IDNC ANNUAL MEETING 2018

DIABETIC NEUROPATHY AND THE WAY FORWARD
7-8 JUNE 2018, AARHUS, DENMARK

AARHUS UNIVERSITY

SOLIDUM PETIT IN PROFUNDIS · UNIVERSITAS ARHUSIENSIS ·

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GENERAL INFORMATION

VENUE
Radisson Blu Scandinavia Hotel
Margrethepladsen 1
DK-8000 Aarhus
Phone: +45 8612 8665
E-mail: guest.aarhus@radissonblu.com

DATES
7-8 June 2018

PARKING
Parking spaces for more than 1.400 cars are available on the premises. Parking is free of charge for all meeting delegates. Ask our staff at the registration desk for a parking ticket to exit from the car park.

INTERNET ACCESS
Free Internet is available for all meeting delegates. You will find wireless internet access throughout the hotel so you can use your computer in the meeting rooms, lobby areas and restaurants.
Network: RadissonGuest

FOOD AND DRINK
Buffet lunches and coffee breaks as indicated in the programme are included. Fresh water is available in the conference room.

ORGANIZER
International Diabetic Neuropathy Consortium
www.idnc.au.dk
The International Diabetic Neuropathy Consortium was awarded a grant of 60 million Danish Kroner for a 6-year period from the Novo Nordisk Foundation in December 2014.

novo nordisk fonden

ORGANIZING AND SCIENTIFIC PROGRAMME COMMITTEE
Troels Staehelin Jensen (Aarhus/DK)
Nanna Finnerup (Aarhus/DK)
Henning Andersen (Aarhus/DK)

CONGRESS ORGANIZATION
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REGISTRATION
Check-in at the registration desk. Our staff will be there to assist you. All delegates will receive a name badge. Admittance to the meeting is only allowed for those with a name badge. Name badges should be worn at all times to promote networking and help staff identify you.

LANGUAGE
The meeting language of the IDNC is English.

VENUE

1 Oral presentations

2 Buffet lunches

3 Coffee breaks and Poster viewing

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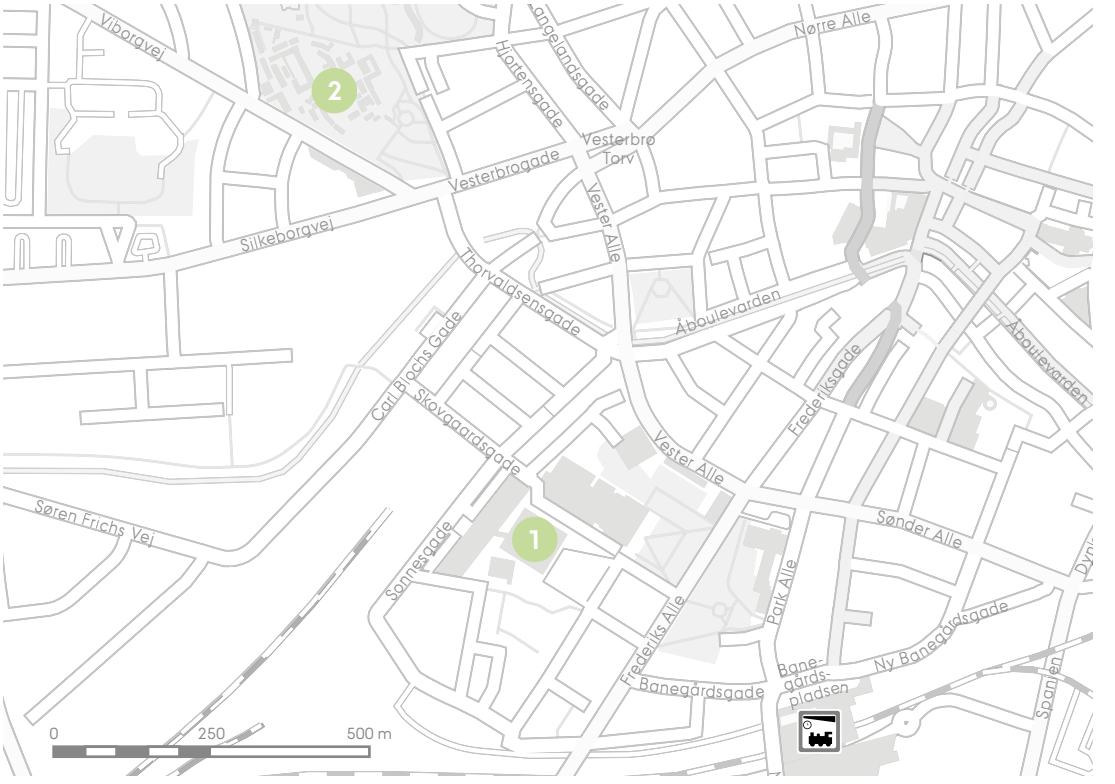

1 **VENUE**: Radisson Blu Scandinavia Hotel, Margrethepladsen 1

2 **EVENT**: Den Gamle By, Viborgvej 2

WELCOME NOTE

Dear colleagues and friends,

It is a great pleasure to welcome you to the Annual Meeting 2018 of the International Diabetic Neuropathy Consortium (IDNC) in Aarhus.

At this meeting, a panel of international experts will discuss different aspects of type 2 diabetes including epidemiology of type 2 diabetes and obesity, experimental studies in diabetic neuropathy, effects of exercise in diabetes, the staging of diabetic neuropathy, autonomic neuropathy in diabetes, and the role of the Steno Diabetes Centers in Denmark for research and management.

Welcome to Aarhus!

On behalf of the Organizing Committee,

Troels Staehelin Jensen
Professor, Director of the IDNC

IDNC AT A GLANCE

Vision: To be a leading research group on diabetic neuropathy in Denmark with an outreach to the world.

Mission: To study and unravel pathophysiological mechanisms of diabetic neuropathy, contribute to early identification, improve, and develop a uniform international classification in order to better treat and prevent the detrimental consequences of diabetic neuropathy. The IDNC does so by bringing researchers and clinicians together in a stimulating and multidisciplinary environment in order to integrate and facilitate translational aspects of diabetic neuropathy.

Structure: A series of interlinked work packages devoted to explore the mechanisms, risks, prognostic factors, and clinical profiles of diabetic patients with and without neuropathy.

Funding: A 6-year Novo Nordisk Foundation Challenge Programme grant (Grant number NNF14OC0011633).

THURSDAY, 7 JUNE

08:00-09:00 **Registration and coffee**

09:00-09:30 **Welcome and introduction**
– Troels S. Jensen, Director of IDNC (Denmark)

SESSION 1: EPIDEMIOLOGY OF TYPE 2 DIABETES AND OBESITY
Chair: Morten Charles (Denmark)

09:30-10:00 **Epidemiological studies in the ADDITION cohort**
– Daniel Witte (Denmark)

10:00-10:30 **The role of fat in diabetic neuropathy**
– Brian Callaghan (USA)

10:30-11:00 **Coffee break**

SESSION 2: EXPERIMENTAL FINDINGS IN DIABETIC NEUROPATHY
Chair: David Bennett (UK)

11:00-11:30 **Lipid changes in diabetic neuropathy**
– Eva Feldman (USA)

11:30-12:00 **New molecular mechanisms in diabetic neuropathy**
– Douglas Zochodne (Canada)

12:00-12:15 **Are Scwann cells critical for diabetic neuropathy?**
– Nadia Goncalves (Denmark)

12:15-13:15 **Lunch break**

SESSION 3: POSTER FLASH TALKS/POSTER VIEWING
Moderator: Reimar Thomsen (Denmark)

13:15-14:20 **Poster flash talks**
Group photo

14:20-15:15 **Poster viewing and coffee**

SESSION 4: EFFECTS OF EXERCISE IN DIABETES
Chair: Henning Andersen (Denmark)

15:15-15:45 **Anti-inflammatory effects of exercise in diabetes**
– Bente Klarlund Pedersen (Denmark)

15:45-16:15 **Exercise as therapy for diabetic neuropathy**
– John R. Singleton (USA)

16:15-16:30 **Effect of exercise in neurological disease**
– Ulrik Dalgas (Denmark)

17:00-17:30 **Pre-dinner walk & talk**

17:30-21:30 **Networking event/Dinner**

FRIDAY, 8 JUNE

08:00-08:55 **Registration and coffee**

SESSION 5: DIABETIC NEUROPATHY STAGING AND PROGRESSION
Chair: Eva Feldman (USA)

09:00-09:30 **Diabetic neuropathy staging**
– A. Gordon Smith (USA)

09:30-10:00 **The diabetic foot**
– Solomon Tesfaye (UK)

10:00-10:15 **Imaging in peripheral diabetic neuropathy**
– Henning Andersen (Denmark)

10:15-10:30 **The PiNS Study**
– Andreas Themistocleous (UK)

10:30-11:00 **Coffee break**

SESSION 6: AUTONOMIC NEUROPATHY AND HOW TO MEASURE IT
Chair: Nanna Finnerup (Denmark)

