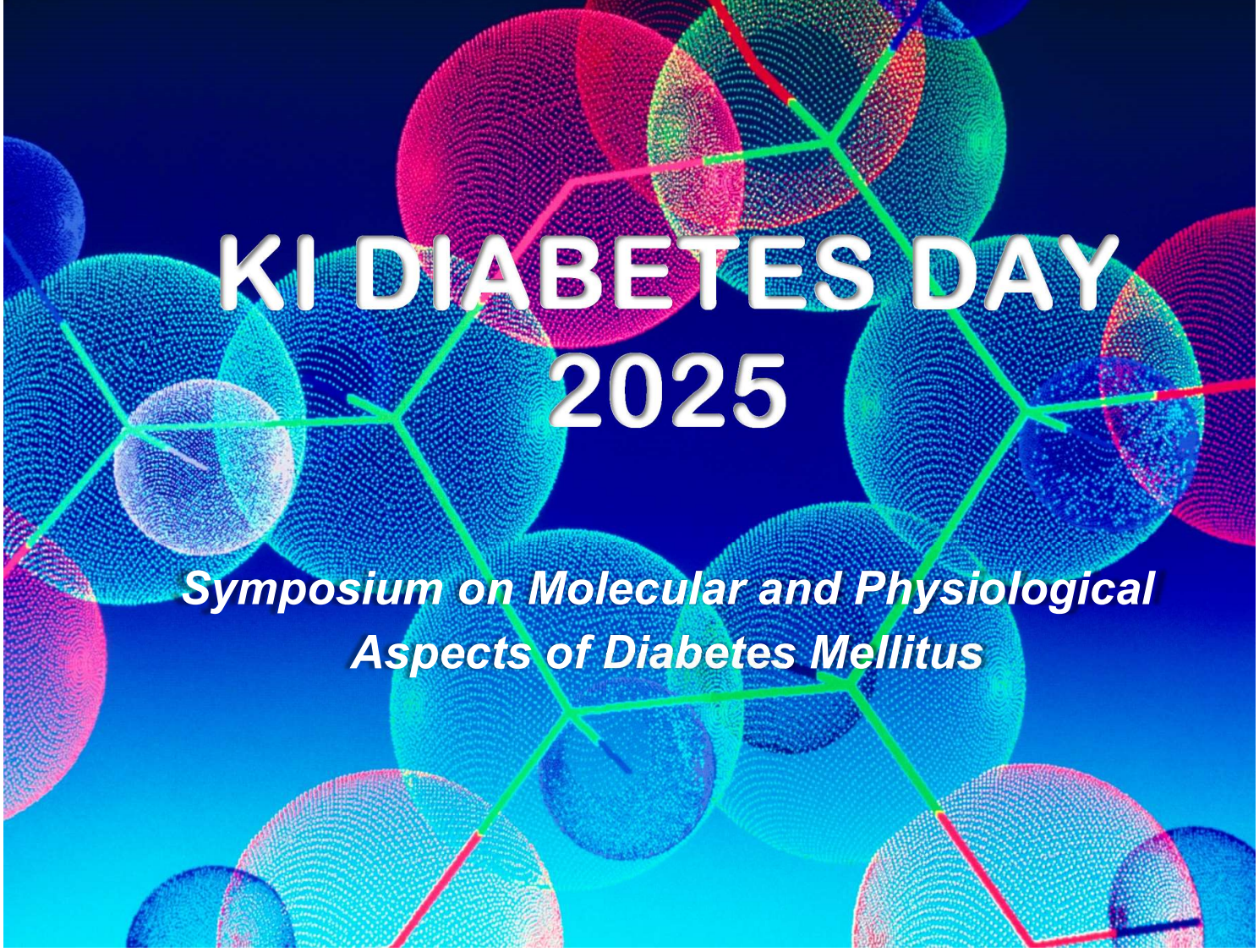

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POSTER ABSTRACTS

world diabetes day

SRP DIABETES

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Poster #1

Blood Glucose and Stress Markers Elevated after Morning versus Afternoon Exercise in Type 2 Diabetes

Martin J. Keller, Aidan J. Brady, Jonathan A. B. Smith, Mladen Savikj, Kirstin MacGregor, Maxence Jollet, Sofia B. Öberg, Carolina Nylén, Marie Björnholm, Anette Rickenlund, Marcus Carlsson, Kenneth Caidahl, Anna Krook, Nicolas J. Pillon, Juleen R. Zierath, Harriet Wallberg-Henriksson

Karolinska Institutet

Aims: The aim of this study was to investigate effects of time-of-day on glucose management in individuals with type 2 diabetes undertaking high-intensity interval exercise. Additionally, the association between eating behaviour and mean amplitude of glycaemic excursions was examined.

