active and intelligent integration of local resistance patterns, automatic consideration of patient-specific factors in drug selection, and the ability to network simultaneously given antibiotics on the same ward. The hypothesis

whether an intelligent antibiotic support system enables a significant quantitative and qualitative improvement in the implementation of antibiotic therapies in accordance with S3 and S2k guidelines will be tested. The primary endpoint is defined as the selection of the administered antibiotic in accordance with the respective guideline recommendation. Secondary endpoints include indication- and patient-specific dosing, timely de-escalation, total duration/guideline-compliant discontinuation of antibiotic therapy, and implementation of additional diagnostic measures as recommended by the guidelines. The technical project implementation includes the generation of a digital patient profile incorporating the real-time treatment course in terms of a digital deep patient representation. In parallel, the decision tree will be digitally implemented in accordance with the S3 and S2k guidelines for pulmonary and urogenital infections. Finally, a prospective, non-interventional, diagnostic accuracy study will be conducted for validation.

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- › Neuroprognostication
- › Deep Learning
- › Generative Artificial Intelligence

Multimodal Explainable Artificial Intelligence for Outcome Prediction of Hypoxic Ischemic Encephalopathy after Cardiac Arrest

Hypoxic-ischemic encephalopathy (HIE) is a major cause of disability and mortality following out-of-hospital cardiac arrest. Early and accurate prognostication is essential for guiding therapeutic decisions and communicating realistic outcomes to families. Early detection and reliable outcome prediction are critical to guiding treatment decisions and informing families, yet current diagnostic methods rely on complex, multidisciplinary evaluations that are often unavailable in non-specialized centers.

While non-contrast brain computed tomography (CT) is widely available, radiological signs of HIE are subtle and subject to significant inter-reader variability. Other recommended modalities, such as electroencephalography (EEG), auditory evoked potentials (AEP), and somatosensory evoked potentials (SEP), are not consistently available across non-specialized hospitals. Laboratory biomarkers like neuron-specific enolase (NSE) and S100 calcium-binding protein B (S100B) show promise but are prone to false positives or negatives due to confounding comorbidities.

To overcome these limitations, our project aims to develop a multimodal artificial intelligence (AI) model that integrates imaging, electrophysiological data, and laboratory biomarkers into a unified, explainable framework for outcome prediction in HIE. By leveraging recent advances in transformer-based architectures, we seek to create a robust decision-support tool that performs reliably across diverse clinical environments.

This work represents one of the first attempts to apply transformer-based models to neuroprognostication after cardiac arrest, using routine brain CT in combination with laboratory and electrophysiological data. Our goal is to develop a reliable and widely accessible tool for predicting outcomes in HIE – one that clinicians can trust to support informed, evidence-based decisions.

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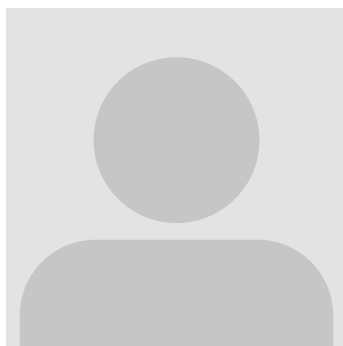
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- › Schizophrenia
- › Mental Illness
- › Digital Markers

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Natural Language Processing of Digital Communication: the Linguistic Markers of Schizo-Bipolar Disorders

Despite over a century of research, schizophrenia remains as a severe mental illness characterized by a serious prognosis and significant personal and societal burdens. Currently there are no concrete (bio)markers available to detect or predict psychotic episodes. Additionally, patients demonstrate fluctuating alterations in language production that correlate with the illness's dynamics. Notably, the widespread use of computer-mediated communication in recent years has led to the accumulation of vast amounts of linguistic data from patients with schizophrenia. Recent advancements in Artificial Intelligence and Natural Language Processing (NLP) provide the technology needed to analyze such data and extract relevant linguistic features from them. In this project, I will collaborate across disciplines, including researchers from Psychiatry, Linguistics, and Computer Science, as well as individuals with lived experience of schizophrenia and their caregivers, to compile a retrospective, longitudinal dataset of linguistic data from authentic digital

communication of patients with schizophrenia and neurotypical controls. In the schizophrenia group, I will annotate the language data based on the participants' medical histories, thus ensuring clinical ground truth. My aim is to identify linguistic features for relapse detection and prevention using methods of Computational Linguistics and NLP.

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DFG Deutsche
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Developing a Standardized scRNA-Seq Analytical Framework to Enable Automated and Unbiased Leukemia and Immune Cell Monitoring

Despite therapeutic improvements for high-risk hematologic malignancies such as acute myeloid leukemia (AML), relapse remains the main cause for disease-related mortality. Long-term disease control can be achieved through immunological approaches that include allogeneic stem cell transplantation (HSCT) and post-transplant modulation of graft-versus-leukemia effects (Penter & Wu, JCI 2020). However, they are most successful when applied to low disease burden and are often rendered ineffective through immune escape mechanisms such as T cell exhaustion (Penter et al., Blood 2023). Early detection of post-HSCT relapse and monitoring of immune escape mechanisms could enable pre-emptive and specific immunotherapy for post-transplant AML relapse.

The availability of single cell RNA sequencing (scRNA-seq) is providing unprecedented insight into cellular hierarchies and states. Intriguingly, beside transcriptional profiles, scRNA-seq can provide genetic information, including simultaneous detection of single nucleotide variants and copy number changes (CNV), annotation of donor/recipient origin, and immunogenetic information

like viral transcripts and T or B cell receptor sequences (TCR, BCR) (Penter et al., Frontiers in Immunology 2022; Penter et al., Nature Communications 2024). Therefore, scRNA-seq appears as an ideal platform for integrated leukemia and immune monitoring that could guide therapeutic decisions.

However, a major obstacle for this concept is a lack of standardization of scRNA-seq analysis. Currently, most scRNA-seq analyses are custom solutions for small datasets with a focus on novelty, thus leading to time-consuming, highly operator-dependent, and often unreproducible results. In addition, large-scale integrative analyses of scRNA-seq datasets provide opportunities for cross-study comparisons that can reveal higher level principles. More streamlined analytical approaches are thus urgently needed that will substantially improve insights gained from scRNA-seq and lay the foundation to develop scRNA-seq as a diagnostic tool. To address this need, I propose to develop a standardized analysis pipeline for blood and bone marrow-derived scRNA-seq profiles to enable streamlined and reproducible leukemia and immune cell monitoring.

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STIFTUNG CHARITÉ

Deep Learning Approach for the Detection and Differentiation of Active Inflammatory Changes on Spinal MRI

The project »Deep Learning Approach for the Detection and Differentiation of Active Inflammatory Changes on Spinal Magnetic Resonance Imaging« aims to develop an advanced artificial intelligence (AI) tool for the automated analysis of spinal MRI scans, focusing on the detection, quantification, and differentiation of inflammatory changes indicative of axial spondyloarthritis (axSpA). While AI applications in medical imaging have advanced significantly, current models primarily assess sacroiliac joints, with limited research on spinal inflammatory changes, crucial for early diagnosis, disease monitoring, and treatment decision-making in axSpA.

This study will develop a deep learning (DL) model based on a U-Net architecture, specifically designed to detect, quantify, and distinguish inflammatory changes from degenerative spinal conditions with accuracy comparable to human experts. Given the complexity of spinal anatomy and the challenges associated with MRI interpretation – such as motion artifacts, variations in imaging protocols, and the subtle nature of inflammatory changes – the model will incorporate robust preprocessing techniques, including denoising, artifact reduction, and intensity normalization, to enhance performance.

The research will leverage extensive, diverse datasets from multiple well-characterized cohorts, including GESPIC, OptiRef, AXIS, and NAKO, ensuring generalizability across different populations and imaging conditions. A rigorous train-retrain approach will be employed, allowing iterative refinement of the model based on expert-labeled MRI images. This strategy will help identify systematic errors, improve classification accuracy, and adapt the model to a wide range of spinal pathologies. The primary outcome is the model's diagnostic performance, measured by receiver operating characteristic (ROC) curves and area under the curve (AUC) values. Secondary outcomes include sensitivity, specificity, accuracy, inter-rater agreement, and comparison of model performance with expert readers.

Potential clinical applications range from clinical diagnostics and decision support to standardized, reproducible imaging assessments in clinical trials. The project is committed to open science principles. Future implementation in research and clinical settings could enhance the efficiency, accuracy, and accessibility of spinal MRI analysis, ultimately leading to improved axSpA diagnosis, disease management, and patient outcomes.

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- › Medical Data Science
- › Predictive Modelling
- › Machine Learning
- › Natural Language Processing
- › Cardiovascular Medicine

Early Prediction of Hemodynamic Instability in Cardiac Surgery ICU Patients

Hemodynamic instability (HI) is frequently observed in postoperative cardiac surgery intensive care unit (CSICU) patients. Etiologies of HI include life-threatening conditions such as cardiac tamponade, low cardiac output syndrome, hemorrhagic shock and severe vasoplegia. Early prediction of HI could streamline diagnostic and therapeutic interventions (e.g., bedside imaging, advanced hemodynamic monitoring, mechanical circulatory support) in high-risk patients, potentially improving survival. According to the existing literature, there is no definition of HI in CSICU patients and previously used criteria to define HI, such as the definition of septic shock, are not specific for our cohort due to the high prevalence of postoperative hypotension and hyperlactatemia. Therefore, a cohort-specific definition of HI is particularly needed. In a first study, a cohort-specific definition of HI will be found through a data-driven and outcome-oriented approach using an optimization algorithm and ubiquitously available biomarkers (vitals, labs,

etc.). This approach provides an alternative to traditional expert opinion-based approaches, such as the Delphi method, which are often used to establish definitions and risk classifications in medicine.

In a second study, supervised machine learning will be used to predict HI according to the previously established definition using large internal and external electronic health record datasets. In the long term, we aim to prospectively validate the clinical utility of the HI prediction model and implement the prediction model in clinical practice to improve patient care.

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- › Navigated Transcranial Magnetic Stimulation
- › Brain Tumor Surgery

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Towards an Automated Risk Assessment in Brain Tumor Surgery Using AI-Based Methods: a Pilot Study

Recently, regression tree analysis using data from navigated transcranial magnetic stimulation (nTMS) and diffusion tensor imaging (DTI) has been established to predict postoperative motor outcome in brain tumor surgery. However, clinical application requires human resources, technical requirements and expertise.

The aim of this project was to establish an automated pipeline using advanced methods as well as deep learning-based models, providing automated risk assessment for the motor outcome. The automated pipeline is structured as follows: 1) preprocessing of MRI data (skull stripping, co-registration) using Freesurfer, nibabel, ANTs and FSL, 2) automated tumor segmentation using deep learning-based models (Vnet), 3) automated motor cortex segmentation using FastSurfer, 4) preprocessing of DTI data including denoising, gibbs-ringing removal and bias correction using MRtrix3, 5) automated tractography of the CST using TractSeg and MRtrix3 and 6.) automated

measuring of the tumor-tract-distance (TTD) and estimating motor cortex infiltration.

Automated risk stratification in brain tumor surgery is a use case for standardizing clinical patient care, improving treatment quality, and economizing medical resources. Through the pipeline, more patients will benefit from differentiated preoperative planning that has been shown to lead to better functional and oncologic outcomes, not least by assigning patients to the right treatment pathway (based on their individual risk profile).

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- › Telemedicine
- › Remote Patient Monitoring
- › Congenital Heart Disease
- › Pediatric Heart Transplantation
- › Heart Failure in Children

Digital Care Hub for In-Home Surveillance of Infants with Single Ventricle Heart Disease

One percent of all children are born with a congenital heart defect. Most severe are single-ventricle heart defects (SVHD) such as hypoplastic left heart syndrome (HLHS). Children born with SVHD have been experiencing substantial improvements from compassionate care to long-term survival due to the development of an innovative three-stage surgical palliation. After the first surgery there are physiological challenges that put children particularly at risk for life-threatening events. High mortality rates during this period have led to the implementation of in-home surveillance strategies for early detection of changes that may lead to hemodynamic decompensation. This project aims to conduct a clinical trial with application-based remote patient monitoring of infants with SVHD. Our aim is to avoid life threatening events during in-home surveillance, to improve somatic growth and to optimize heart failure therapy. We will furthermore investigate the adoption of technology and the impact on adherence by the caregivers. Additionally,

we aim to investigate a potential reduction of the psychological burden for caregivers and the long-term health-related quality of life of the children reported as patient-reported outcome measures (PROMs).

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- › Digital Medicine
- › Image Based Modeling
- › Artificial Intelligence/Machine Learning
- › Cardiology

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Forschungsgemeinschaft

Precision Therapy Planning – Clinical Application of Image Based in Silico Modeling

Valvular heart diseases are one of the most common causes of heart failure that can affect both children and adults. Accurate treatment planning is crucial but can be very challenging especially in complex valve defects such as combined aortic stenosis (AS) and mitral valve regurgitation (MR). To date, clinical guidelines are based on rather indirect parameters such as valve orifice area, heart size or pressure gradients to determine if, and when, an intervention is recommended. However, they do not provide any support as to which procedure will have the best functional effect in each patient. Decision support systems that help the physician not only with the optimal timing but also with deciding which type of intervention provides the best results are therefore of high clinical relevance.

The aim of the project is to develop a decision support system that combines AI- and mechanistic models, allowing physicians to interactively simulate treatment

strategies and their hemodynamic outcomes for heart valve diseases, including complex cases such as combined AS/MR.

In order to train the AI-based algorithms we will use data from real as well as synthetic patient cohorts. While synthetic data allows to accelerate clinical AI development and application, they also have the potential to include pathophysiological conditions that are rare and therefore often underrepresented in real patient cohorts. With the use of synthetic data, we aim to increase the prediction accuracy of the AI-based algorithms and to facilitate the clinical translation of image-based in silico modeling for a better precision therapy in cardiovascular medicine.

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- › Applied Deep Learning/Machine Learning

Early Prediction of Clinical Deterioration Using Ambient Intelligence in the Pediatric Cardiac Intensive Care Unit

The postoperative intensive care management of patients with congenital heart defects is complex and newborns and infants are particularly vulnerable to postoperative complications. Early detection of clinical deterioration is crucial for reducing morbidity and mortality by timely initiation of treatment. Development of a model for real-time prediction of critical events before they occur could help clinicians to evaluate information and augment decision-making processes. Ultimately, this could lead to a reduction in complications and greater patient safety. The objective of the study is a real-time analysis of movement and perfusion patterns using a high-resolution multispectral camera (visual and infrared spectrum). In addition, routinely recorded parameters and the high-frequency curve data of the vital sign monitor are analyzed with the help of machine learning pattern recognition. The aim is to use the prospectively collected data to develop a model for the early detection of clinical deterioration after cardiac surgery in newborns and infants,

which enables a real-time prediction of the individual patient's risk for cardiorespiratory failure and neurological complications. We plan the research, development, validation and exploratory and predictive modeling on a prospectively collected data set.

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- › Cardiology
- › Intensive Care Medicine
- › Artificial Intelligence
- › Quality and Outcomes

Development and Validation of a Deep Neural Network to Aid Discharge Processes for Patients in the Cardiac Arrest Center

The demand for cardiac intensive care unit (ICU) beds remains substantial to effectively monitor and treat patients with severe cardiovascular diseases, such as acute coronary syndrome, cardiac arrest or fulminant myocarditis. Efficient patient management strategies, including timely discharge policies, are critical in addressing the increasing need for ICU beds. However, ICU transfer to normal wards is a high-risk event, often associated with increased hospital stays, costs and patient mortality, as evidenced by our studies at Massachusetts General Hospital and the International SNAP-2 Trial across 248 hospitals.

The extensive volume of clinical, physiological, laboratory, and monitoring data in ICUs poses challenges for critical care providers in making well-informed decisions on discharge-readiness under constrained capacity. Current machine learning algorithms for ICU discharge-readiness are limited to surgical or general medical populations and have not been validated in the specialized environment of cardiac arrest centers within the German healthcare system. These existing models do not adequately address the complexities of critically ill cardiovascular patients.

In response, we developed a Feedforward Neural Network (FNN) algorithm during the Junior Digital Clinician Scientist Program at the Berlin Institute of Health, analyzing 2,498 adult patients from three cardiac ICUs at Deutsches Herzzentrum der Charité – Universitätsmedizin Berlin. This model, based on 127 ICU variables, demonstrated superior predictive performance for unplanned ICU readmissions within 72 hours after discharge, achieving 97.9% sensitivity, 86.9% specificity, 99.4% precision and 97.4% accuracy. The FNN outperformed conventional logistic regression and the so called »SWIFT score«, with an AUC of 90.8 compared to 79.51 and 71.1, respectively.

To make this algorithm clinically practical, we propose developing an intuitive »ICU traffic light« application to assist intensivists in discharge decision-making. We aim to externally validate the FNN algorithm in four additional cardiac ICUs, followed by a bi-centric randomized controlled trial to compare clinician-based versus AI-supported discharge strategies. Successful implementation and validation could lead to the commercialisation of the first AI-based algorithm tailored for cardiac arrest centers, enhancing discharge decision support for critically ill cardiac patients.

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- › Neuromuscular Scoliosis
- › Spinal Muscular Atrophy
- › Gene-Therapy
- › Data-Driven Artificial Intelligence

Optimizing Surgical Timing: Development of a Data-Driven Disease Progression Model for Scoliosis in 5q-SMA Patients Under Novel (Gene) Therapies

Spinal muscular atrophy (SMA) is a rare neuromuscular disorder causing progressive muscle weakness and scoliosis in up to 90% of patients. With the advent of gene and antisense oligonucleotide therapies, SMA patients now reach unprecedented motor milestones, necessitating updated treatment strategies. Traditional scoliosis management relied on data from untreated patients, which is now insufficient. Given the high complication rates of spinal surgery in SMA (25-75%), precise surgical timing is critical. Early observations suggest an increasing incidence of early-onset scoliosis in this new patient cohort.

Building on our previous work in feature engineering for rare disease datasets and scoliosis prediction modeling, this project aims to develop a data-driven disease progression model to anticipate scoliosis evolution under novel SMA therapies. By integrating clinical, imaging, and functional data, our model will support decision-making

for optimal surgical intervention. Despite AI advancements, rare diseases present challenges due to limited datasets, necessitating expert-driven data curation to ensure clinical applicability and interpretability. Our goal is to provide an accessible, reliable tool to improve scoliosis management in SMA, enhancing patient outcomes in the era of gene therapy.

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- › Predictive Modeling and Machine Learning
- › Health Data Analysis

DFG Deutsche
Forschungsgemeinschaft

Development of a Score for the Automated Identification of Patients at Risk of CPSP

Chronic pain poses a significant global health challenge, contributing substantially to disability worldwide. Chronic postsurgical pain (CPSP), with its identifiable onset, is particularly suitable for preventive measures. However, existing predictive models for CPSP are limited, often specific to certain surgeries, and resource-intensive. This project aims to develop an innovative, automated risk assessment score for CPSP using advanced machine learning algorithms. The goal is to create a continuously optimized tool that integrates into clinical systems, providing real-time alerts to physicians about high-risk patients and enabling timely preventive interventions.

The project will systematically review potential CPSP risk factors and test these factors using historical patient data. This retrospective analysis will inform the development of the risk assessment algorithm, which will then be trained and validated under real-world conditions.

By focusing on patients likely to develop CPSP, the project aims to refine the predictive accuracy and practical application of the algorithm.

This project represents a significant innovation in chronic pain management by offering a data-integrated approach to identifying and preventing CPSP. By enabling timely interventions for high-risk patients, the project aims to improve patient outcomes and optimize healthcare resource utilization. The collaboration with leading institutions and the use of advanced digital methodologies underscore the project's potential for widespread clinical adoption.

Success will be measured by improved patient outcomes, increased satisfaction, and more efficient use of healthcare resources, setting a precedent for future initiatives in pain management and chronic disease care at Charité and beyond.

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- › Computational Medicine
- › Multiomics

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Identification of Stroke Biomarkers Based on -Omics Datasets of the BeLOVE Study

The Berlin Long-Term Observation of Vascular Events (BeLOVE) is a large prospective longitudinal observational cohort study that is rooted inside the BIH Infrastructure. BeLOVE is aiming to improve prediction and disease-overarching mechanistic understanding of cardiovascular disease progression and outcomes by comprehensively investigating a high-risk patient population with different organ manifestations. It combines the full spectrum of deep molecular phenotyping and deep clinical phenotyping to monitor patients with acute cardiovascular events (including stroke). These cardiovascular events are the leading cause of death and disability worldwide. Patients with a history of a cardiovascular events or multiple risk factors are at very high risk for future major cardiovascular events (MACEs) or death, but substantial uncertainties remain in risk estimation and in our understanding of disease progression. This is particularly true for stroke, a highly heterogeneous disease with different etiologies ranging from cardioembolism and large artery atherosclerosis to small vessel disease.

This diversity translates into a highly variable individual disease progression with wide-ranging risk of poor functional outcomes and of secondary MACEs.

The integration of proteomics and metabolomics data together with clinical data will help to identify a stroke population at high risk of poor functional outcome and secondary cardiovascular events. I propose the implementation of a generalizable computational workflow to investigate the O-link proteomics dataset and the metabolomics dataset from the BeLOVE study to identify novel biomarkers that predict long term outcomes of stroke patients. The ultimate goal is to establish a well-documented »gold standard« workflow that integrates pre-processed Olink Proteomics data and Metabolomics data with clinical outcomes. This will not only facilitate biomarker studies for other subpopulations of the BeLOVE study, but also future -omics based biomarker studies at BIH.

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- › Extrachromosomal DNA (ecDNA)
- › Amplicons

Max-Eder-Nachwuchsgruppe

Essential Regulatory Elements of High-Level Amplifications in Breast and Nonsmall Cell Lung Cancer

DNA amplifications are the most common gain-of-function alterations in cancer genomes. They promote cancer through overexpression of oncogenes and therefore include regulatory elements that promote their transcription. A systematic exploration of how amplifications exploit transcriptional regulation is lacking. We hypothesize that most high-level amplifications exploit tissue-specific enhancers and transcription factors (TFs) to promote oncogene expression and thereby cancer growth. Understanding if tissue type specific regulatory factors are essential for cancer growth could lead to therapies that target tissue-specific transcription. To understand the role of and mechanisms behind tissue-specific transcription in cancer, we need insights into three points: 1) The enhancer loci that are recurrently incorporated into amplifications and their lineage or oncogene specificity. 2) Necessary and sufficient TFs to activate oncogene transcription from the amplicon. 3) The heterogeneity of these mechanisms across cells in a tumor to understand how changes in differentiation state alter transcriptional regulation from amplifications. I therefore propose three specific aims: Aim 1. Test the

hypothesis that enhancer loci with binding sites for tissue- or oncogene-specific TFs are recurrently incorporated into high-level amplifications. This will nominate enhancers necessary to drive expression from the amplifications depending on lineage and oncogene. Aim 2. Test the hypothesis that expression of oncogenes in high-level amplicons requires tissue- or oncogene-specific TFs. These experiments will reveal vulnerabilities in breast and lung cancer resulting from the regulation of their high-level amplicons. Aim 3. Test the hypothesis that transcriptional regulation of high-level amplicons varies across cells in a tumor with different differentiation states. These data will assess the feasibility of targeting the essential regulators of oncogene amplifications therapeutically and could allow predicting likely resistance mechanisms. Overall, we expect this project to reveal cancer type specific mechanisms of oncogene activation. These could prove to be vulnerabilities specific to the cancer cells and therefore attractive therapeutic targets.

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- › Cognitive Neuroscience
- › Psychopharmacology

Emmy Noether Nachwuchsgruppe

The Neurocognitive Mechanisms of Serotonergic Antidepressants

Pharmacotherapy of depression targets monoaminergic neurotransmission, with selective serotonin reuptake inhibitors (SSRIs) constituting first-line antidepressant intervention.

Serotonergic antidepressants work well for many patients, however, it typically takes multiple weeks before they experience clinical benefit. Clinically, this delayed onset of action is problematic, as it prolongs patients' suffering and complicates therapeutic decision-making. Scientifically, it is puzzling, as SSRIs boost brain serotonin levels within hours of treatment initiation.

To date, it remains unknown why neurochemical and therapeutic effects operate on different timescales, with SSRIs impacting neurocognitive function within hours, and clinical benefits only emerging after several weeks. Further, psychiatrists lack early markers that predict who may show a delayed clinical response to antidepressant intervention. Arguably, this arises from a paucity of knowledge about how antidepressants work, as, even 60 years after their serendipitous discovery, the exact neurocognitive mechanisms of their action remain elusive.

Thus, the aim of my research is to refine depression treatment by providing a neuroscientifically grounded, mechanistic understanding of antidepressant drug action. In my prior work, I used a combination of cognitive experiments, computational modelling, and pharmacological neuromodulation to assess how SSRIs impact reward processing, learning, and decision-making in healthy individuals. Further, I developed a neurocomputational framework of how these neurocognitive changes can lead to gradual mood improvement over time.

In this BIH Clinician Scientist fellowship, I aim to probe this mechanistic account of antidepressant drug action in a bench-to-bedside translational approach in depressed patients initiating antidepressant treatment. Here, the goal of my fellowship is two-fold: First, I will determine how a serotonergic impact on neurocognitive processes in patients gives rise to mood improvement and a delayed clinical onset of SSRI therapy. Second, I will identify early neurocognitive markers, occurring within hours or days, that may serve to predict who will show a delayed clinical response after weeks of serotonergic intervention.

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- › Autoimmunity
- › Neurology
- › B Cells

Emmy Noether Nachwuchsgruppe

From Deep Phenotyping to Complete Remission in Autoimmune Encephalitis

Autoimmune encephalitis is an immune-mediated condition that induces inflammation of the brain and a variety of neurological and psychiatric manifestations. This proposal focuses on three pivotal objectives: achieving a profound understanding of the pathophysiology of autoimmune encephalitis caused by autoantibodies and related disorders, improving the treatment options, and leveraging the capabilities of deep phenotyping combined with deep learning for predictive modelling of patient outcomes. Central to the therapeutic advancements proposed is the CAAR T cell approach, a novel therapeutic strategy capable of selectively targeting antigen-specific B cells. Concurrently, the proposal emphasizes the potential of advanced machine learning techniques in refining predictive models for both disease progression and treatment efficacy. By intertwining wet-lab and dry-lab techniques, the proposal aims to bridge the gap between current knowledge and the pressing challenges in the field. The ultimate vision is a transformative shift in how autoimmune encephalitis and related disorders are understood, treated, and predicted.

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The Immune Modulation Effect of Locoregional Therapies and Its Potential Synergy with Immunotherapy

Recent successes in the field of immuno-oncology have generated considerable interest in the investigation of approaches that combine standard of care treatments with immunotherapies. Locoregional therapies (LT) represent attractive candidates for this approach given the potential for immune system stimulation through the large-scale release of tumor-associated antigens (TAA) following LT-induced cell death. In fact, LT-induced necrosis of tumor cells leads to massive release of tumoral neoantigens, facilitating recruitment and activation of dendritic cells into the microenvironment. This effect can be leveraged to transform an immunosuppressive microenvironment that is not conducive to checkpoint inhibitor therapy into an immunosupportive setting, in which systemic therapies might be more effective.

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Department of Cardiology, Angiology
and Intensive Care Medicine

Director

Prof. Dr. med. Ulf Landmesser

Fields of Research

- › Cardiovascular Disease
- › Cardiomyopathy
- › Myocarditis

Identification of Biomarkers in Inflammatory Cardiomyopathy and Potential Novel Therapeutic Targets

The spectrum of inflammatory cardiomyopathies (iCMP) encompasses diverse etiologies. It is increasingly recognized that myocardial inflammation may influence heart function and outcomes even in »non-classic« iCMP such as amyloidosis.

Understanding of the pathophysiology remains limited, which is reflected in a lack of targeted therapies. Steroids remain mainstay treatment in most acute cases, yet they fall short in addressing the etiological specificity for effective treatment. Patients may progress to severe heart failure.

Also, early detection is critical; myocarditis and sarcoidosis continue to be diagnosed late – sometimes only at autopsy. My team is dedicated to address both challenges: 1) We develop diagnostic and prognostic markers for iCMP to enable timely intervention. 2) We decipher mechanisms of iCMP to pave the way for targeted therapies. My team identified novel targets and collaborates with industry to expedite translation of our findings into clinical practice.

PD Dr. med. Petra Hühnchen

**In Program from – to**

07.2021–07.2027

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Clinic

Charité – Universitätsmedizin Berlin
Department of Neurology with
Experimental Neurology

Director

Prof. Dr. med. Matthias Endres

Fields of Research

- › Neuroimmunology
- › Cell Death
- › Neuroprotection

Neurology & Oncology – Neurological Sequelae of Tumor (Therapy)

Neurological sequelae are among the most common side effects of many tumor therapies. They can affect the peripheral nervous system resulting in a mono- or polyneuropathy (e.g. chemotherapy-induced polyneuropathy, radiation-induced neuropathy) or lead to diffuse changes in cognitive functions (e.g. post-chemotherapy cognitive impairment, radiation-induced leukoencephalopathy). In addition, immune-related adverse events (irAE) are becoming more common due to the increased use of tumor immunotherapies such as immune checkpoint inhibitors or cell-based therapies like CAR-T cell therapy. In general, the underlying pathomechanisms of neurological sequelae after tumor therapy are poorly understood and often no or insufficient treatment options exist. At the same time, they further reduce patients' quality of life, lead to potentially long-term disability and frequently cause treatment limitations, which directly affect patients' chance of survival. Our research group investigates the molecular mechanisms by which

tumor therapies damage the peripheral and central nervous system. We use (personalized) induced pluripotent stem cell (iPSC)-based cell and organoid models as well as animal models. Preclinical findings are validated in clinical cohorts, in which we also investigate predictive and disease biomarkers. After identification of target candidates, we conduct interventional trials with a focus on prevention of tumor therapy-associated neurological sequelae (e.g. PREPARE study). Lastly, our group is involved in the design and evaluation of novel infrastructures and interdisciplinary approaches to enhance patient care of cancer patients and cancer survivors.

PD Dr. med. Felix Krenzien

**In Program from – to**

10.2021–10.2027

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Clinic

Charité – Universitätsmedizin Berlin
Department of Surgery
Center for Musculoskeletal Surgery

Director

Prof. Dr. med. Johann Pratschke

Fields of Research

- › Surgical oncology
- › Hepatobiliary surgery
- › Liver cancer

Improving Outcomes in Hepatobiliary Surgery

Felix Krenzien is principle investigator and highly qualified surgical oncologist, currently working full time at Charité. He is actively engaged in both basic and clinical research, being funded by the Else-Kröner Fresenius Foundation and the Innovation Fund of the Federal Joint Committee. His primary focus is on liver cancer, specifically on the development of early detection methods and the identification of key factors for progression to fibrosis and cirrhosis, which are significant risk factors. In recognition of his exceptional accomplishments in this field, he was honored with the prestigious Ferdinand Sauerbruch Research Prize and was inducted into the esteemed Academy of Excellence of the German Society of General and Visceral Surgery by the full professors.

PD Dr. med. Lena Oevermann

**In Program from – to**

06.2025–07.2027

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Clinic

Charité – Universitätsmedizin Berlin
Department for Pediatric Oncology and
Hematology

Director

Prof. Dr. med. Hedwig Deubzer

Fields of Research

- › Hemoglobinopathies
- › Gene Therapy
- › Stem Cell Transplantation

Hemoglobinopathies – Optimizing Current and Future Therapies

I have been head of the outpatient clinic for haemoglobinopathies since 2017. The outpatient clinic is one of the largest in Germany and is growing continuously due to migration movements and the introduction of newborn screening in October 2021. I currently look after around 270 children. During my time as a clinician scientist, I was able to set up a biodatabase for pediatric stem cell transplantation, the data, samples and results of which are now available to us for further projects. Further clinical research questions relate to endothelial dysfunction in haemoglobinopathy patients and its impact on outcome after stem cell transplantation and the optimization of red blood cell supply in haemoglobinopathy patients. With the approval of Casgevy as the first CRISPR Cas-based gene therapy for haemoglobinopathies, my field is now taking an exciting step towards an additional treatment option. We are establishing a scientific support program in parallel to the clinical application. Jonathan Groß will be working on the topic as part of his Clinician Scientist Fellowship and, thanks to the networking within the German Center for Child and Adolescent Health (DZKJ), we can embed the program across all sites. The main focus here is on the assessment of haematopoiesis in patients with haemoglobinopathies before and after cell or gene therapy intervention. Beyond clinical

research, the aspect of global health research and the optimization of care for haemoglobinopathy patients is very important to me. As part of a cooperation project (Else Kröner-Fresenius-Stiftung funding) with Dr. Maria Vittoria De Vita, Nairobi, Kenya, we are working on improving care structures in countries with limited medical resources. The medium-term goal is to set up a national register and newborn screening for sickle cell disease in Kenya and, as a next step, to establish a clinical trial platform for the safe introduction of innovative treatment options. Nationally and internationally, I am closely linked to the relevant experts and networks and play a key role in the development of new therapy guidelines, for example. As I consider the education and dissemination of information about the diseases to be an essential building block for improving care, I am involved in two different expert committees for doctor and patient training. The collaboration with the Stem Cell Transplantation division also offers a broad platform for innovative, clinical and translational research approaches.

Prof. Dr. med. Tobias Penzkofer



In Program from – to

09.2021–08.2027

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Clinic

Charité – Universitätsmedizin Berlin
Department of Radiology (including
Pediatric Radiology)

Director

Prof. Dr. med. Bernhard Gebauer (acting)

Fields of Research

- › Quantitative Imaging Biomarkers for Oncology
- › Explainable AI
- › Research Networks

Quantitative Imaging / Prostate Imaging, Abdominal / Urogenital Radiology

The scientific focus of my laboratory (Quantitative Imaging Lab, www.qilab.org) is on the development, validation and implementation of quantitative imaging biomarkers, validation of structured reporting tools and explanatory machine learning based applications for oncology imaging. In these areas, external funding has been successfully obtained and the results obtained have been published in high-impact publications. Understanding the value of image-based diagnostic tools such as structured diagnostic criteria or automated quantitative analyses including AI-based biomarkers is a complex and challenging task. Not only the technology used, but also high-quality data sets – accurately representing the population under study – are critical and seminal components for exploring the diagnostic and prognostic value of radiologic imaging. Because data quality management is of central importance, a key element of this work is the development of highly specific tools for organizing and annotating the data used. We have incorporated two principles into our KI-driven biomarker development: (a) rigorous end-to-end validation of findings against radiologist performance and (b) application of explainable artificial intelligence to identify and correct biased models for decision making. Projects planned for the next few years specifically address the implementa-

tion of the results obtained to date: (a) work on PI-RADS classification (analysis of PI-RADS classifications, evaluation of new PIRADS versions), which will be incorporated into the classification via broad evaluation (participation in multicenter studies, contribution to ESUR/ACR Prostate Working Group) (b) prototypical implementation and paraclinical validation of the Explainable AI model we developed for prostate MRI diagnostics, focus on end-to-end validation with analysis of the clinical value of the method by translating the models for image-based biomarkers. Further development, expansion and participation in national and international network projects in the field of imaging research is another stated goal. In doing so, we will build on our previous experience, especially in setting up the Germany-wide Radiological Cooperative Network (RACOON) for COVID-19 research, but also the oncological imaging initiatives at national (National Center for Tumor Diseases, NCT, German Network for Personalized Medicine, DNPM) and international level (CHAIMELEON – EU-wide structured repository for health imaging data as an open source for artificial intelligence (AI) experimentation in cancer management; EUCAIM – European Federation for CANcer IMages).

Prof. Dr. med. Nathanael Raschzok

**In Program from – to**

10.2021–09.2027

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ClinicCharité – Universitätsmedizin Berlin
Department of Surgery**Director**

Prof. Dr. med. Johann Pratschke

Fields of Research

- › Liver Transplantation
- › Organ Reconditioning
- › Colorectal Liver Metastases

Regenerative Concepts in Transplantation

The success of liver transplantation is limited by the number of available liver grafts, and a relevant number of liver grafts is declined due to quality concerns. The aim of our work is to increase the number of liver grafts for transplantation by applying of concepts from regenerative medicine. We are using ex vivo liver machine perfusion to assess the quality of marginal liver grafts, which are compromised e.g. by steatosis hepatis or by elevated age of the donor, and we are developing strategies for reconditioning of these grafts. We have developed a rodent model of normothermic liver machine perfusion which we are using for dose-response studies, and we are using a rat liver transplant model to assess liver grafts after reconditioning. Moreover, we are developing clinical protocols for quality assessment of marginal or declined liver grafts by machine perfusion to expand the donor pool for transplantation.

Prof. Dr. med. Jan Scheitz



In Program from – to

07.2021–07.2027

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Clinic

Charité – Universitätsmedizin Berlin
Department of Neurology with
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Director

Prof. Dr. med. Matthias Endres

Fields of Research

- › Post-Stroke Cardiovascular Complications
- › Brain-Heart Interaction
- › Cardiac Imaging After Stroke

Brain-Heart Interaction

Jan F. Scheitz is consultant stroke neurologist and Professor of Clinical Stroke Research at the Department of Neurology Charité in Berlin, Germany and at the Center for Stroke Research Berlin (CSB). Dr. Scheitz is head of the research group »Integrative Cardio-Neurology« (#brain-heartlab). Together with his group, his major research interests include all aspects of Heart & Brain interaction, poststroke (cardiac) complications, mechanisms and prognostic impact of cardiac troponin elevation after stroke, and use of cardiovascular MRI in acute stroke. His major motivation is to improve clinical awareness of poststroke cardiac complications, and to promote interactive collaborations between stroke neurologists and cardiologists. He is Fellow of the European Stroke Organisation (FESO), founding member of the »Heart&Brain Task Force« of the World Stroke Organisation (WSO), and member of the ESO Brain & Heart Council.

PD Dr. med. Stephanie Thee, PhD

**In Program from – to**

01.2023–01.2028

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Clinic

Charité – Universitätsmedizin Berlin
Department of Pediatric Respiratory
Medicine, Immunology and Critical Care
Medicine

Director

Prof. Dr. med. Marcus A. Mall

Fields of Research

- › Chronic Lung Disease
- › Infection and Diagnostic in Lung Disease
- › Telemedicine

Optimizing Care for Patients with Chronic Lung Disease

In my current role as consultant in the Clinic for Pediatrics m.S. Pneumology, Immunology and Intensive Care Medicine, my clinical focus lies on the outpatient and inpatient care of patients with acute and chronic lung diseases. In this context, our Christiane Herzog Cystic Fibrosis Center with about 400 patients and our outpatient clinic for Primary Ciliary Dyskinesia (PCD) with 100 patients as well as the immunological-infectious outpatient clinic are among the largest centers in Germany. My clinical work allows me a close connection with my scientific focus, which is on pulmonary infections in patients with and without chronic lung disease. In recent years, I have not only contributed to an improved, evidence-based therapy of tuberculosis in children through extensive work on the pharmacokinetics and toxicity of antituberculotics, but also characterized the current management of children with different forms of tuberculosis in low-incidence countries through international collaborations. This highlighted the particular challenge of pathogen diagnosis in patients who cannot produce sputum. In collaboration with the Research Center Borstel (National Reference Center *ycobacteria*) we have initiated a study to evaluate the application of an innovative diagnostic method, the so-called »face-mask-

sampling«. Based on this, I plan to evaluate this non-invasive diagnostic procedure in other lung diseases such as cystic fibrosis (CF) or PCD. In a cooperation with the Max Delbrück Center for Molecular Medicine (MDC) it is further planned to investigate the lung and gut microbiome of patients with chronic lung diseases and to associate it with clinical data. Building on our cohorts, expansion to international cohorts is planned with the addition of registry data. In addition to diagnostics, the adherence of patients with chronic lung diseases to therapy plays a decisive role in the course of the disease and the development of resistance. As part of the multicenter »conneCT CF study«, I am currently evaluating an innovative form of care using coaching and telemonitoring in patients with cystic fibrosis with the aim of improving adherence and thus reducing progressive pulmonary deterioration. The structures and knowledge gained from the conneCT study will be used to evaluate similar forms of care for other chronic lung diseases.

Dr. med. Felix Zirngibl



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06.2025–07.2027

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Clinic

Charité – Universitätsmedizin Berlin
Department for Pediatric Oncology and
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Director

Prof. Dr. med. Hedwig Deubzer

Fields of Research

- › Oncologic Immunotherapies
- › Allogeneic Stem Cell Transplantation
- › Immune Reconstitution

Pediatric Allogeneic Stem Cell Transplantation

My clinical work focusses on allogeneic stem cell transplantation (SCT) in children and adolescents with malignant and non-malignant diseases. Particularly of interest is the management of acute complications such as infections or graft-versus-host disease (GVHD), as well as the close follow-up care of transplanted patients in order to prevent the occurrence of relapse of malignant diseases or long-term side effects.

I have several academic specialisations. As Principal Investigator, I am involved in several phase I/II studies in the field of paediatric SCT. My scientific projects cover the following areas of pediatric SCT: I. The optimisation of GVHD prophylaxis, in particular through the use of Everolimus and its influence on immune reconstitution after SCT. A prospective clinical study in collaboration with PD Dr Oevermann. II. As a post-SCT intervention in high-risk patients with acute myeloid leukaemia, the combination therapy of decitabine and venetoclax is being investigated in a translational approach. III. In parallel, I am working on establishing a database for pediatric SCT patients at the Charité, which is intended to combine the improvement of the quality of care with scientific questions.

Furthermore, I am continuing the work from my Junior Clinician Scientist fellowship and benefiting from the close network I established there in the translational research context. In collaboration with Trion Research (Munich), a bispecific trifunctional antibody for the treatment of patients with neuroblastoma is being investigated and prepared for clinical use.

My research concept pursues a translational approach with a direct link between clinical questions and pre-clinical research. The aim is to establish a dedicated research group ›Clinical Research in Pediatric SCT‹, which systematically develops and clinically tests innovative therapeutic approaches. Topics include immune reconstitution and immune monitoring, complication management and post-SCT interventions. A particular focus is on patient-oriented research in which, for example, the results of immunological analyses are directly incorporated into clinical management.

Alumni of the BIH Charité Clinician Scientist Program

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Junior Digital Clinician Scientists

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Excellence Track

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Advanced Clinician Scientists

Deubzer, Hedwig
 Haghikia, Arash
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Clinician Scientist Board Members (incl. Deputies)

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Tumor Immunology and BIH

Prof. Dr. med. Britta Siegmund (ex officio)

Deputy Director of the BIH Charité Clinician Scientist
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Prof. Dr. med. Christopher Baum (ex officio)

Chairman of the Board of Directors of Berlin Institute
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