11:00-11:30 **Clinical and experimental assessment of autonomic neuropathy**
– Roy Freeman (USA)

11:30-12:00 **What is known/unknown about cardiac autonomic neuropathy**
– Rodica Pop-Busui (USA)

12:00-12:15 **Heart rate variability in diabetes**
– Jesper Fleischer (Denmark)

12:15-12:30 **Peripheral and central autonomic disturbances**
– Astrid Terkelsen (Denmark)

12:30-13:30 **Lunch break**

SESSION 7: STENO DIABETES CENTERS DENMARK: A NEW APPROACH IN RESEARCH TREATMENT AND PREVENTION OF DIABETES
Moderator: Troels.S. Jensen (Denmark)

13:30-13:50 **The Philosophy behind Steno Diabetes Centers**
– Jannik Hilsted (Denmark)

13:50-14:10 **The Steno Diabetes Centers in Denmark: From traditional structures to cross-sectorial collaboration**
– Allan Flyvbjerg (Denmark)

14:10-14:30 **Steno Diabetes Center Aarhus: Current status**
– Troels Krarup Hansen (Denmark)

14:30-15:00 **Panel debate**
– Jannik Hilsted, Allan Flyvbjerg, Troels Krarup Hansen

15:00-15:15 **Closing and goodbye**
– Troels S. Jensen (Denmark)

POSTER FLASH TALKS/ POSTER VIEWING

THURSDAY,7 JUNE, 2018, 13:15-15:20

Moderator: Reimar Thomsen (Denmark)

13:15-14:20 **Poster flash talks**
Suecia Room

14:20-15:15 **Poster viewing and coffee**
Foyer

FLASH TALKS
Poster presenters will give a brief verbal introduction (max. 3 minutes) of the data that they will later present at the poster viewing session. 'Questions and Answers' time right after the poster flash talk will not be provided. IDNC grants an award for the best poster flash talk.

TIME	POSTER NO.	PRESENTING AUTHOR
13:20-13:23	001	Reimar Thomsen
13:24-13:27	002	Karolina Snopek
13:28-13:31	005	Diana Christensen
13:32-13:35	006	Anete Dudele
13:36-13:39	007	Anne-Marie L. Wegeberg
13:40-13:43	008	Sandra S. Gylfadottir
13:44-13:47	009	Christian S. Hansen
13:48-13:51	011	Pall Karlsson
13:52-13:55	013	Mette W. Klinge
13:56-13:59	014	Alexander Kristensen
14:00-14:03	015	Anders Stouge
14:04-14:07	016	Simon Comerma-Steffensen
14:08-14:11	017	Signe T. Andersen
14:12-14:15	018	Thomas A. Aloysius
14:16-14:19	019	Peter L. Kristensen

Posters will display throughout the meeting. Please display your poster in the Foyer on arrival. The abstract number indicates your assigned poster board number. Poster presenters/co-author must stand next to their posters for informal discussions during the morning coffee breaks from 10:30 to 11:00 as well as during the designated poster session time on Thursday from 14:20 to 15:15.

POSTER NO.	PRESENTING AUTHOR	TITLE
Flash + paper 001	Reimar Thomsen	Prevalence and characteristics associated with microvascular complications at time of type 2 diabetes diagnosis: Danish Center for Strategic Research in Type 2 Diabetes (DD2) study
Flash + paper 002	Karolina Snopek	Symptoms of Diabetic Polyneuropathy are related to Falls in Patients with Type 2 Diabetes
Paper 004	Diana Christensen	The Danish Centre for Strategic Research in Type 2 Diabetes (DD2) Cohort: Presentation of 7,011 patients with new type 2 diabetes enrolled 2010-2016
Flash + paper 005	Diana Christensen	Using ICD-10 discharge diagnoses and prescription data to validly identify diabetic polyneuropathy and diabetic foot ulcers in Danish registries
Flash + paper 006	Anete Dudele	Effect of near nerve temperature on capillary transit time hetero geneity in sural nerve in mice
Flash + paper 007	Anne-Marie L. Wegeberg	Gastroparesis Cardinal Symptom Index is Associated with Gastrointestinal Tract pH, but not contractility or transit times, in Type 1 and Type 2 Diabetes
Flash + paper 008	Sandra S. Gylfadottir	Painful symptoms in diabetic neuropathy decreases quality of life in Danish type 2 diabetic patients
Flash + paper 009	Christian S. Hansen	Autonomic function is associated with future changes in glucose metabolism in non-diabetic individuals: the Whitehall II study
Flash + paper 011	Pall Karlsson	Corneal Confocal Microscopy in Screen-Detected Type 2 Diabetes: ADDITION-Denmark
Paper 012	Mette W. Klinge	Ambulatory assessment of gastrointestinal motility in patients with diabetes
Flash + paper 013	Mette W. Klinge	^{11}C Donepezil PET/CT for assessment of enteric neuropathy in diabetes
Flash + paper 014	Alexander Kristensen	Detection of early motor involvement in diabetic polyneuropathy using a novel MUNE method – MScanFit MUNE
Flash + paper 015	Anders Stouge	Composition and size of striated muscles in patients with type 2 diabetes with and without diabetic polyneuropathy – a Magnetic Resonance Imaging study
Flash + paper 016	Simon Comerma-Steffensen	Involvement of $K_{Ca}2.3$ Channels in Relaxation of Erectile Tissue is Altered in Type 2 Diabetic Mice
Flash + paper 017	Signe T. Andersen	Risk factors for the presence and the progression of cardiac autonomic neuropathy in type 2 diabetes; ADDITION-Denmark
Flash + paper 018	Thomas A. Aloysius	Chicken peptides in relation to obesity, diabetes, and inflammation: Assessment of different Chicken peptides and their effect in C57BL/6 mice
Flash + paper 019	Peter L. Kristensen	Prevalence of and risk factors for gustatory sweating amongst people with type 2 diabetes mellitus

NETWORKING EVENT AND DINNER

After the meeting all participants who have signed up for the networking event and dinner will walk together to Den Gamle By.

Date	Thursday, 7 June, 2018
Departure time	17:00 from the Hotel Foyer
Location	Den Gamle By.

17:30-18:45 **DEN GAMLE BY**
After a short introduction to Den Gamle By, which will take place at the Helsingør Theatre, you can experience the museum on your own. The buildings will be closed, but you can still take a walk around Den Gamle By and see the buildings and/or visit the exhibition the History of Aarhus.

18:45-21:30 **DINNER**
We meet at the Hobro House at approx. 18:45 for a welcome drink followed by the networking dinner.

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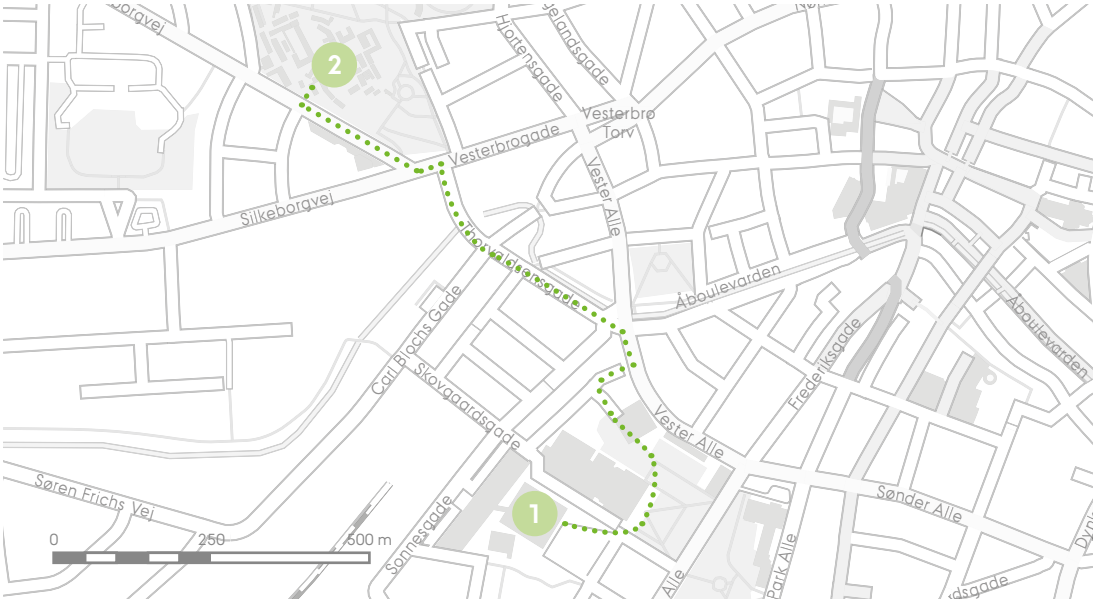

1 **VENUE**: Radisson Blu Scandinavia Hotel, Margrethepladsen 1

2 **EVENT**: Den Gamle By, Viborgvej 2

Photos: Den Gamle By

Den Gamle By

Photo: Lars Kruse, Aarhus University

ARoS, Museum of Modern Art

LIST OF SPEAKERS

(IN ALPHABETICAL ORDER)

A. Gordon Smith

A. Gordon Smith, MD, FAAN, is Professor and Chair of the Department of Neurology at Virginia Commonwealth University. Prior to this role, he served as Vice Chair for Research, Chief of the Division of Neuromuscular Medicine and Director of the Jack H. Petajan EMG Laboratory at the University of Utah. Smith is a graduate of the University of Virginia and the Mayo Medical School. He completed his neurology residency and a neuromuscular fellowship at the University of Michigan. Smith's NIH funded research team focuses on peripheral neuropathy in diabetes and obesity. He has a particular interest in biomarker development and novel clinical trial design in peripheral neuropathy. He was principal investigator of the Utah Regional site in the NINDS-funded Network for Excellence in Neuroscience Clinical Trials (NeuroNEXT), and sits on the NeuroNEXT Executive Committee. Dr. Smith is PI of the Topiramate as a Disease Modifying Therapy for Cryptogenic Sensory Peripheral Neuropathy (TopCSPN) trial being performed by NeuroNEXT. TopCSPN is the first large scale randomized trial of a treatment to alter the natural history of CSPN, which is one of the most common neurological disorders. He is also PI of the Activity for Diabetic Neuropathy (ADAPT) Trial. Dr. Smith has led or participated in numerous clinical trials in neuromuscular disorders including the Impaired Glucose Tolerance Neuropathy Study (IGTN) and the Utah Diabetic Neuropathy Study (UDNS). Smith serves on the Board of Directors of the American Academy of Neurology and Board of Trustees of the American Brain Foundation Board of Directors.

Allan Flyvbjerg

Allan Flyvbjerg graduated from Aarhus University, Denmark in 1986, defended his thesis (DMSc) in 1993, became specialist in Endocrinology and Internal Medicine in 1999, Chief Physician in 2001, Professor in Experimental Medical Research in 2005, Chair in Endocrinology in 2009 and Dean of the Faculty of Health, Aarhus University in 2011. In 2016 he was appointed CEO of Steno Diabetes Center Copenhagen (SDCC) and Professor in Clinical Endocrinology at the University of Copenhagen, Denmark. His research embraces pre-clinical and clinical research focused on the pathogenesis leading to - and the treatment of - diabetic angiopathy. He is author of >600 scientific publications and well-cited. He has contributed as editor and author to numerous Textbooks within Diabetes. Finally, he has receive numerous national and international awards for his scientific contribution. He was member of the editorial board of the Journal of the American Society of Nephrology from 2001–2004, and Associate Editor of Diabetologia from 2007–2013. Allan Flyvbjerg was President of the Danish Diabetes Association from 2000–2011 and is member of several national and international boards and councils. In the period 2016-2017 he was member of the 'Growth team for life-science' established by the Danish Government. Since 2018 he has been Chairman of Board of Directors in the Danish Diabetes Academy, a talent development program for future researcher and clinicians within diabetes.

Andreas Themistocleous

Andreas Themistocleous is a Clinical Research Fellow in Neurology and works under the supervision of Prof David Bennett within the Neural Injury Group, Nuffield Department of Clinical Neurosciences, University of Oxford. Andreas completed his undergraduate and postgraduate studies at the University of Witwatersrand, Johannesburg, South Africa before moving to Oxford in 2013. His research is focused on understanding the mechanisms of neuropathic pain. He uses a combination of techniques, such as threshold tracking, intraepidermal nerve fibre density assessment and quantitative sensory testing, to assess the functional and structural integrity of the peripheral nervous system. These techniques allow him to explore peripheral neuronal processing in patients suffering from peripheral neuropathy.

Astrid Terkelsen

Astrid Juhl Terkelsen, MD, PhD, Dr.MSci, Associate Professor, Aarhus University Hospital and Aarhus University, Denmark. Dr. Astrid Terkelsen graduated from the Medical School at Aarhus University in 2000 followed by internship. She became a board-qualified specialist in Neurology in 2014. She is at present a full-time neurologist at the Department of Neurology, Aarhus University Hospital, Denmark, where she is responsible for the Autonomic Laboratory with focus on neurogenic autonomic dysfunctions and small fibre neuropathy. Furthermore, she is affiliated with the Neuropathic Pain Clinic and the Neuromuscular Unit at the University Hospital. She has been affiliated with the Danish Pain Research Center, Aarhus University, since 1998. She obtained her PhD from Aarhus University in 2006 and her doctoral degree in 2016. She has published 29 original articles and three book chapters on autonomic measures, complex regional pain syndrome (CRPS) and small fibre neuropathy. Her research areas include a human model for CRPS and pathophysiology in CRPS. Her research has focused on methodological issues to increase the validity of various autonomic measures. Thus, in the Autonomic Laboratory it is possible to measure cardiovascular adrenergic and cardiovagal autonomic changes. The peripheral autonomic changes can be evaluated using the quantitative sudomotor axon reflex test, laser doppler flowmetry and cutaneous microdialysis measuring catecholamines. At present, her primary research focus is adrenergic dysfunction in autonomic disturbances. Link to publications: https://orcid.org/0000-0001-6708-1958.

Bente Klarlund

Bente Klarlund Pedersen, MD MDSc, is Professor of Integrative Medicine and a specialist in infectious diseases and internal medicine. She is the Director of 1) Centre of Inflammation and Metabolism (CIM) and 2) Centre for Physical Activity Research (CFAS) funded by TrygFonden. CIM and CFAS counts 16 senior researchers/postdocs, 15 PhD students, 15 other academic and technical personnel, 15 pre-graduate students and an administration of 4 persons (http://aktivsundhed.dk). She has supervised 41 PhD projects and been a mentor of 5 doctoral theses. The research group has identified skeletal muscle as an endocrine organ that produces and releases so-called "myokines". The identification of myokines provides a conceptual basis for understanding how muscles communicate with other organs. Through translational research, the aim is to develop targeted exercise training regimes for specific disease groups by applying a translational strategy: "from bedside to bench and back". BKP has had many positions of trust and is a member of the Royal Danish Academy of Sciences and Letters. BKP has more than 600 scientific publications and her "H"-index is 100 (Web of Science).

Brian Callaghan

I completed my undergraduate studies at the University of Michigan in 1999. Next, I graduated from the University of Pennsylvania Medical School in 2004. After an internship in Internal Medicine, I entered the Neurology residency program at the University of Pennsylvania. I then joined the University of Michigan as a clinical neuromuscular fellow in July of 2008, and then I completed a two-year research fellowship on a NIH T32 training grant. In July 2011, I became an Assistant Professor at the University of Michigan, and became the first Fovette E. Dush Early Career Professor of Neurology. During my time as an Assistant Professor, I have studied the metabolic factors that are associated with neuropathy. Early on, I conducted a systematic review of the effects of enhanced glucose control on the incidence of neuropathy in patients with type 1 and type 2 diabetes. Interestingly, we found that enhanced glucose control had a large effect on prevention of neuropathy in patients with type 1 diabetes, but did not have a statistically significant effect on patients with type 2 diabetes either in individual studies or in a meta-analysis. This prompted exploration of the difference between neuropathy in those with type 1 and type 2 diabetes, and led to us postulating metabolic syndrome components as potential metabolic drivers in type 2 diabetes. Since then, we have completed four observational studies (2 United States, Denmark, and China) that have demonstrated that hyperglycemia, obesity, and the number of metabolic syndrome components, but not hypertension or dyslipidemia are associated with neuropathy.

Daniel Witte

Daniel Witte is Danish Diabetes Academy Professor of Diabetes Epidemiology; based at the department of Public Health, Aarhus University in collaboration with the Department of Clinical Epidemiology at Aarhus University Hospital. Daniel started his career at the University of Utrecht, the Netherlands, where he studied medicine and completed a PhD in clinical epidemiology with a thesis focused on the non-invasive assessment of endothelial function in patients at high cardiovascular risk. He subsequently worked for 5 years as a post-doctoral clinical research fellow at the department of Epidemiology and Public Health, University College London, UK. His work there focused on screening models for type 2 diabetes and the analysis of risk factors for diabetes and its complications based on several large cohorts such as EURODIAB-Prospective Complications Study (Type 1 Diabetes) and the Whitehall II study (Type 2 Diabetes). Between 2008 and 2012 Daniel was head of the diabetes epidemiology group at Steno Diabetes Center in Gentofte, Denmark. In this position he further developed his main research interests: the study of the pathophysiological mechanisms which determine the transition from normal glucose control via pre-diabetes to diabetes and the early stages of its complications at the level of large populations. He has a special focus on longitudinal trajectory analyses and analysis of clustering of diabetic complications. His work uses data from several large longitudinal studies, such as the Inter99 and ADDITION trials, the ADDITION-PRO and Whitehall II cohorts, as well as routine medical and population registers. Daniel has a strong interest in the use of routine registers to enrich cohort datasets with a view to studying the long-term consequences of dysglycaemia, diabetes risk and the clinical management of diabetes. Between 2012 and 2014 Daniel was the Principal Investigator for the Luxembourg Cohort at the Luxembourg Institute of Health, Strassen, Luxembourg, tasked with the design and concept development for a national cohort in Luxembourg. In January 2015 he was appointed Professor of Diabetes Epidemiology at Aarhus University, Denmark; based on a grant from the Danish Diabetes Academy. He teaches at national and international courses at the post-graduate level and supervises several PhD students. Daniel is a member of the Danish Diabetes Academy and of the Steering Committees of the ADDITION-Denmark and ADDITION-Europe studies.

Douglas Zochodne

Dr. Douglas Zochodne is a Neurologist and Neuroscientist, Divisional Director of Neurology at the University of Alberta and Director of the Neuroscience and Mental Health Institute, UofA. His training included an MD and FRCPC in Neurology at the University of Western Ontario under the supervision of Dr. Charles Bolton and a research fellowship with Drs. Philip Low and Peter J. Dyck at Mayo Clinic. Dr. Zochodne's career has included faculty positions at Queen's University, Canada, the University of Calgary and more recently the University of Alberta. He has devoted his career toward understanding the biology and diseases of the peripheral nervous system. Dr. Zochodne's roles have included Editor-in-Chief of the Canadian Journal of Neurological Sciences (1999-2007), President of the Peripheral Nerve Society (2009-2011) and lead on the Regeneration Unit in Neurobiology, University of Calgary (2009-2014). He is a Fellow of the Canadian Academy of Health Sciences. Dr. Zochodne has channeled his research role as an investigator of diabetic polyneuropathy and peripheral neuron regeneration. His most recent work emphasizes intrinsic molecular mechanisms involved in adult sensory neuron plasticity including unexpected roles for tumour suppressor pathways repurposed within neurons. His laboratory has been funded continuously since 1988 and it has published over 270 research papers and chapters including four books. His work currently focuses on identifying new molecular mechanisms of peripheral neuron plasticity, regeneration and resistance to diabetes. His laboratory is currently funded by CIHR,CDA/DC, and ADI.

Eva Feldman

Eva Feldman is the Russell N. DeJong Professor of Neurology, Director of the A. Alfred Taubman Medical Research institute, Director of the University of Michigan Neuropathy Center, Past President of the Peripheral Nerve Society, Immediate Past President of the American Neurological Association, and serves as the Principal Investigator of the University of Michigan International Diabetic Neuropathy Consortium site. She is annually listed in Best Doctors of America and is the recipient of multiple awards for her role in clinical translational neuropathy research. She serves as the PI or Co-I on multiple basic and clinical studies aimed at understanding the pathogenesis of diabetic neuropathy, diagnosing this disorder and developing new treatments. Feldman is an established expert in the standardized protocols currently in use for characterizing neuropathy in mouse models of type 1 and type 2 diabetes and has led the neuropathy section of Diabetes Complications Consortium. Her basic investigations are paralleled with her clinical investigations into diabetic neuropathy, where she has completed similar phenotyping in man, and published novel results on the human metabolome and transcriptome in diabetic neuropathy. She also serves as the Lead Neuropathy Investigator on two NIH funded clinical trials, CACTI and SEARCH. She has received a lifetime achievement award from the American Diabetes Association for her studies on diabetic neuropathy.

Henning Andersen

Henning Andersen was born in 1963 and graduated from the Medical School at Aarhus University, Denmark, in 1991. In 1999 he was awarded a PhD degree and in 2000 he received a Doctorate Degree at Aarhus University. He has been a visiting scientist in Uppsala, Sweden and Charlottesville, VA. Dr. Andersen has been a consultant in Neurology since 2003 at the Department of Neurology, Aarhus University Hospital. In 2011 he was appointed Clinical Professor at Aarhus University and in 2015 he was elected Chair of Neurology at Aarhus University. For many years the main focus of his research has been neuromuscular disorders including peripheral neuropathies and myasthenia gravis.

Jannik Hilsted

Jannik Hilsted graduated as a Medical Doctor from the University of Copenhagen, Denmark in 1976, he defended his doctoral dissertation (Doctor of Medical Science) at the University of Copenhagen, Denmark in 1983 and received his Specialist certification in Internal Medicine and Endocrinology in 1988. He is currently positioned Chief Medical Officer, Head of Steno Grants, Novo Nordisk Foundation at Rigshospitalet, Copenhagen University Hospital in Denmark. His major Research Interest relates to the pathophysiology of diabetic neuropathy. He has published more than 200 international publications (H Index 42) and supervised three medical doctors who have obtained the Doctor of Medical Science degree and nine postgraduate students who have achieved the Ph.D. degree.

Jesper Fleischer

Jesper Fleischer, MSc PhD, is Associate professor at the Department of Clinical Medicine, Aarhus University, Denmark. He holds a MSc in Biomedical Engineering and a PhD in Medicine both from Aarhus University, Denmark. The cornerstone study of his PhD thesis was a Danish multi-center study focusing on large-scale screening of cardiovascular autonomic neuropathy using a Point-of-Care device, VAGUS, which he invented. His research and clinical interests encompass diabetes technology for early diagnosis and treatment of diabetes complications, with the main focus on neurological complications of diabetes, autonomic dysfunction, cardiovascular autonomic neuropathy and possible association to glycemic disorders including glycemic variability and hypoglycemia. Recently, he and colleagues showed that combining information of autonomic modulation and continuous glucose measurements enables prediction and improves detection of spontaneous hypoglycemic events. He has a strong interest in bridging the gap between clinical problems and technology development, using a combination of sensor technology and machine learning algorithms, to predict and/or perform early diagnosis of diabetes complications. Jesper has managed to be active in clinical research with more than 20 scientific publications in the last 4 years and has supervised PhD, MD, MSc and pre-graduate students, this despite that he is head of research, development and operations in private held medical technology company, reQbo.

J. Robinson Singleton

I am a neuromuscular specialist with expertise in clinical investigation of metabolic nerve and muscle disease. My research has focused on the relationship between early hyperglycemia, dyslipidemia, obesity and length dependent nerve injury. Our research group established an epidemiological link between otherwise idiopathic sensory predominant neuropathy and prediabetes, identified obesity and hypertriglyceridemia as risk factors for diabetic neuropathy independent of glucose control, and showed that small fiber aspects of prediabetic neuropathy improve with diet and exercise. We developed and validated a clinical exam scale for sensory predominant neuropathy, the Utah Early Neuropathy Scale, and showed that skin biopsy for intraepidermal nerve fiber density (IENFD) determination is a sensitive measure of the painful, small fiber predominant neuropathy that is among the most common early complications of type I diabetes. We have shown that reduced IENFD predicts progression to clinical neuropathy symptoms in patients with diabetes. We have adapted IENFD following capsaicin axotomy to demonstrate that metabolic syndrome inhibits cutaneous reinnervation, and that intensive diet counseling and observed exercise improves reinnervation rate in these patients. We have received NIH funds to study lifestyle intervention to prevent neuropathy in patients with diabetes. I have had continuous NIH funding for 15 years, and have used the Utah GCRC and CCTS as an Investigator since 2001. As Director of the Utah CCTS Clinical Services Core I oversee the 140 adult and pediatric human study protocols currently using the CSC. In this role I have developed a predictive and tracking instrument for nursing effort and protocol acuity, integrated the CCTS protocol and IRB applications, and expanded CCTS outreach to the Intermountain Health Care affiliated Primary Children's Medical Center and non-medical University departments that conduct human subject research.

Nadia Pereira Goncalves

Nadia Pereira Goncalves was trained in Veterinary Medicine at the University of Porto, Portugal, and completed her Ph.D. in the Institute for Molecular and Cell Biology at the same University (2015), investigating the role of neuroinflammation in the pathogenesis of a familiar form of amyloid peripheral polyneuropathy. Her main research interests are in the fields of nerve regeneration, peripheral neuropathies and neuroimmunology. Dr. Goncalves is currently a Postdoctoral Fellow at Aarhus University and has been a visiting scientist in the University of Melbourne (Australia) and University of Michigan (Ann Arbor, USA). Her main goal is to undertake further research on the role of Schwann cells for the pathophysiology of diabetic neuropathy.

Rodica Pop-Busui

Rodica Pop-Busui, MD, PhD, is a prominent diabetologist at Michigan Medicine and a recognized leader in the field of diabetes and diabetes complications. She is the Associate Chair for Clinical Research in the Department of Internal Medicine. Her research interests involve the chronic complications of diabetes, diabetic peripheral and cardiovascular autonomic neuropathy, nephropathy and cardiovascular disease, and novel technologies for treating type 1 diabetes. Dr. Pop-Busui has published over 150 peer-reviewed manuscripts and book chapters, and received awards from the Fulbright Foundation, the American Diabetes Association (ADA), and the University of Michigan. She has been an elected member of the Clinical Practice Committee of the ADA, and most recently was elected as the Chair of the ADA Scientific Research Review, and of Research Committee of the landmark DCCT/EDIC study. She has contributed er research efforts and leadership to some of the most important national and international diabetes clinical trials to date including: the NHLBI-funded Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial, the following NIDDK-funded trials: Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications, A Multicenter Clinical Trial of Allopurinol to Prevent GFR Loss in Type 1 Diabetes ("PERL), "Glycemia Reduction Approaches for Diabetes: A Comparative Effectiveness Study (GRADE), Bypass Angioplasty Revascularization Diabetes 2 (BARI- 2D), Targeting Inflammation with Salsalate for Type 2 Diabetes (TINSAL T2D), and many others funded by Pharma such as the Cardiovascular Safety of Insulin Degludec versus Insulin Glargine in Patients with Type 2 Diabetes at High Risk of Cardiovascular Events (DEVOTE).

Roy Freeman

Roy Freeman is Professor of Neurology at the Harvard Medical School and director of the Center for Autonomic and Peripheral Nerve Disorders in the Department of Neurology at Beth Israel Deaconess Medical Center in Boston, Massachusetts. His research and clinical interests are the physiology and pathophysiology of the small nerve fibers and the autonomic nervous system. His research encompasses the neurological complications of diabetes; neuropathic pain; the autonomic complications of Parkinson's disease and multiple system atrophy; biomarkers in neurodegenerative diseases; and the diagnosis and treatment of autonomic and peripheral nervous system disorders. He has a special interest in clinical trial design in neuropathic pain in diabetic peripheral neuropathy and other peripheral nerve disorders. He has been principal investigator on many neuropathic pain clinical trials. He is author of more than 200 original reports, chapters and reviews. He is the principal investigator on National Institutes of Health-funded studies on the neurological complications of diabetes and biomarker development in alpha-synucleinopathies. Dr. Freeman is former chairman of the World Federation of Neurology research group on the autonomic nervous system, former president of the American Autonomic Society and former chairman of the Autonomic Section of the American Academy of Neurology. He serves on the Executive Committee and the Steering Committee of the Analgesic, Anesthetic, and Addiction Clinical Trial Translations, Innovations, Opportunities, and Networks (ACTTION), a public-private partnership with the United States FDA. He is Editor-in-Chief of Autonomic Neuroscience: Basic and Clinical and on the editorial boards of The Clinical Journal of Pain, Pain: Clinical Updates and Clinical Autonomic Research.

Solomon Tesfaye

Solomon Tesfaye is a Consultant Physician/ Endocrinologist at Sheffield Teaching Hospitals and Honorary Professor of Diabetic Medicine at the University of Sheffield. His research projects include the epidemiology, risk factors, pathogenesis, central nervous system involvement and treatment of diabetic neuropathy and neuropathic pain. He has published a book, over 150 original articles, reviews and book chapters in the field of diabetic neuropathy including a landmark study in the New England Journal of Medicine. Professor Tesfaye was awarded the Prestigious Camillo Golgi Prize of the European Association for the Study of Diabetes (EASD) in 2014 for major scientific contributions in Diabetic Neuropathy. Professor Tesfaye has had international leadership roles including chairmanship of the International Expert Group on Diabetic Neuropathy that published 7 consensus recommendation papers in 2010/11, and of NEURODIAB (2006-9) which is the largest diabetic neuropathy scientific group in the world. He is also a member of the Science and Research Committee of Diabetes UK; a review panel member for the Medical Research Council, a Board Member of the Global Quantitative Sensation Testing Society; a member of the Advisory Council of the Neuropathy Trust and Secretary of International Insulin Foundation. He is also currently the Associate Editor of Experimental Diabetes Research, Frontiers in Endocrinology, European Endocrinology and Diabetes Management and was previous Associate Editor of Diabetologia. Professor Tesfaye has served as a member of the JDRF, NIDDK, and UK NIHR scientific review panels and as a member of a Diabetes & Neuropathic pain Review Group for the National Institute for Clinical Excellence (NICE).

Troels Krarup Hansen

Troels Krarup Hansen is CEO of Steno Diabetes Center Aarhus, Clinical Professor at the University of Aarhus, and President of the Danish Endocrine Society. Troels Krarup Hansen has a long-standing interest in diabetes complications, in particular related to the pathophysiological mechanisms underlying diabetic nephropathy. His research is directed towards identification of new markers for the development and maintenance of diabetic complications.

Ulrik Dalgas

Ulrik Dalgas, PhD, is an exercise physiologist specialized in clinical exercise physiology in neurological patients. In particular, most of his research focuses on the effects of exercise therapy in subjects with multiple sclerosis (MS), but also includes other neurological diseases such as stroke, Parkinson's disease, myasthenia gravis and diabetic neuropathy. His main research covers the effects of different exercise modalities (e.g. resistance training and endurance training) and the disease-modifying potential of exercise in neurological populations. Until 2017 he was board member and secretary for the Rehabilitation in Multiple Sclerosis (RIMS) organization and has served as principal investigator of several multicenter studies. Also, he is editorial board member of Multiple Sclerosis Journal. Finally, he has authored more than 80 scientific papers on the topic as well as several book chapters.

Photo: Ida Marie Jensen, Aarhus University

Aarhus harbour

POSTER ABSTRACTS

PREVALENCE AND CHARACTERISTICS ASSOCIATED WITH MICROVASCULAR COMPLICATIONS AT TIME OF TYPE 2 DIABETES DIAGNOSIS: DANISH CENTER FOR STRATEGIC RESEARCH IN TYPE 2 DIABETES (DD2) STUDY

Flash Talk/Poster
no. 001

Anne Gedebjerg[a], Thomas Peter Almdal[b], Klara Berencsi[a], Jørgen Rungby[c], Jens Steen Nielsen[d], Daniel Witte[e], Søren Friborg[f], Ivan Brandslund[g], Allan Vaag[h], Henning Beck-Nielsen[d], Henrik Toft Sørensen[a], **Reimar Wernich Thomsen[a]**

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BACKGROUND AND AIMS
To investigate prevalence and clinical characteristics of type 2 diabetes (T2D) patients who present with microvascular complications already at time of diagnosis.

METHODS
Cross-sectional baseline study using medical health databases to examine specific microvascular complications (retinopathy, neuropathy, and nephropathy) among newly diagnosed T2D patients enrolled in the Danish Center for Strategic Research in Type 2 Diabetes (DD2) cohort during 2010-2016. We calculated age- and gender-adjusted prevalence ratios (aPRs) of each complication using log-binomial regression.

RESULTS
Among 6958 recently diagnosed T2D patients, 20% (n=1385) had any hospital-diagnosed microvascular complication recorded; 12.8% (n=887) had diabetic retinopathy; 3.8% (n=264) had diabetic neuropathy; and 3.4% (n=234) had diabetic nephropathy. Compared with female T2D patients, males had clearly higher risk of neuropathy (aPR 1.67, 95% confidence interval (CI): 1.29-2.17) and nephropathy (aPR 1.71, 95% CI: 1.30-2.26), but not retinopathy (aPR 0.96, 95% CI: 0.85-1.09). Compared with tight glucose control (HbA1c<7.0%), increased HbA1c levels (e.g. 7%-8%) were associated with both retinopathy (aPR: 1.37, 95% CI: 1.15-1.64), neuropathy (aPR 1.52, 95% CI: 1.09-2.10), and nephropathy (aPR 1.59, 95% CI: 1.12-2.28). High BMI, central obesity, high C-peptide ≥800 pmol/L, and dyslipidemia were all associated with nephropathy, but not with the other microvascular complications. In contrast, low C-peptide <550 pmol/L and tobacco smoking predicted neuropathy, whereas high blood pressure and hypertriglyceridemia was associated with retinopathy.

CONCLUSIONS
One out of five patients with T2D had microvascular diabetes complications recorded already around time of diagnosis. Our findings suggest differences in underlying pathophysiological mechanisms behind specific microvascular complications

SYMPTOMS OF DIABETIC POLYNEUROPATHY ARE RELATED TO FALLS IN PATIENTS WITH TYPE 2 DIABETES

Flash Talk/Poster
no. 002

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OBJECTIVE
Distal sensorimotor polyneuropathy may cause impaired balance and unstable gait, which combined with decreased joint mobility and incoordination leads to an increased risk of falls. Falls may have serious consequences including decreased mobility, physical inactivity and higher morbidity and mortality.

MATERIALS AND METHODS
We performed a cross-sectional analysis of survey data on patients with type 2 diabetes included in the cohort established by the Danish Center for Strategic Research in Type 2 Diabetes (DD2) in 2011. Questionnaires were sent to 7,011 patients with type 2 diabetes.

RESULTS
We analysed data from 5,315 patients with type 2 diabetes that had answered questions concerning MNSI and fall frequency. Falls were reported in 17% (896) of patients during the past year, and 9% (505) had experienced 2 or more falls.

CONCLUSION
Cross-sectional data from this large national database show that patients with type 2 diabetes with 4 or more neuropathic symptoms have a 3-4 fold higher odds ratio of falls unrelated to alcohol consumption, smoking, physical activity, BMI, gender and age.

Poster no. 004

THE DANISH CENTRE FOR STRATEGIC RESEARCH IN TYPE 2 DIABETES (DD2) COHORT: PRESENTATION OF 7,011 PATIENTS WITH NEW TYPE 2 DIABETES ENROLLED 2010-2016.

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BACKGROUND AND AIMS
The Danish Centre for Strategic Research in Type 2 Diabetes (DD2) Cohort and biobank continuously enrolls newly diagnosed type 2 diabetes (T2D) patients throughout Denmark. We will describe the baseline characteristics.

METHODS
We linked primarily collected anthropometric and clinical data for each participant with data from the Danish Diabetes Database for Adults as well as with Danish registry data on comorbidities, drug prescriptions, and mortality.

RESULTS
By May 2016, 7,011 patients had been recruited from general practitioners (53%) and hospital specialist outpatient clinics (47%). At baseline, median age was 61 years (quartiles 52-68), and 58% were men. The median waist-to-hip ratio was 1.02/0.92 (men/women). Two-thirds did no sports during leisure time, and 53% reported a family history of diabetes. Additional data from the Danish Diabetes Database for Adults (currently obtained for 73% of the DD2 participants) showed that microalbuminuria was present in 16% of patients and macroalbuminuria in 2%. Tobacco smoking and alcohol overuse was similarly prevalent as in the general population. A proportion of 78% received oral antidiabetics only, and 7% received insulin (mono- or combination therapy), with 70% having HbA1c <7.0%. 72% received antihypertensives and 71% hypolipidemic treatment. At baseline, 22% and 15% had a hospital history of macrovascular and microvascular complications, respectively. Mortality in the cohort was low (212 deaths; mortality rate 1.12 per 1000 person-years; 1.36 in men and 0.81 in women).

CONCLUSIONS
The DD2 cohort is a large and data-rich T2D cohort that can serve as a platform for exhaustive T2D research.

USING ICD-10 DISCHARGE DIAGNOSES AND PRESCRIPTION DATA TO VALIDLY IDENTIFY DIABETIC POLYNEUROPATHY AND DIABETIC FOOT ULCERS IN DANISH REGISTRIES.

Flash Talk/Poster
no. 005

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BACKGROUND AND AIMS
Our understanding of risk factors and exact mechanisms behind diabetic polyneuropathy (DN) in patients with type 2 diabetes is lacking, thus hampering prevention and disease-modifying treatment. The use of linked Danish population-based medical registries is an ideal resource to study DN, but only if diagnoses are valid. We aim to determine the best way to identify DN and diabetic foot ulcers in Danish registries.

METHODS
Based on hospital diagnoses (Type 2 diabetes, polyneuropathy and ulcer) and prescription medication codes (neuropathic pain medication), we have defined 3 algorithms to identify type 2 diabetes patients with DN, painful DN or diabetic foot ulcers. Within each of these 3 groups, we will randomly select 60 patients from the Central Denmark Region, 2009-2017 and review their medical records. A pre-defined checklist of symptoms, signs and diagnostic test results described in the medical record will serve as the gold standard. We will calculate positive predictive values (PPVs) as the proportion of DN/painful DN/diabetic foot ulcer-patients that can be confirmed as truly having the diagnosis after medical record review. We will perform post-hoc analyses in order to identify the algorithm with the highest PPV.

RESULTS
Preliminary data for DN (N=24 patients) show promising but heterogeneous results. PPVs for presence of true DN were 30% for ICD-10 codes E10.4-E14.4 (diabetes with neurological complication, n=10), 80% for G62.9 (polyneuropathy unspecified, n=10), and 100% for G63.2 (diabetic polyneuropathy, n=4).

CONCLUSIONS
The best algorithm will enable comprehensive investigations of DN and diabetic foot ulcer incidence, risk factors and prognosis in Danish registries.

EFFECT OF NEAR NERVE TEMPERATURE ON CAPILLARY TRANSIT TIME HETEROGENEITY IN SURAL NERVE IN MICE

Flash Talk/Poster
no. 006

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BACKGROUND AND AIM

It is well known that temperature affects nerve conduction and function. This becomes especially important when performing neurophysiological measurements on rodents, due to their large surface to body weight ratio and resultant rapid heat loss under anesthesia. While it is reasonable to assume that temperature will influence not only nerve conduction and performance, but also nerve perfusion, data to support this in mice models is currently absent. Therefore, we aim to assess the role of near nerve temperature (NNT) on nerve perfusion in peripheral nerves in mice.

METHODS

All measurements will be performed on sural nerves of adult (12-15 weeks old) C57 male mice under isoflurane anesthesia. We will assess capillary function by using the state-of-the-art two-photon in vivo microscopy, by implementing bolus tracking method and measuring red blood cell velocities in sural nerve capillaries. These measurements will enable us to estimate capillary transit-time heterogeneity (CTH) – a parameter of crucial importance in tissue perfusion and oxygen availability. We will measure CTH and red blood cell velocities at different NNTs that will be adjusted using a custom-made leg holder with a heating element and a temperature probe implanted within four mm of the nerve at the imaging location.

RESULTS AND CONCLUSION

Our results will enable us to understand the importance of NNT during studies investigating nerve capillary function in mice.

GASTROPARESIS CARDINAL SYMPTOM INDEX IS ASSOCIATED WITH GASTROINTESTINAL TRACT PH, BUT NOT CONTRACTILITY OR TRANSIT TIMES, IN TYPE 1 AND TYPE 2 DIABETES

Flash Talk/Poster
no. 007

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BACKGROUND
Gastroparesis is an important complication of diabetes and symptom severity can be assessed using the validated gastroparesis cardinal symptoms index (GCSI) score with subscale bloating, postprandial fullness and nausea/vomiting. The wireless motility capsule (WMC) is a minimally invasive technique that investigates temperature, pH and pressure throughout the gastrointestinal tract. We aimed to investigate the co-relationships between GCSI and measures derived from the WMC in people with diabetes.

METHODS AND MATERIALS
A cohort of 87 people with diabetes (68 type 1, 45 male, mean age 48 years and 19 type 2, 10 male, mean age 63 years) filled out GCSI and underwent a standardized WMC test.

RESULTS
GCSI score was positively associated with gastric mean and median pH (r=0.2584, p=0.03 and r=0.30, p=0.01, respectively). GCSI score was negative associated with change in pH across ileo-caecal junction (r=0.47, p<0.00001). No association was found between GCSI score and mean or median pH in the small bowel and large bowel or with the rise in pH across the antro-duodenal sphincter, neither with regional transit times or contractility measures. Similar associations were seen with GCSI. Multivariate regression between GCSI and pH, controlling for gender, type of diabetes and use of PPI, demonstrated that gender and type were confounding factors.

CONCLUSION
In this cohort, GCSI is associated with the regional pH within the gastrointestinal tract but not with transit times or contractility measures. These results suggest that pH may be important in the genesis of symptoms as measured by the GCSI.

PAINFUL SYMPTOMS IN DIABETIC NEUROPATHY DECREASES QUALITY OF LIFE IN DANISH TYPE 2 DIABETIC PATIENTS

Flash Talk/Poster
no. 008

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BACKGROUND AND AIMS
Painful diabetic polyneuropathy (PDPN) is a disabling complication of diabetes. This study aims to determine its prevalence and relationship with Quality of Life (QoL) in a nationwide prospective cohort of Danish type 2 diabetic patients.

METHODS
We sent a detailed questionnaire on neuropathy, pain and QoL to 6,726 patients prospectively enrolled from general practitioners and hospital specialist outpatient clinics into the Danish Centre for Strategic Research in Type 2 Diabetes (DD2) cohort. Patients who reported pain in both feet and a score ≥3 on the Douleur Neuropathique (DN4) questionnaire were considered to have possible painful DPN. QoL and pain intensity were measured on a numeric rating scale (NRS, 0-10). The Michigan Neuropathy Screening Instrument (MNSI) was used to assess neuropathy.

RESULTS
A total of 5,513 (82.0 %) returned a complete questionnaire. Of the 5,372 patients with valid answers on the question on pain in both feet who completed the DN4 questionnaire, 536 (10%) had a DN4 score ≥ 3 and pain in both feet. Mean pain intensity was 5.2 (SD 2.2). Patients with possible PDPN had a substantially lower QoL score than those without PDPN (median QoL score 6 versus 8 (p 0.001)), also when correcting for MNSI score.

CONCLUSION
Ten percent of newly diagnosed type 2 diabetic patients in Denmark had possible PDPN. Patients with possible PDPN had lower QoL than patients without.

AUTONOMIC FUNCTION IS ASSOCIATED WITH FUTURE CHANGES IN GLUCOSE METABOLISM IN NON-DIABETIC INDIVIDUALS: THE WHITEHALL II STUDY

Flash Talk/Poster
no. 009

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BACKGROUND AND AIMS
Deterioration in autonomic nervous system function is associated with adverse changes in glucose metabolism in patients with prediabetes and diabetes. The temporal nature of this association is not investigated in non-diabetic individuals. We investigated autonomic function (AF) and 5-year changes in glucose metabolism in individuals without diabetes.