Methods: A crossover trial was conducted, comprising 12 men and 12 women with type 2 diabetes and 12 men and 12 women without diabetes. Participants performed high-intensity interval sessions in the morning (09:00 hours) or afternoon (16:00 hours) on separate days at least 7 days apart. Standardised meals were provided the day before exercise, on the day of exercise and on the day after exercise. Continuous glucose monitoring was used to estimate blood glucose levels.

Results: Morning exercise increased blood glucose during the 2 h post-exercise period in men ($p<0.05$) and women ($p<0.01$) with type 2 diabetes, but blood glucose was unaltered following afternoon exercise. Glycaemic variability was reduced during the 3-day meal intervention in men ($p<0.001$) and women ($p<0.05$) with type 2 diabetes, but not in individuals without diabetes. Participants exhibited higher morning cortisol levels ($p<0.001$) compared with afternoon cortisol levels, independently of diagnosis. Individuals with type 2 diabetes exhibited higher levels of the inflammation marker C-reactive protein ($p<0.001$) and the heart failure marker NT-proBNP ($p<0.001$) in the morning than in the afternoon.

Conclusions: In type 2 diabetes, afternoon high-intensity interval exercise appears to be more effective than morning high-intensity interval exercise for maintaining glucose management. Additionally, consistent meal timing and controlled energy intake are recommended for reducing the mean amplitude of glycaemic excursions.

Poster #2

Exploration of glucose metabolism in adipose tissue-specific Diacylglycerol Kinase Delta knock-out mice

Maxence Jollet, Dimitrius Santiago Guimarães, Joaquin Ortiz de Zevallos, David Rizo-Roca, Marie Björnholm, Anna Krook, Juleen R. Zierath

Karolinska Institutet

Type 2 diabetes mellitus (T2D) is a progressive metabolic disorder resulting from genetic and environmental factors that impair insulin secretion and action in peripheral tissues. Our previous research identified reduced diacylglycerol kinase delta (DGKδ) expression and DGK activity in skeletal muscle from individuals with T2D. In mice models, we demonstrated that reducing DGKδ activity leads to aberrant insulin signalling associated with reduced glucose uptake in skeletal muscle and adipose tissue. Since glucose uptake in skeletal muscle and adipose tissue accounts for the majority of peripheral glucose disposal, we generated adipose tissue-specific DGKδ knock-out mice (KO) to elucidate the tissue-specific contribution of DGKδ to systemic glucose homeostasis and insulin sensitivity. MRI-based body composition analysis revealed comparable lean and fat mass between KO and wild-type (WT) littermates within each sex. Glucose tolerance was unaltered between genotypes. Additionally, the euglycemic-hyperinsulinemic clamp assay indicated similar glucose infusion rate. Gene expression analysis of fatty acid metabolism markers in white and brown adipose tissue showed no significant differences between KO and WT animals. However, the primary component of triglycerides hydrolysis into fatty acid (ATGL) exhibited a significant reduction ($p<0.05$) in adipose tissue-specific DGKδ KO mice, specifically in brown adipose tissue. Despite the downregulation of DGKδ in its primary site of expression, adipose tissue, our findings suggest other organs, most likely the skeletal muscle, as the key site for DGKδ-mediated metabolic regulation. Further investigations under metabolic stress conditions, including high-fat diet, are warranted to elucidate the potential role of adipose tissue-specific DGKδ expression in the pathogenesis of type 2 diabetes and obesity.

Poster #3

Elucidating the function of BUD13 in the pathogenesis of vascular endothelial dysfunction associated with type 2 diabetes

Álvaro Santana-Garrido, Eftychia Kontidou, Allan Zhao, Jiangning Yang, Tong Jiao, Qiaolin Deng, John Pernow, Aida Collado, and Zhichao Zhou

Karolinska Institutet & Karolinska University Hospital

Background: Cardiovascular diseases in type 2 diabetes (T2D), including both macro- and microvascular complications, remain a leading cause of mortality worldwide. Despite their high prevalence, effective therapeutic options are currently limited. BUD13, a nuclear RNA-binding protein, plays a crucial role in RNA splicing. Emerging evidence suggests its involvement in the pathogenesis of metabolic syndrome; however, the role of BUD13 in cardiovascular disease is unclear. This study investigates the contribution of BUD13 to T2D-associated cardiovascular complications.

Methods: BUD13 expression was assessed in the aorta (macrovasculature) and in ocular vascular tissues (choroid, microvasculature) from wild-type (WT) and db/db mice (a T2D model). Endothelium-dependent relaxation (EDR) was measured in db/db aortas pre-treated with BUD13 siRNA using wire myograph. In human carotid arterial endothelial cells (HCtAECs), the formation of reactive oxygen species (ROS), nitric oxide (NO) production, and cell migration were measured following exposure to high glucose (HG), with/without BUD13 siRNA. RNA sequencing and transcriptomic analysis were also performed.

Results: BUD13 was predominantly localized to the endothelium of aortic and choroidal vessels, with elevated expression in db/db mice compared to WT mice. EDR was impaired in db/db aortas but was significantly attenuated by the BUD13-siRNA. BUD13-siRNA restored the disrupted ROS/NO balance and normalized cell migration in HG-treated HCtAECs. Transcriptomic analysis revealed that BUD13 regulates key pro-inflammatory signaling pathways.

Conclusion: Our findings suggest that endothelial BUD13 contributes to T2D-related vascular dysfunction through oxidative and inflammatory pathways. Targeting endothelial BUD13 may represent a potential therapeutical strategy for preventing both macrovascular and microvascular complications in T2D.

Poster #4

Whole-Organ Imaging of Mouse Pancreatic Endocrine Cells Uncovers Regional Islet Heterogeneity

Azam Mahmoudi-Aznaveh, Björn Morén, Stefanie M. A. Willekens, Tomas Alanentalo, Ulf Ahlgren

Umeå University

Using deep-tissue optical 3D imaging, we recently demonstrated that human islets are far more heterogeneous than previously believed regarding their size distribution and cellular makeup. Strikingly, 50% of Insulin positive islets are glucagon negative, an observation with a wide range of potential implications for islet research. Although the mouse is the predominant model system for pre-clinical diabetes research, whole-organ analyses of islet cellularity have not been reported in this species at a similar level of detail.

Aim:
To investigate inter- and intra-lobular differences in endocrine density and islet cellularity across the entire volume of the mouse pancreas with regard to insulin (INS), glucagon (GCG), somatostatin (SOM), pancreatic polypeptide (PP), and ghrelin (GHR) producing cells.

Methods:
This study employed optical 3D imaging to generate comprehensive whole-organ maps of islet cellularity across the pancreas in male and female C57Bl/6 mice (n=3+3), at microscopic resolution. The protocol includes organ dissection, ex vivo antibody labelling, tissue clearing, 3D imaging via Light Sheet Fluorescence Microscopy (LSFM), and image processing using Imaris software for 3D visualization and quantification.

Results and conclusion:
Our data show significant differences in b-cell density between the lobular compartments of the mouse pancreas. Further, they lay bare regional differences in islet cellularity with regard to whether INS-positive islets harbor GCG or PP cells. In all, our data underscore that studies of the mouse pancreas and of isolated islets must take their spatial origin into account.

Poster #5

Deciphering molecular pathways of exercise in type 2 diabetes through multi-omic approaches

Ben Stocks, Stephen P Ashcroft, Jeppe Kjærgaard, Signe Schmidt Kjølner Hansen, Kirstin MacGregor, Dimitrius Guimaraes, Marc Pielies Avelli, Simon Wengert, Mladen Savikj, David Rizo-Roca, Roger Moreno-Justicia, Julia Otten, Tommy Olsson, Simon Rasmussen, Kenneth Caidahl, Harriet Wallberg-Henriksson, Anna Krook, Atul S Deshmukh, and Juleen R Zierath

