

### **Danish Society for Gastroenterology and Hepatology**

9. årsmøde

3-4 september 2021

på

### **Hotel Comwell Kolding**

Skovbrynet 1, 6000 Kolding



## Indholdsfortegnelse

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| Fredag 3. september 2021 |  |  |  |
|--------------------------|--|--|--|
| 09.30 - 10.00            | Registrering og kaffe  |  |  |
| 10.00 - 10.15            | Velkomst   |  |  |
| 10.00 - 10.13            | DSGHs formand Ebbe Langholz  |  |  |
|                          | Legater  |  |  |
| 10.15 - 10.30            | Sidste års modtager (Gorm Roager Madsen) fortæller, hvad legatet har været brugt |  |  |
| 10.12 10.20              | til  |  |  |
|                          | Afsløring af de nye legatmodtagere   |  |  |
|                          | Autoimmun hepatitis – Fra epidemiologi til behandlingsrefraktær autoimmun        |  |  |
| 10.30 - 11.05            | hepatitis  |  |  |
|                          | Henriette Ytting /Lisbet Grønbæk   |  |  |
|                          | Chair: Mette Munk Lauridsen  |  |  |
| 11.05 - 11.35            | Tværfaglig behandling af inficeret walled-off-necrosis ved nekrotiserende        |  |  |
|                          | pancreatitis   |  |  |
|                          | Camilla Nøjgaard, John Gásdal Karstensen og Morten Laksáfoss Lauritsen           |  |  |
|                          | Chair: Ove Schaffalitzky De Muckadell  |  |  |
| 11.25 12.05              | Palliation til patienter med fremskreden leversygdom                             |  |  |
| 11.35 – 12.05            | Ane Teisner, Mette Munk Lauridsen  |  |  |
|                          | Chair: Peter Jepsen  |  |  |
| 12.10 - 12.55            | ePosterpræsentation (talk-no-walk á 2 min)                                       |  |  |
|                          | Chairs: Signe Wiese & Anders Bergh Lødrup  |  |  |
| 12.55 - 13.55            | Frokost og besøg på udstilling   |  |  |
|                          | Udenlandsk foredragsholder (virtuel)   |  |  |
| 13.55 - 14.40            | Janneke van der Woude, Professor fra Erasmus Medical Centre, Rotterdam           |  |  |
| 13.33 - 14.40            | Stratifying IBD therapy  |  |  |
|                          | Chairs: Jakob Seidelin   |  |  |
| 14.45 - 15.30            | ePosterpræsentation (talk-no-walk á 2 min)                                       |  |  |
| 14.43 - 13.30            | Chair: Lotte Fynne & Bjørn Stær Madsen   |  |  |
| 15.30 – 16.15            | Kaffe og besøg på udstilling   |  |  |
| 16.15 - 17.45            | Foredragskonkurrence   |  |  |
| 10.13 - 17.43            | Chairs: Henning Grønbæk & Maja Thiele  |  |  |
| 17.45 - 18.00            | DSGH-generalforsamling   |  |  |
| 19.00                    | Festmiddag med Festtale og partybandet Sustain                                   |  |  |

|               | Lørdag 4. september 2021   |  |  |
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| 9.15 - 9.45   | YDSGHs corner  Uddannelsesmiljø i Hepato/Gastroenterologi: Er vi gode nok?  Emilie Dahl, Fredrik Bergenheim og Jonathan Yde  Chair: Nina Kimer |  |  |
| 9.45 - 10.10  | Guideline opdateringer  Eosinofil Esophagitis v. Anne Lund Krarup  Alkoholisk Hepatitis v. Henning Grønbæk  Chair: Lise Lotte Gluud            |  |  |
| 10.10 – 10.25 | Orientering om ny national IBD kvalitetsdatabase under RKKP  Lone Larsen  Chair: Peter Holland-Fischer   |  |  |
| 10.25 - 10.55 | Kaffe og Pause   |  |  |
| 10.55 - 11.35 | IBD-Cases  2 cases v. