Tissue and Motion Conference 2022

Djurönäset

November 9-10th

#TnMConference

For the digital version of the Tissue and Motion conference program, scan this QR code

Contact us: tissueandmotion@clintec.ki.se

Welcome to the Tissue and Motion Conference 2022

Who we are in the KI Research Network Tissue and Motion

Tissue and Motion is a research network supported by Karolinska Institutet, and which connects researchers and clinicians with a common interest in the multidisciplinary fields of regenerative medicine, motion and health. The Tissue and Motion network aims to represent a well-balanced combination of research areas, departments, and basic and clinical researchers.

The aim of Tissue and Motion

The aim of Tissue and Motion is to facilitate and stimulate new alliances, research constellations and interdisciplinary-collaborations, and to integrate clinical & pre-clinical research, especially for junior researchers.

Join Tissue and Motion!

Are you doing research within the area of Tissue and Motion? Do you want to contribute to networking and translating research at Karolinska Institutet? Tissue and Motion arranges and supports scientific meetings, symposia and workshops, networking and out-reach activities. Let us know if there is an activity that you would like to arrange!

Sincerely,
Cecilia Götherström
Chair of Tissue and Motion

Tissue and Motion Conference

November 9-10th 2022

GENERAL PROGRAM OUTLINE

Wednesday, November 9th:

Bus from **07:15**

Arrival, coffee and registration **08:30**

Welcome and introduction **08:45**

Session Part 1: Tissue **09:00**

Group photo **12:40**

Lunch **12:45**

Session Part 2: Motion **13:45**

Fika **15:30**

Career Panel Discussion **17:00**

Hotel Check-in **18:00**

Dinner **20:00**

Thursday, November 10th:

Breakfast and hotel check-out **07:45**

Oral session **09:00**

Poster session **10:30**

Lunch **12:45**

Awards and closing **13:45**

Bus departure **15:00**

Arrival Stockholm **16:15**

1-7 Konferenslokaler & hotellrum / Conference & Hotel rooms
8 Seregården
9 Reception, Restaurang Matsalen, Barer / Reception, Restaurant & Bars
10 Skärgårdsspa / Spa
11 Spapaviljongen / Spa treatments
12 Skärgårdskrogen Sjöboden / Restaurant Sjöboden

13 Vedeldad bastu & badtunnor / Wood burning sauna & hot tubs
14 Svit & Längan / Suite & Hotel rooms

A Varm infinitypool / Hot outdoor infinity pool
B Cyklar / Bicycles
C Naturstig / Nature trail
D Badstrand & Äventyrscenter / Beach

E Helikopterplatta / Helipad
F Motionsslinga / Running trail
G Utegymstationer / Outdoor gym stations
H Busshållplats / Bus stop
I Tennisbana / Tennis court
J Folkparken / Outdoor event area
K Ångbåtsbrygga / Steamboat jetty

L Kanoter & båtar / Canoes & boats
M Gästhamn / Guest harbour
N Mötesplats / Meeting spot

P1 Parkering / Parking
P2 Parkering / Parking

Practical Information

TRANSPORT & RETURN

Bus departs from Cityterminalen on Wednesday, November 9th, at 7:15am (the extended terminal building at the Stockholm main railway station), entrance next to World Trade Center, Klarabergviadukten. Please check the departure screen information with "**KI BUS TO DJURÖNÄSET"** (Tissue and Motion Conference 2022) sign. The bus ride takes approximately one hour. We return to the Stockholm City terminal from Djurönäset on Thursday, November 10th at 15:00 departure.

ARRIVAL AT DJURONASET

The registration open at 8:30 am at reception. Please find us at the hotel reception (you can find the reception building in the map attached). There's luggage storage space available near the reception.

ACCOMODATION

The rooms are reserved at Djurönäset for those who have pre-booked through the registration - single room.

MEALS

Lunch and dinner will be provided at the restaurant near the reception. Lunch would be a buffet style and dinner would be a formal sitting dinner.

POSTERS

The poster room is close to the main conference room in House 4. Please mount the poster before evening of 9th November (pins will be provided). More information will be provided on site.

INTERNET

Wi-Fi is available in the venue and your room. Please ask for the password at the reception.

LEISURE

You are welcome to enjoy the swimming pool and sauna as they are included in your stay. However, note that room service, extra drink, spa, any purchases, etc are not included. Payment must be settled in conjunction with the order and remember to bring your credit/debit card to the restaurant/bar/spa/shop upon personal purchase.

Day 1 (November 9th):

08:30 **Coffee / Check-in**

08:45 **Welcome speech** (Cecilia Götherström)

Part 1: Tissue (Chair: Fredrik Lanner)

09:00 **Keynote lecture 1:** *"Human heart and cardiomyocyte development during early fetal life"*, Christer Sylvén

10:00 *"Cell replacement therapy for diabetes"*, Siqin Wu

10:30 **Fika**

11:00 *"Role of Laminin α4 in leukemia progression and drug resistance"*, Huan Cai

11:25 **Keynote lecture 2:** *"Rebuilding the heart with human ventricular progenitors"*, Kenneth Chien

12:25 Introduction to KI innovations, Patrik Blomquist

12:40 **Group Photo**

12:45 **Lunch**

Part 2: Motion (Chair: Sara Windahl)

13:45 *"Qualitative and quantitative movement analyses in the clinic and in research"*, Wim Grooten

14:10 *"Recovery from radiation-induced damage to growing bones involves functional compensation by growth plate chondrocytes"*, Phillip Newton

14:35 *"Bone forming effects of mechanical loading in mice"*, Sara Windahl

15:00 *"On Brains and vessels, how perivascular fibroblasts contribute to ALS neurodegeneration"*, Sebastian Lewandowski

15:30 **Fika**

16:00 **Keynote lecture 3**: *"WNT signaling system regulates cortical and trabecular bone differently"*, Ulf Lerner

17:00 **Panel discussion: Career options – Academia & Industry**
(Chair: Hong Qian - Academia, Lilian Walther Jallow - Industry)

Industry panelists: Bramasta Nugraha, Roger Chang, Mochammad Fadjar Wibowo, Anna Vidina

Academia panelists: Evren Alici, Elisabet Åkesson

18:00 **Hotel check-in, mingle with fika and sauna**

20:00- **Dinner**

Sauna

Day 2 (November 10th):

07:45 **Breakfast + Check out**

09:00 **Oral presentations**

10:30 **Poster presentations** + **Fika** on the side

12:45 **Lunch**

13:45 **Awards + finishing talks**

15:00 **Departure**

Oral Presentation

Thursday, November 10th 2022 (09:00), Conference Hall

Time	Presenter	Title
09.00 – 09.08	Suchita Desai	Mechanical loading prevents Arthritis-induced bone loss in male mice
09.10 – 09.18	Jose Marchan-Alvarez	Media composition regulates chondrogenic potential and extracellular vesicles production in chondrocytes
09.20 – 09.28	Ferdinand von Walden	Multi-transcriptome analysis following an acute skeletal muscle growth stimulus in mice yields tools for discerning global and MYC regulatory networks
09.30 – 09.38	Ya-Wen Fu	Dynamics and competition of CRISPR–Cas9 ribonucleoproteins and AAV donor-mediated NHEJ, MMEJ and HDR editing
09.40 – 09.48	Linnéa Bielicz	Cardiorespiratory response to acute incremental exercise in individuals with cerebral palsy
09.50 – 09.58	Laia Sadeghi	Histone demethylase KDM6B modulates NF- κB signaling pathway in Mantle Cell Lymphoma cells to promote their adhesion to stromal cells