University of Copenhagen & Karolinska Institutet

Exercise is a potent intervention for type 2 diabetes, enhancing glucose uptake and increasing insulin sensitivity even after a single session. Despite these well-documented clinical observations, the underlying molecular mechanisms mediating these metabolic adaptations remain incompletely elucidated. Employing an integrative multi-omics approach, encompassing transcriptomics, proteomics, phosphoproteomics, and metabolomics, we comprehensively investigated the molecular responses of skeletal muscle immediately following exercise and after three hours of rested recovery in a cohort of 88 men and women. We delineated exercise-responsive signaling pathways involved in glucose metabolism and insulin sensitivity in type 2 diabetes. In particular, we observed a conserved upregulation of proteins and phosphosites involved in RHO GTPase cycling after exercise, including IQGAP1, which we found to regulate glycogen synthesis in human primary muscle cells. Conversely, we observed an elevated immune response to exercise and a proteomic signature indicative of impaired mitochondrial protein turnover in individuals with type 2 diabetes. These multi-omic insights advance the understanding of the molecular pathways underlying the beneficial effects of exercise and identify candidate molecular targets for future therapeutic strategies aimed at optimising and replicating the metabolic benefits of exercise in individuals with type 2 diabetes.

Poster #6

Validated preclinical mouse models of type 2 diabetes mellitus

Takashi Willebrand, Amanda Miles, Jing Zhang, Qikuan Chen, Yinfei Yin

ChemPartner

Obesity has reached epidemic proportions over the past four decades, driving major public health and economic challenges due to its association with insulin resistance, type 2 diabetes (T2D), and liver disease. T2D, a chronic disorder characterized by persistently elevated blood glucose, affects approximately 6% of the global population and contributes to an estimated $827 billion in annual healthcare costs.

ChemPartner offers a suite of validated preclinical mouse models for T2D research, with established standard-of-care (SoC) treatments and accelerated study timelines. Two mutant strains have been utilized to generate robust models: the leptin receptor-deficient db/db mouse, commonly used in T2D studies, and the leptin-deficient ob/ob mouse, frequently employed in obesity and diabetes research.

In the db/db model, alogliptin benzoate SoC treatment significantly reduced fasting blood glucose and HbA1c to prediabetic levels, enhanced insulin secretion, improved glucose tolerance, and lowered plasma lipid levels without affecting body weight. In the ob/ob model, semaglutide treatment reduced food intake, body weight, triglycerides, LDL-c, and adipose mass while modestly improving glucose tolerance. Pioglitazone treatment yielded reductions in glucose tolerance, triglycerides, and perirenal fat weight, without influencing food intake or body weight.

The global rise in obesity and T2D underscores the demand for reliable, well-characterized preclinical models. ChemPartner's db/db and ob/ob mouse models, supported by comprehensive metabolic, biochemical, and pathological assessments, provide a rapid, validated, and cost-effective platform to accelerate discovery and development in metabolic disease research.

Poster #7

Atypical ceramides in adipose tissue as potential mediators of insulin resistance and type 2 diabetes remission

Tova Eurén, Timotej Strmeň, Louise Nenzén, Elisabeth Stener-Victorin, Anja Dekanski , Lena Schobloch, Pär Steneberg, Helena Edlund, Julia Otten, Elin Chorell

Umeå University

Adipose tissue is a key regulator of insulin sensitivity, and its dysfunction contributes to insulin resistance and type 2 diabetes (T2D). Beyond fat mass reduction, remission of T2D requires restoration of healthy adipose function. Sphingolipids, particularly ceramides, are bioactive lipids that influence insulin signalling and lipid handling. Yet their role in adipose dysfunction remains poorly defined due to low abundance and analytical complexity. We hypothesize that specific ceramides, rather than inert storage lipids, accumulate in adipocytes under metabolic stress, driving inflammation, altered adipokine signalling, and impaired lipid metabolism, thus contributing to insulin resistance.

To test this, we profiled the lipidome, sphingolipidome, and transcriptome of adipose tissue from individuals with T2D (n=23) before and after a hypocaloric diet intervention, alongside baseline-matched overweight controls (n=12). Insulin sensitivity was assessed using a hyperinsulinemic-euglycemic clamp and remission defined as HbA1c <41mmol/mol. Parallel analyses were performed in mice fed a low-fat diet (LFD), high-fat diet (HFD), or HFD supplemented with the AMPK-activator ATX-304, which preserves insulin sensitivity despite adipose expansion. Lipidomic analyses were also conducted on adipose secretome and plasma.