METHODS
The analysis is based on up to 7,421 person-examinations for 3,104 study participants of the Whitehall II cohort. Measures of AF included 2-minute resting heart rate (rHR) and six heart rate variability (HRV) indices. Associations between baseline AF measures and subsequent 5-year changes in fasting and 2-hour glucose and insulin concentrations, insulin sensitivity (HOMA-IS and ISI_{0-120}) and beta-cell function (HOMA-) were estimated using mixed-effects models adjusting for baseline measures of the metabolic outcome, age, sex, ethnicity (Model 1) and subsequently also for body mass index (BMI), metabolic covariates and medication (Model 2).

RESULTS
In the fully adjusted models a doubling in the HRV index RMSSD was associated with a 5-year decrease in HOMA-β of -2.2% (95%CI -4.2;0.0, p=0.046), and a doubling of the LF/HF ratio was associated with an increase in HOMA-β of 2.3% (95%CI 0.6-4.1, p=0.009). A doubling of LF power and LF/HF ratio was associated with 5 year change in fasting serum insulin of -1.1%(95%CI -2.2;0.1, p=0.039) and 1.9%(95%CI 0.2;3.6, p=0.029), respectively. A doubling of the HRV index SDNN was associated with a decrease in 2-hour insulin concentration of -4.2% (95%CI -8.1;-0.1, p=0.045). rHR remained significantly associated with all measures of insulin resistance, beta-cell function and insulin in the fully adjusted model

CONCLUSIONS
More beneficial levels of AF measures were associated with improved insulin sensitivity and recuded beta-cell function and lower serum insulin concentrations in non-diabetic individuals. Autonomic dysfunction may be a novel risk marker for diabetes and could be a future target for intervention.

CORNEAL CONFOCAL MICROSCOPY IN SCREEN-DETECTED TYPE 2 DIABETES: ADDITION-DENMARK

Flash Talk/Poster no. 011

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BACKGROUND AND AIMS

We aimed to compare corneal confocal microscopy (CCM) measures between participants with type 2 diabetes with and without confirmed diabetic polyneuropathy (DPN) and controls without diabetes.

METHODS

CCM, nerve conduction studies, and assessment of symptoms and deficits of DPN were undertaken in 144 participants with type 2 diabetes and 25 controls without diabetes. DPN was defined according to the Toronto criteria for confirmed DPN. We used ANOVAs to compare CCM measures between groups and regression analyses to determine clinical variables associated with CCM measures.

RESULTS

Corneal nerve fiber density (CNFD) was lower in participants with confirmed DPN (n = 27) (P = 0.04) and without confirmed DPN (n = 117) (P = 0.01) compared with controls. No difference between participants with and without confirmed DPN was observed (P = 0.98). There were no differences in corneal nerve fiber length and corneal nerve branch density between participants with and without confirmed DPN P = 0.06 and P = 0.29, respectively). CNFD was associated with age (b -0.150 [95% CI 0.297; 0.003]), height (b 0.154 [95% CI 0.049; 0.260]), total cholesterol (b 1.090 [95% CI 0.067; 2.112]), and LDL cholesterol (b 1.358 [95% CI 0.022; 2.694]). The effect of age on CNFD remained stable in multiple linear regressions including DPN status, sex, and diabetes duration.

CONCLUSIONS

This study supports CNFD being lower in participants with type 2 diabetes compared with controls. CCM measures were unable to differentiate between participants with type 2 diabetes with and without confirmed DPN.

AMBULATORY ASSESSMENT OF GASTROINTESTINAL MOTILITY IN PATIENTS WITH DIABETES

Poster
no. 012

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BACKGROUND/AIM
Gastrointestinal (GI) symptoms are common among patients with diabetes mellitus (DM). Diabetic gastroparesis has been studied in detail, but data on the rest of the GI tract is scarce. A novel, ambulatory, wireless capsule system, Motilis 3D-Transit, allows assessment of transit patterns throughout the whole GI tract in one single examination.

METHODS
We studied 11 DM patients referred for GI symptoms and 15 healthy controls by means of 3D-Transit.

RESULTS
All subjects were studied without complications. Median transit time through the entire GI tract was 73 hours (range 36 to 440) in DM patients vs 30 hours (range 14 to 71) in healthy ($p=0.01$). Median gastric emptying time was 3.4 hours (range 2.4 to 38.9) vs 2.4 (range 0.6 to 3.3) ($p<0.01$), median small intestinal transit time was 5.1 hours (range 2.5 to 9.1) vs 4.7 (range 1.4 to 7.6) ($p=0.78$), and median colorectal transit time was 48.5 hours (range 14.6 to 431.4) vs 24.7 (range 7.2 to 61.2) ($p=0.11$).

CONCLUSION
3D-Transit allows safe, detailed and ambulatory assessment of GI transit in patients with DM. In many patients with DM and GI symptoms, prolonged transit is not restricted to the stomach.

^{11}C DONEPEZIL PET/CT FOR ASSESSMENT OF ENTERIC NEUROPATHY IN DIABETES

Flash Talk/Poster
no. 013

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BACKGROUNDS AND AIMS
Gastrointestinal symptoms are common and often severe in patients with diabetes. Furthermore, gastrointestinal dysfunction can compromise absorption of food and drugs. Unfortunately, knowledge of diabetic enteric neuropathy is sparse. Gastrointestinal contractions are mainly stimulated by cholinergic enteric nerve cells, which are located between the muscle layers deep in the bowel wall. The enteric nerve cells are difficult to access and cardiac autonomic neuropathy is widely used as a surrogate of autonomic neuropathy of the bowel. Unfortunately, cardiac neuropathy is not correlated to gastrointestinal symptoms and other methods for diagnosis of diabetic enteric neuropathy are needed.

METHODS
^{11}C Donepezil is a novel PET-tracer binding the acetylcholinesterase. A ^{11}C-Donepezil PET/CT scan quantifies the density of acetylcholinesterase in visceral organs. Preliminary data from 7 diabetes patients with gastrointestinal symptoms and 7 healthy controls are available.

RESULTS
Compared to healthy controls, the ^{11}C Donepezil signal in colon and the small intestine was lower in diabetes patients. Also, a trend towards larger volume in colon and the small intestine was shown.

CONCLUSIONS
^{11}C Donepezil is a promising method for objective assessment of diabetic enteric neuropathy.

DETECTION OF EARLY MOTOR INVOLVEMENT IN DIABETIC POLYNEUROPATHY USING A NOVEL MUNE METHOD – MSCANFIT MUNE

Flash Talk/Poster
no. 014

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BACKGROUND AND AIMS
Detection of motor involvement in diabetic polyneuropathy (DPN) with nerve conduction studies (NCS) is delayed by collateral sprouting. Our aim is to examine whether a novel Motor Unit Number Estimation (MUNE) method so called MScanFit MUNE (MScan) can detect early motor involvement in DPN.

METHODS
We prospectively included 45 patients with diabetes mellitus type II and 39 healthy subjects (HS). NCS of three motor (median, peroneal, tibial) and three sensory (bilateral sural and median) nerves and MScan in abductor pollicis brevis (APB) muscle were done in all participants. NCS results were compared to laboratory controls. All participants with carpal tunnel syndrome were excluded.

RESULTS
DPN diagnosis was given using Dyck's criteria requiring NCS abnormality in at least two nerves. Twelve patients (26.6%) had DPN (DPN+) and 33 patients had normal NCS (DPN-).
DPN+ patients had a significantly lower number of motor units (78.58 ± 8.01) when comparing with both HS (116.7 ± 4.97) (p=0.00048) and DPN- patients (113.4 ± 6.44) (p=0.005). Motor unit size was significantly larger in DPN+ patients (6.343 ± 0.658) compared to HS (4.254 ± 0.301, p= 0.00252) and DPN- patients (3.84 (3.25, 5.17), p=0.01384). No difference between HS and DPN- patients in motor unit number (p=0.68375) or size (p=0.53793).
Compound muscle action potential (CMAP) amplitude was within normal ranges for all DPN+ patients.

CONCLUSIONS
MScan abnormality with normal CMAP amplitude in APB muscle suggests that MScan may be a sensitive measure of early motor involvement in DPN. Further studies with application of MScan in lower extremity are needed.

COMPOSITION AND SIZE OF STRIATED MUSCLES IN PATIENTS WITH TYPE 2 DIABETES WITH AND WITHOUT DIABETIC POLYNEUROPATHY – A MAGNETIC RESONANCE IMAGING STUDY

Flash Talk/Poster
no. 015

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OBJECTIVE
The aim of the study is to assess the quality and quantity of striated muscles in patients with type 2-diabetes, in relation to the presence of diabetic polyneuropathy (DPN) using MRI-techniques.

METHODS
Presence of neuropathy is determined based on clinical examinations, and nerve con-duction studies. Muscle strength of the extensors and flexors at the knee and ankle joint is deter-mined by isokinetic dynamometry at the non-dominant leg. Muscles are visualized by a novel multi-acquisition MRI-protocol: Dixon, T2-mapping, and Diffusion Weighted Imaging.

RESULTS
Here we present the preliminary results of 15 DPN, 15 non-DPN, and 15 healthy fully processed subjects. The groups were matched on age, sex, and height. The groups were not matched on BMI. Regression-analysis of maximal muscle strength, with the explanatory variables age, gender and BMI, showed a reduction in muscle strength in the DPN-group. Muscle strength described as a percentage of healthy-subjects: dorsal-flexors(DF)(80%, p=0.01), plantar-flex-ors(PF)(71%, p<0.01), knee-extensors(KE)(82%, p=0.01), and knee-flexors(KF)(81%, p=0.02). Intrinsic muscle-strength was decreased in plantar-flexors of both diabetes groups compared to healthy subjects (p=0.03). MRI measures of DPN-patients as compared to non-DPN, and healthy subjects: Fat-fraction was significantly increased in all muscle-groups. T2muscle was prolonged in knee-extensors and plantar-flexors (KE,p<0.01, PF,p=0.01). FA was significantly increased in the dorsal and plantar-flexors of the most severely affected DPN-patients (DF:p=0.02, PF:p<0.01).

CONCLUSION
MRI is able to identify muscle-changes in relation to muscle-dysfunction in DPN diabetes patients. T2muscle proved to be a sensitive measure, able to detect muscle-changes even in mildly affected DPN-patients. Further studies on more severely affected DPN-subjects are needed to fully understand the potential of MRI-measures in muscle-dysfunction.

INVOLVEMENT OF $K_{Ca}2.3$ CHANNELS IN RELAXATION OF ERECTILE TISSUE IS ALTERED IN TYPE 2 DIABETIC MICE

Flash Talk/Poster
no. 016

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OBJECTIVE
Activation of endothelial small conductance calcium-activated K^+ channels ($K_{Ca}2.3$/SK3) and intermediate conductance calcium-activated K^+ channels (IK/$K_{Ca}3.1$) leads to vascular relaxation. Our previous studies have shown that $K_{Ca}2.3$ down-regulation diminishes erectile function. In the present study we hypothesized that $K_{Ca}2.3$ channel function is altered in erectile tissue of type 2 diabetic animals.

METHODS
Erectile function measured in diabetic db/db or heterozygous control mice were compared. Corpus cavernosum strips were mounted for isometric tension recording, and they were processed for qPCR/Western Blot.

RESULTS
In anesthetized diabetic db/db mice erectile function was diminished compared to control animals. Concentration-dependent contractions to noradrenaline were increased in corpus cavernosum strips from db/db compared to control mice. Apamin, a blocker of $K_{Ca}2$ channels inhibited acetylcholine relaxation in corpus cavernosum from db/db and control animals, being less effective in db/db compared to control animals. NS309 (0.5 µM), an activator of $K_{Ca}2$ and $K_{Ca}3.1$ channels, potentiated concentration-response curves for acetylcholine in corpus cavernosum from control, but not in corpus cavernosum from db/db mice. SNP relaxation was similar in corpus cavernosum from db/db and control animals. $K_{Ca}2.3$ gene and protein expression was increased in corpus cavernosum from db/db mice, nevertheless protein expressed in aorta was diminished.

CONCLUSION
Our results suggest that in type 2 diabetes $K_{Ca}2.3$ channel expression is down-regulated in the systemic circulation, and up-regulated in erectile tissue. In erectile tissue noradrenaline contraction is increased and the effect of NS309 reduced which may be related to impaired $K_{Ca}2.3$ channel function. These changes may contribute to impairment of erectile function in diabetes.

RISK FACTORS FOR THE PRESENCE AND THE PROGRESSION OF CARDIAC AUTONOMIC NEUROPATHY IN TYPE 2 DIABETES; ADDITION-DENMARK

Flash Talk/Poster
no. 017

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BACKGROUND AND AIMS
We will examine the association of cardiometabolic risk factors with the presence and progression of cardiac autonomic neuropathy (CAN) in patients with type 2 diabetes.

METHODS
CAN was assessed along with cardiometabolic risk factors in a subset of participants from ADDITION-Denmark at the 6- and 13-year follow-up of this cohort (in 777 and 452 participants, respectively). 306 participants had CAN measures at both time-points. CAN was assessed by cardiovascular autonomic reflex tests (CARTs) (R-R responses to lying-to-standing, deep breathing and the Valsalva maneuver) and by two minute resting heart rate variability (HRV) by the Vagus® device. We will examine the presence, the progression and the cumulative incidence of CAN between these two examinations. Cardiometabolic risk factors associated with the levels of CARTs, HRV indices and the changes of these levels over time will be estimated using linear mixed models adjusting for sex, age and time-interval between examinations.

RESULTS
Preliminary results showed a prevalence of early CAN of 20% and 27% at first and the second examination, respectively. Corresponding values were 9% and 16% respectively for manifest CAN. The cumulative incidence of early or manifest CAN was 38% during 6.5 years in the sub-group with CAN measures at both time-points and negative in CAN at the first examination.

CONCLUSIONS
Preliminary results showed CAN to be largely present and progress substantially over 6.5 years in this cohort with type 2 diabetes. We will present cardiometabolic risk factors associated with the presence and progression of CAN at the IDNC meeting.

CHICKEN PEPTIDES IN RELATION TO OBESITY, DIABETES, AND INFLAMMATION: ASSESSMENT OF DIFFERENT CHICKEN PEPTIDES AND THEIR EFFECT IN C57BL/6 MICE

Flash Talk/Poster
no. 018

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BACKGROUND AND AIMS
Recent studies have shown that the dietary source of protein can affect energy metabolism, and hydrolyzed peptides could have potent effects on metabolic diseases including diabetes. We have generated several chicken peptides (CP) from chicken by-products through specific enzymatic procedures. This study aimed to investigate the impact of these different CP in a mouse obesity model.

METHODS
Male C57BL/6 mice were fed a high-fat control diet (20% w/w casein) or four different CP diets (10% w/w CP and 10% casein) for 12 weeks (n=12). Body weight and food intake was measured throughout the study. The glucose tolerance after an intraperitoneal injection of glucose was measured in fasting mice at baseline and after 11 weeks. At week 12, mice were sacrificed, and blood samples were collected and adipose tissue mass determined. Plasma cytokines were determined by multiplex analysis.

RESULTS
Eleven weeks of high-fat feeding significantly increased fasting blood glucose levels in the control group compared to baseline (1.65 fold, p=0.009), and the glucose area under the curve after an intraperitoneal glucose injection was also increased (1.3 fold increase, p=0.071). The CP diets did not influence glucose tolerance and mice developed insulin resistance. The body weight and adipose tissue depots increased similarly in the CP groups compared to control, and the feed intake was comparable. High plasma cytokine levels are a sign of systemic inflammation in obesity. A significant reduction in IL-1 , IFN , TNF and MCP-1 was observed in all CP groups compared to control, which indicated the anti-inflammatory effect of all tested CP. In addition, CP diet group 4 and 5 significantly reduced plasma levels of IL- , IL-2, IL-6 and GM-CSF compared to control.

CONCLUSIONS
Overall, our data show that these peptides from chicken were not able to counteract obesity and glucose intolerance in a mouse obesity model, but strongly reduced inflammatory parameters associated with obesity.

PREVALENCE OF AND RISK FACTORS FOR GUSTATORY SWEATING AMONGST PEOPLE WITH TYPE 2 DIABETES MELLITUS

Flash Talk/Poster
no. 019

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BACKGROUND AND AIMS
Gustatory Sweating (GS) is known as a complication to diabetes mellitus (DM) and is characterised by profuse sweating during or immediately after ingestion of food. Most reports on gustatory sweating have been case reports suggesting it to be a rare late diabetic complication. The aim of this study was to determine the prevalence of gustatory sweating in an unselected cohort of patients with type 2 diabetes. To generate hypothesis on the pathophysiology of GS, associations between GS and classic complications of DM were explored.

METHODS
In a cross-sectional study all people with type 2 DM in the DM outpatient clinic at Nordsjællands Hospital, Denmark, received a questionnaire by mail with eight questions regarding GS. Answers were paired with medical data from the electronic patient records. Prevalence of GS was primary endpoint. Association between GS and complications to DM were secondary endpoints. Univariate and multivariate logistic regression analyses were performed including the following variables: Sex, age, HbA1c, albuminuria, retinopathy and neuropathy. Variables associated with or nearly associated with GS in the univariate analyses (defined by $p<0.15$) were included in the multivariate analysis.

RESULTS
Out of 991 persons receiving the questionnaire, 510 people answered, four were excluded, leaving a response rate at 51%. 22% of the patients sweat in relation to ingestion of food, and 13% (95% CI: 10-16%) also in relation to non-spicy foods. In the multivariate logistic regression analysis, we found that decreasing age ($p=0.007$) was associated with increasing probability of GS. Also presence of severe peripheral neuropathy (threshold of biothesiometry > 50 V) was associated with GS ($p=0.01$).

CONCLUSIONS
A prevalence of gustatory sweating of 13% was found in a large unselected cohort of people with type 2 DM. Gustatory sweating was associated with lower age and severe peripheral neuropathy.

Photo: Lars Kruse, Aarhus University

The Latin Quarter, Aarhus

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