Poster Presentation

Thursday, November 10th 2022 (10:30), House 4

Presenter(s)	Poster No.	Category	Title
Eva Wärdell, Agneta Månsson Broberg	001	Tissue	Cardiomyocytes derived from hi-PSCs as a model for studies of cardiotoxicity
David Brenière-Letuffe	002	Tissue	Driving photoreceptor differentiation in hPSCs through Activin A or COCO induction
Laura Baque Vidal	003	Tissue	Molecular profiling of stem cell-derived retinal pigment epithelial cell differentiation established for clinical translation
Mathilde Bergamelli	004	Tissue	Extracellular vesicles arising from human embryonic stem cells: what impact on blastoid phenotype and endometrial implantation rate?
Emine Begum Gencer Oncul	005	Tissue	Gene expression profiling of Fetal Mesenchymal Stem Cells during osteogenic differentiation
Yuk Kit Lor	006	Tissue	Transcriptomic differences between Fetal and Adult Mesenchymal Stem Cells
Jari van Diermen	007	Tissue	Genetic deconvolution of regenerative phenotypes in Acomys cahirinus
Bhavna Rani	008	Tissue	10x Cite-Seq As A Tool To Decipher The Role of Integrins in Ex-Vivo Expansion of Hematopoietic Stem Cells (HSCS) to Treat Hematopoietic Disorders
Suria Jahan	009	Tissue	Investigation of changes in platelets function and membrane receptor sin patients with chronic myelomonocytic leukemia (CMML)
Estela Santos-Alves	010	Motion	Reduced mitochondrial health is linked to muscle weakness in mice with arthritis
Julia Starck	011	Motion	Muscle activation during flywheel training in children
Jennifer Geara	012	Motion	Elucidating the role of mitochondria encoded circular RNA hsa_circ_0089762 in Diabetic Foot Ulcer (DFU)
Yashar Mehrbani Azar	014	Motion	Recovery from radiation-induced damage to growth plates involves functional compensation
Alma Månsson	015	Motion	Identification of a cytokine as potential therapeutic target in Chronic Myeloid Leukemia Patient Bone Marrow Niche
Baptiste Jude	016	Motion	Induction of arthritis results in capillary rarefaction and reduced glucose uptake in skeletal muscle
Clara Chamorro	017	Motion	Bladder micrografting for bladder reconstruction. An in vitro study

Abstracts

Keynote Speakers

Professor Emeritus Christer Sylvén

Department of Medicine, Huddinge

Karolinska Institutet

Wednesday 9th, 09:00

Human heart and cardiomyocyte development during early fetal life

Spatial transcriptomics (ST) and single cell transcriptomics (scRNA) were performed on fetal human heart. scRNA revealed 17 cell types. Cardiomyocytes were subclustered. Differential expression and gene ontology analysis suggested spatially niched cardiomyocyte types that was confirmed in ST maps. Heterogeneity was high both within and between cardiomyocyte types. Ligand-recceptor interactions were low between cardiomyocytes and high between cardiomyocytes and other cell types. Using in situ sequencing, diffusion and pseudotime maps suggested that epithelial cells via epithelial mesenchymal transition can develop both into cardiomyocytes and fibroblasts.

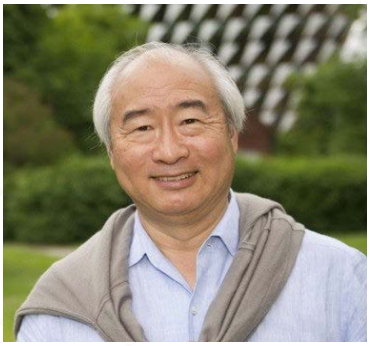
In conclusion, in the human fetal heart cardiomyocytes develop and adapts to the local environment with a more fibroblast-like phenotype in the conduit parts of atria and ventricles.

Prof. Kenneth R. Chien

Department of Cell and Molecular Biology

Karolinska Institutet

Wednesday 9th, 11:25



Rebuilding the Failing Heart with Human Ventricular Progenitors (HVPs)

Heart regeneration is an unmet clinical need, hampered by limited renewal of adult cardiomyocytes and fibrotic scarring. Stem cell-based strategies are emerging, but the identification of the ideal cell therapeutic has been challenging (1), as well as defining the pathways the mediate specific steps (i.e., survival, expansion, vascularization, migration, maturation, and electrical coupling) critical for the formation of sufficiently large ventricular muscle grafts that can modify global function of a failing human heart.

For the past two decades, our lab has been developing a lineage map of heart progenitors during cardiogenesis (2-10), but, until recently, has not identified the ideal heart progenitor worthy of clinical translation. Toward this goal, our previous studies have identified a unipotent ISL+ ventricular progenitor in murine model systems (11), and most recently, via the development of an optimized protocol, have been able to generate billions of human ISL+ ventricular progenitors from human pluripotent stem cells that are fully capable of forming large chunks of functioning human ventricular muscle during *in vivo* grafting (12). Recently, by combining lineage-tracing and single-cell transcriptomics in injured non-human primate heart bio-mimics, we uncover the coordinated action modes of human progenitor-mediated muscle repair (13). Chemoattraction *via* CXCL12/CXCR4 directs cellular migration to injury sites. Activated fibroblast repulsion targets fibrosis by SLIT2/ROBO1 guidance in organizing cytoskeletal dynamics. Ultimately, differentiation and electromechanical integration lead to functional restoration of damaged heart muscle. *In-vivo* transplantation into acutely and chronically injured porcine hearts illustrated CXCR4-dependent homing, *de novo* formation of heart muscle, scar-volume reduction, and prevention of heart failure progression. Concurrent endothelial differentiation contributed to graft neovascularization. Our most recent studies show a dramatic improvement in the incidence of life threatening ventricular arrythmias following transplantation into a porcine MI model versus previous reports utilizing differentiated human ES derived cardiomyocytes as the cell therapeutic. Our studies demonstrate that inherent developmental programs within cardiac progenitors are sequentially activated in disease, enabling the cells to sense and counteract acute and chronic injury. Furthermore, they provide direct *in vivo* proof of concept of the clinical tractability of HVPs for functional ventricular muscle regeneration for patients with severe heart failure, setting the stage for FTIH studies in 2024.

Prof. Ulf H. Lerner

Center for Bone and Arthritis Research at

Institute for Medicine, Sahlgrenska

Academy

University of Gothenburg

Wednesday 9th, 15:30

Wnt signaling system regulates cortical and trabecular bone differently

The family of WNT ligands signals through canonical pathway by binding to a complex between Frizzled receptors and LRP co-receptors, a signaling pathway that can be inhibited by several extracellular inhibitors such as sclerostin, Dickkopfs and secreted Frizzled-related proteins (sFRPs). WNTs can also signal through non-canonical pathways such as WNT/Ca^{2+}-dependent and WNT/polar cell polarity pathways. WNTs have numerous functions during development and in adult life. WNT was originally found to be important for embryonic development in *Drosophila* during the mid 1970´s and for breast cancer in mice during early 1980´s. During the mid 1990´s, it was found that juvenile-onset osteoporosis in patients with osteoporosis pseudoglioma syndrome was associated with chromosome 11q12-13 and that high bone mass in individuals who did not fracture the skeleton despite great violence and who could not float when trying to swim could also be assigned to chromosome 11q12-13. During early 2000´s, it was found that the osteoporotic patients had a loss-of-function mutation in the *LRP5* gene, whereas those with high bone mass had a gain-of-function in the same gene. These observations were the first findings indicating that WNT signaling is of importance for bone mass. Later it has been shown that high bone mass in patients with van Buchem´s disease is due to loss-of-function in the *SOST* gene, encoding the inhibitor sclerostin and recently an inhibitor of sclerostin has been approved as an anabolic drug for treatment of osteoporosis. Mutations of several WNTs, inhibitors, activators, receptors, co-receptors and intracellular regulators have been shown to be associated with different human skeletal dysplasias. In large-scale genome-wide association studies (GWAS) several foci have been shown to be associated with bone mineral density and fracture susceptibility. Among those, *WNT16* locus is the strongest genetic determinant for fractures and *RSPO3* is the second strongest locus. In clinical and translational studies, we have shown that WNT16 regulates preferentially cortical bone mass with no effect on trabecular bone, whereas RSPO3 is a regulator of trabecular bone with no effect on cortical bone. The experimental studies in mice demonstrating these differences and the mechanistic studies explaining how WNT16 and RSPO3 regulates bone resorption and bone formation will be presented.

Abstracts

Invited Speakers

Dr. Siqin Wu

Department of Clinical Science, Intervention and Technology

Karolinska Institutet

Wednesday 9th, 10:00

Human Pluripotent Stem Cell Derived Pancreatic Islets for Treatment of Type 1 Diabetes

Type 1 diabetes can be treated with transplantation of cadaveric pancreatic islets, but this approach is limited by shortage of donor islets and low viability of islets after transplantation. Transplantation of pluripotent stem cell derived pancreatic islets (SC-islets) into the anterior chamber of eye (ACE) can potentially solve these problems by utilizing a renewable cell source and the possibility of non-invasive longitudinal evaluation post transplantation. In this study, we have developed a new protocol for *in vitro* differentiation of hPSCs into functional SC-islets. The SC-islets showed good proportions of mono-hormonal α and β cells and strong glucose responsiveness *in vitro*. To understand if ACE is suitable for clinical transplantation, we transplanted SC-islets into ACE of a large-eyed pre-clinical animal model. Our results show that the injected SC-islets were integrated onto the iris tissue and became vascularized. The transplants closely resemble human islets in both size and morphology, and contain mainly mono-hormonal endocrine cells expressing either insulin, glucagon or somatostatin. Human insulin c-peptide could be detected in both serum and anterior chamber fluid collected after transplantation. We also transplanted SC-islets into both ACE and kidney capsule of a diabetic mouse model. Two months after transplantation, SC-islets reversed diabetic symptoms in mice. Taken together these results show that ACE transplantation of SC-islets generated using our new protocol could be an ideal solution for treatment of type 1 diabetes in the future.

Dr. Huan Cai

Department of Medicine, Huddinge

Karolinska Institutet

Wednesday 9th, 11:00

Role of Laminin α4 in leukemia progression and drug resistance

Impairment of normal hematopoiesis and leukemia progression are 2 well-linked processes during leukemia development and are controlled by the bone marrow (BM) niche. Extracellular matrix proteins, including laminin, are important BM niche components. However, their role in hematopoiesis regeneration and leukemia is unknown. Laminin a4 (Lama4), a major receptor-binding chain of several laminins, is altered in BM niches in mice with acute myeloid leukemia (AML). So far, the impact of Lama4 on leukemia progression remains unknown. We here report that Lama4 deletion in mice resulted in impaired hematopoiesis regeneration following irradiation-induced stress, which is accompanied by altered BM niche composition and inflammation. Importantly, in a transplantation-induced MLL-AF9 AML mouse model, we demonstrate accelerated AML progression and relapse in *Lama4*$^{-/-}$ mice. Upon AML exposure, *Lama4*$^{-/-}$ mesenchymal stem cells (MSCs) exhibited dramatic molecular alterations, including upregulation of inflammatory cytokines that favor AML growth. *Lama4*$^{-/-}$ MSCs displayed increased antioxidant activities and promoted AML stem cell proliferation and chemoresistance to cytarabine, which was accompanied by increased mitochondrial transfer from the MSCs to AML cells and reduced reactive oxygen species in AML cells *in vitro*. Similarly, we detected lower levels of reactive oxygen species in AML cells from *Lama4*$^{-/-}$ mice postcytarabine treatment. Notably, LAMA4 inhibition or knockdown in human MSCs promoted human AML cell proliferation and chemoprotection. Together, our study for the first time demonstrates the critical role of Lama4 in impeding AML progression and chemoresistance. Targeting Lama4 signaling pathways may offer potential new therapeutic options for AML.

Dr. Wim Grooten

Department of Neurobiology,

Care Sciences and Society

Karolinska Institutet

Wednesday 9th, 13:45

Qualitative and quantitative movement analyses in the clinic and research

What is "*movement*"? In physics, movement is defined as "*a change in position*", according to the Cambridge dictionary there are several other meanings related to "movement", such as "*a group of people with a particular set of aims or ideas*" (for example the antiwar movement), or "*an occasion when something develops, changes, or happens in a particular way or direction*" (for example in pedagogics: "Recently there has been some movement away from traditional methods of teaching").

In my speech, I will start with a focus based on the first definition, by talking about the kinematic, kinetic, and motor control parts of movements and how we can measure these features validly and reliably. I will show some examples from our uMove center and our movement laboratory together with some clinical tests and elaborate on the difference between quantitative and qualitative analyses of human movement. However, by the end of the speech, I hope that I have started *a movement* with ki-researchers toward a greater understanding of how important it is to incorporate *movement* in all of the studies that are performed within the Tissue and Motion network.