Preliminary results reveal that adipose tissue from insulin-resistant mice and individuals with T2D accumulate saturated and monounsaturated atypical deoxyceramides, whereas these species are reduced in individuals achieving remission after caloric restriction. Unlike canonical ceramides, deoxyceramides cannot be converted into complex sphingolipids and may accumulate or be secreted into circulation, potentially impairing systemic lipid metabolism. Ongoing studies aim to define the mechanistic role of deoxyceramides in adipose tissue dysfunction and insulin sensitivity.

Poster #8

Whole organ 3D Characterization of β-Cell Topology in the T1D Pancreas - preferential preservation of extra-islet β-cells

Joakim Lehrstrand, Max Hahn, Björn Morén, Wayne. I. L. Davies, Olle Korsgren, Tomas Alanentalo, Ulf Ahlgren

Umeå University

Residual β-cell function may have positive effects on diabetes regulation in T1D. However, an exact account on the spatial distribution and mass of the remaining β-cells across the pancreas has not been presented. To address this, we implemented an optical 3D imaging pipeline to generate a first account of the 3D-spatial and volumetric distribution of the remaining β-cells throughout the volume of an entire human late-onset, long-standing T1D pancreas at a microscopic resolution. As expected, β-cell mass was dramatically lower than in a non-diabetic pancreas. Intriguingly, the pancreatic head displayed a morphology and size resembling the control pancreas and inhabited a 3 times higher β-cell density compared to the rest of the organ. Surprisingly, only a fraction of these residual β-cells were located within islet structures. Instead, the absolute majority were present as extra-islet β-cells, either as scattered individual cells or as clusters of β-cells, spatially separated from all other endocrine cell-types. Strikingly, these extra-islet β-cells appeared roughly 60x less prone to autoimmune destruction compared to islet associated β-cells.
This 3D whole organ depiction of a long standing T1D pancreas shows that individual β-cells may be preserved in a highly regionalized manner and emphasize that extra-islet β-cells might be a clinically important target for further research on how to protect and maintain β-cells in several forms of diabetes.

Poster #9

GRK-biased adrenergic agonists for the treatment of type 2 diabetes and obesity

Aikaterini Motso, Benjamin Pelcman, Anastasia Kalinovich, Volker M. Lauschke, Shane C. Wright, Tore Bengtsson

Stockholm University

Biased agonism of G protein-coupled receptors (GPCRs) offers potential for safer medications. Current efforts have explored the balance between G proteins and β-arrestin; however, other transducers like GPCR kinases (GRKs) remain understudied. GRK2 is essential for β2 adrenergic receptor (β2AR)-mediated glucose uptake, but β2AR agonists are considered poor clinical candidates for glycemic management due to Gs/cyclic AMP (cAMP)-induced cardiac side effects and β-arrestin-dependent desensitization. Using ligand-based virtual screening and chemical evolution, we developed pathway-selective agonists of β2AR that prefer GRK coupling. These compounds perform well in preclinical models of hyperglycemia and obesity and demonstrate a lower potential for cardiac and muscular side effects compared with standard β2-receptor agonists and incretin mimetics, respectively. Furthermore, the lead candidate showed favorable pharmacokinetics and was well tolerated in a placebo-controlled clinical trial. GRK-biased β2AR partial agonists are thus promising oral alternatives to injectable incretin mimetics used in the treatment of type 2 diabetes and obesity.

Poster #10

Calcium and Chloride Interaction in Ciliary Signaling Revealed in ß-Cells

Gonzalo Sanchez & Olof Idevall

Uppsala University

We study ciliary signaling in pancreatic ß-cells, we have established that the organelle is an autonomous and active compartment in the islet and that different pathways converge on ciliary calcium. We set out to understand a potential link between calcium and chloride transport in the cilium.
We used organoids grown from clonal β-cells as a model for both ciliary calcium imaging and immunofluorescence detection of proteins of interest.
We found that both ciliary GABA-B1 and SSTR3 receptors can trigger Ca2+ transients though with different features: GABA-B1 elicited signals are not mediated by Gi protein while SSTR3 evoked responses are Gi-dependent and modulated by cAMP. By scrutinizing Ca2+ dynamics at high temporal resolution we try to gain mechanistic insight of these two pathways.
We found that in chloride-free extracellular buffer the kinetics of spontaneous ciliary calcium transients were affected, displaying slower onset and reduced amplitude. In an attempt to characterize ciliary channels involved in modulation of calcium signaling, we undertook immunodetection and pharmacological studies. We were able to detect the ciliary localization of CFTR with an antibody targeting its extracellular loop 1, while ANO1 and ANO6, channels implicated in chloride homeostasis, were absent from primary cilia.
We used CFTRinh-172 (0.5 μM) to block CFTR, niclosamide (1 μM) for ANO1 and ANO6 blockade and MONNA (0.5 μM) for selective disruption of ANO6 in an attempt to interfere with calcium signaling.
Taken together, the data presented here indicate that ciliary transduction involves finely tuned non canonical mechanisms converging on calcium and that chloride and its channels and perhaps even transporters are players to take into account for understanding the function of the cilium.

Poster #11

Leveraging Organ Donor Biobanks for Tissue Immunology and Spatial Profiling in Type 1 Diabetes

Fabian Byvald, Emma E. Ringqvist[, Vera Nilsén, Carlos Fernández Moro, Stephanie L. Hunter, Lars Krogvold, Sarah J. Richardson, Knut Dahl-Jørgensen, Carl Jorns, Jenny Mjösberg, Marcus Buggert, Malin Flodström Tullberg

Karolinska Institutet

The IHOPE (Immunology Human Organ Donor Programme) biobank enables research in tissue immunology using organ samples from deceased organ donors. The biobank includes formalin-fixed paraffin-embedded tissue blocks from several human organs that were collected under standardized protocols and linked to donor metadata.

In this study, we focused on pancreatic tissue from IHOPE and compared it to pancreas samples from two additional biobanks: laparoscopic biopsies from recently diagnosed type 1 diabetes (T1D) patients from the DiViD biobank, and resection material from living organ donors from the Exeter Archival Diabetes Biobank. Our aim was to investigate whether the cytokine interferon-lambda (IFN-λ) is expressed in the pancreas at T1D onset.

Immunohistochemistry was used to detect and localize IFN-λ expression within the pancreas across the three cohorts. Whole-slide imaging was performed on the stained pancreas sections, and images were analysed using QuPath to quantify total cell numbers and IFN-λ positivity.

We observed positive IFN-λ staining in nearly all pancreas samples. Notably, IFN-λ(+) cells tended to reside in small clusters primarily located in the exocrine compartment. The IFN-λ(+) cell frequency in living donors was higher in pancreas specimen from recently diagnosed T1D patients compared to non-diabetes controls.

These findings provide new insights into pancreatic immune responses during early T1D and demonstrate the value of organ donor biobanks for translational immunological research. The IHOPE biobank provides a robust platform for high-resolution mapping of human tissue biology, enabling spatial analysis of cellular localization, gene and protein expression, and cell-cell interactions through advanced technologies such as spatial omics.

Poster #12

Environmental Microbial Diversity in Early Childhood Settings and Possible Links to Type 1 Diabetes Risk

Marianne Nymark, Emma Ringqvist, Marta Butrym, Anirudra Parajuli, Marja Roslund, Olli Laitinen, Aki Sinkkonen, Malin Flodström-Tullberg

Karolinska Institutet

The incidence of immune mediated diseases such as type 1 diabetes has increased in recent decades. Epidemiological evidence suggests that children raised in rural environments have a lower risk of developing these conditions, likely due to greater exposure to environmental microbes. Since young children spend substantial time in nurseries, the microbial diversity of these environments may influence immune development and disease risk.

The 2025 edition of Forskarhjälpen, "Bakteriejakten," is a citizen science initiative organized by the Nobel Prize Museum in collaboration with Karolinska Institutet. Schools across Sweden (n=33) collected samples from sandpits, slides, and entrances at nurseries in both urban and rural areas. Microbial composition was analysed by 16S sequencing. Environmental mapping was performed to quantify land types and urbanization. Information on diabetes incidence is collected from the Swedish Diabetes Registry.

Samples were collected during the spring (May 19–23; n = 567) and autumn (September 15–19; n = 531), encompassing 109 nurseries in urban locations and 85 in rural settings. DNA extraction and sequencing have been completed for 336 spring samples. On average, each sample contained 820 bacterial species (range: 130–1597), with a mean Shannon diversity index of 7.89 (range: 2.46–9.12). Sequencing will continue, alongside environmental mapping and analyses to explore associations between microbial diversity, geographic context, and disease incidence.

Long-term, this project aims to clarify whether biodiversity exposure influence the risk of type 1 diabetes and other immune-mediated diseases, ultimately informing strategies to enhance microbial diversity in early-life settings, including preschool playgrounds.

Poster #13

Reducing Animal Use While Enabling High-Frequency Proteomic Monitoring via Dried Blood Spots

Bleona Koxha, Annika Bendes, Anirudra Parajuli, Virginia M. Stone, Fabian Byvald, Emma E. Ringqvist, Marta Butrym, Niclas Roxhed, Jochen M. Schwenk, Malin Flodström-Tullberg

Karolinska Institutet

Background
Studies of the circulating proteome in experimental animal models - such as cytokine and chemokine production following infection, or changes in metabolic hormones - have traditionally required large blood volumes, often necessitating the sacrifice of an animal at each time point. There is a growing need for minimally invasive, high-frequency sampling strategies that can be applied across experimental models and, ultimately, in humans.

Aims
To demonstrate that microsampling using dried blood spots (DBS), combined with high-throughput proteomics, reduces the need for larger numbers of animals and enables frequent monitoring of the circulating proteome.

Methods
Minute blood samples (5 µl) were collected from the tail tip of NOD mice, either weekly during development of type 1 diabetes or daily after infected with Coxsackievirus-B3. Samples were dried on volumetric devices, stored at room temperature and analyzed using proximity extension assay (PEA)-based technology.

Results
Blood sampling was performed on unrestrained, unanesthetized animals, which tolerated the procedure well. Longitudinal proteomic profiling revealed dynamic changes preceding diabetes onset and during acute viral infection, including immune and metabolic pathways. Several of the changes were transient and would have been missed with less frequent sampling.

Conclusions
DBS combined with PEA-based proteomics enable high-frequency monitoring in small-volume samples. It eliminates the need for cold-chain storage and reduces animal use by enabling repeated sampling from the same individual, aligning with the 3R principle. This strategy is broadly applicable to any experimental model requiring longitudinal monitoring and supports translation to human studies for pre-symptomatic disease monitoring and precision intervention.

Poster #14

Long Duration of Type 2 Diabetes Promotes Erythrocyte-Induced Endothelial Dysfunction: Role of microRNA-210

Eftychia Kontidou, Aida Collado, Rawan Humoud, Kesavan Manickam, Tong Jiao, Michael Alvarsson, Jiangning Yang, Ali Mahdi, John Pernow, Zhichao Zhou

Karolinska Institutet

Background: Type 2 diabetes (T2D) increases cardiovascular risk, with endothelial dysfunction underpinning its complications. We previously demonstrated that red blood cells (RBCs) in T2D impair endothelial function via reduced miR-210. Prolonged diabetes elevates cardiovascular risk, however its influence on the RBC-mediated endothelial dysfunction remains unclear. We hypothesize that prolonged T2D duration exacerbates this dysfunction through altered RBC miR-210 levels.

Methods: RBCs were collected from wild-type mice (22 weeks), T2D db/db mice with different disease durations (7, 14, 22 weeks), and humans with newly diagnosed (<1 year) or long-standing T2D (>8 years). Rodent aortas were incubated with RBCs, followed by measurement of endothelium-dependent relaxation (EDR) using a wire myograph. miR-210 in RBCs was quantified by qPCR. In humans, EDR was reassessed in the newly diagnosed group after 8 years.

Results: db/db mice at all ages showed higher blood glucose than wild-type controls. RBCs from 14- and 22-week-old db/db mice impaired EDR, whereas RBCs from 7-week-old db/db mice did not. RBCs from 22-week-old wild-type mice also did not impair EDR, indicating that T2D duration, not aging, drives dysfunction. miR-210 levels in RBCs were lower in 14- and 22-week-old db/db mice than in 7-week-old mice. In humans, RBCs from individuals with long-lasting T2D, but not newly diagnosed T2D, impaired EDR. After 8 years, RBCs from the newly diagnosed group also impaired EDR, an effect improved by miR-210 mimic.

Conclusions: The duration of T2D appears to be a key factor in RBC-induced endothelial dysfunction, with this effect linked to reduced miR-210 levels in RBCs.