Dr. Phillip Newton

Department of Women's and Children's Health

Wednesday 9th, 14:10

Recovery from radiation-induced damage to growing bones involves functional compensation by growth plate chondrocytes

An important function of our skeletons is their involvement in locomotion. If the skeleton grows asymmetrically during childhood, affected individuals can experience impaired mobility involving, for example, leg-length differences or scoliosis/kyphosis. Childhood cancer patients commonly receive radiotherapy for various types of cancer, which is a highly effective treatment. However, radiotherapy can cause skeletal asymmetry because radiation directly damages the organs responsible for bone elongation: epiphyseal growth plates. Growth plates house epiphyseal stem cells, which give rise to columns of cartilage cells (chondrocytes) that are required to fuel long-bone growth. Using clonal genetic tracing in mice to visualize the cellular recovery within growth plates after irradiation, we made two inter-connected observations: radiation dose-dependently (i) prevents a larger proportion of chondrocytes from further dividing and, (ii) causes remaining chondrocytes to generate significantly increased numbers of columns. Hence, we found that growth plate chondrocytes have the ability to functionally compensate for the lost column-forming cells. These findings improve our understanding of the healing process occurring in growth plates following irradiation and may allow us to devise better treatment strategies to prevent skeletal asymmetry in children undergoing radiotherapy.

Dr. Sara Windahl

Department of Laboratory Medicine

Karolinska Institutet

Wednesday 9th, 14:35

Bone forming effects of mechanical loading in mice

Loading is the major determinant of bone architecture and strength. This talk will begin with a brief history of the discovery of the importance of load for bone architecture, and then I will talk about more recent research on the mechanisms whereby loading effects bone architecture. The talk will cover the importance of tartrate resistant acid phosphatase on the osteogenic effects of loading as well as some sex-, age- and site-specific effects of loading.

Dr. Sebastian Lewandowski

Department of Clinical Neuroscience

Karolinska Institutet

Wednesday 9th, 15:00

On brains and vessels, how perivascular fibroblasts contribute to ALS neurodegeneration

For decades, ALS has been defined, diagnosed and evaluated based almost exclusively on symptoms of rapid, age-dependent degeneration of motor neurons. Apart from the well-defined neuron-centric factors, few reports consider that variability of sporadic ALS progression can depend on the less-defined contributions from non-neuronal cell types including glia and blood vessels. Nonetheless, inaccurate survival prognosis continues to confound clinical trial design and effective treatments will likely remain elusive unless we better understand how non-neuronal cells contribute to ALS aetiology. We found that perivascular fibroblast cell gene activity during presymptomatic disease stage remodels blood vessel matrix and provides distinct plasma protein biomarker that can independently predict short ALS patient survival at diagnosis.

Panelists Profile

Dr. Evren Alici

Principal Investigator
Department of Medicine, Huddinge
Karolinska Institutet

Co-director
NextGenNK

Evren Alici received his MD at Ege University in 1999 and received his PhD from Karolinska Institutet in 2006. He leads his group with the primary research focus to optimize and clinically adapt strategies to retarget natural killer (NK) cells to tumors with limited off-target effects and toxicity profiles. For this purpose, his group is currently performing clinical trials where NK cell responses are enhanced and retargeted against tumor cells in patients with multiple myeloma. More specifically, his group aims to develop novel NK cell-based therapies including next-generation engagers, next-generation chimeric antigen receptors for NK cells (CAR-NK), and next-generation small molecules for NK cell modulation.

Align with this purpose, Evren is the co-director of NextGenNK, a Competence Center for the development of next generation NK cell-based cancer immunotherapies. The Center is coordinated by Karolinska Institutet and collaborates with the Karolinska University Hospital and prominent national and international industrial partners. The Center was launched in 2020, and is jointly funded by Vinnova, KI, and industrial partners.

Dr. Elisabet Åkesson
Adjunct Senior Lecturer
Department of Neurobiology, Care Sciences and Society

Acting head of R&D
Stockholms Sjukhem

Elisabet Åkesson is a senior lecturer and has a group with the interest in central nervous system lesions including spinal cord injury and treatment strategies to counteract neurodegenerative processes. Her group develops and utilizes novel and unique model systems to evaluate specifically human spinal cord injury processes and repair strategies. In addition, her group also participates in the international clinical trials TransEuro and BOOSTB4.

Moreover, Elisabet is a clinical physician and the coordinator of R&D activities in clinical rehabilitation and primary care research at Stiftelsen Stockholms Sjukhem.

Bramasta Nugraha, Ph.D.

Associate Principal Scientist *in vitro* imaging specialist in cardiovascular, renal and metabolism

AstraZeneca

BioPharmaceuticals R&D,

Early CVRM, Bioscience Cardiovascular

Bramasta (Bram) is a passionate researcher in multidisciplinary and multicultural environments. A global citizen with experience studying and working in Indonesia, Singapore, Taiwan, Japan, USA, Switzerland and Sweden. His current employment at AstraZeneca is to lead and support drug discovery pipeline research projects involving complex *in vitro* 2D and 3D cellular models and advanced high throughput/high content imaging to evaluate cellular and molecular events as lead identification and optimization in the early drug discovery within the therapeutic research areas of cardiovascular, renal and metabolic diseases (CVRM). He also manages the imaging modality, data curation and analysis within CVRM department and he is working with various scientists across different AstraZeneca sites across the globe as a solid and experienced team player.

Roger Chang, Ph.D.

Life Science Sales Manager

Somalogic

Roger Chang is currently an account manager at TATAA Biocenter but will later take on the role as life science sales manager for Somalogic. He received his doctorate at Karolinska Institutet researching on microRNA and cancer in Dr. Weng-Onn Lui's group. He was passionate about RNA research and decided to form Stockholm RNA club, a network for RNA researchers. It was through the RNA club that Roger met Prof. Victor Ambros, who discovered the first microRNA who later became his mentor and postdoc advisor. In the Ambros laboratory, UMass Medical School, USA, Roger was working on a novel technology to study RNA-proteins interactions *in vivo*.

In September 2021, Roger transitioned to a commercial role in TATAA Biocenter working as a key account manager. TATAA Biocenter is a Swedish CRO that provide molecular analysis services for pharma, biotech, and academic clients. A year later, Roger was headhunted by Soma logic to join their team as life science sales manager for Sweden. Somalogic is a global leader in proteomics and has developed a unique proteomic platform that can measure over 7000 proteins. The aim is to revolutionize precision medicine to help address unmet medical needs.

Mochammad Fadjar Wibowo, M.Sc.

International Health Specialist

Asian Development Bank (ADB)

Fadjar is a global health professional with eleven years of combined experience in managing global public health projects with civil society, academia, and the public sector and adding value to the medical and safety operational strategy of digital health and ride-hailing platforms in Southeast Asia.

He holds a medical doctor degree from Gadjah Mada University, master in global Health from Karolinska Institute, and a Diploma in Sustainability Management from the Swedish Institute. Applying comprehension in global health, health policy, management, economics, and sustainability management, he translates insight to recommend effective and sustainable health and safety interventions at the global, national, and digital platform levels. His works have been recognized by Indonesia's National Development Planning Agency, Swedish Institute, the Australia Department of Foreign Affairs and Trade, and the UK Royal Public Health Society. He has also shared opinions with the US, China, and Indonesia media on global health and digital health topics.

Anna Vidina, M.Sc.

Impact Officer

Global Shapers Stockholm

After receiving a BSc degree in Biomedicine from Karolinska Institutet, participating in the Amgen Scholars program at Institut Pasteur and completing her BSc thesis at ETH Zurich, it was clear for Anna that she wanted to continue her career at the intersection of basic research and its applications to day-to-day life. Anna proceeded with studying the Erasmus Mundus Joint Master Degree in Innovative Medicine and acquiring her first experience working in the biotech industry. She worked on the Vanadis NIPT assay at PerkinElmer for 3 years to enable better family planning for those expecting. Having experienced the corporate world, Anna observed that project and team management has a huge impact on the output of results. She decided to broaden her knowledge with a MSc in Entrepreneurship and Innovation Management at KTH in Stockholm and she is currently finding her way into the startup world.

Tissue and Motion Conference 2022

Organizing Committee

From left to right:

Bhavik Rathod

Sarah Saietz

Frederik Bär

Begüm Gençer Öncül

Elory Leonard


Thank you
and
see you next year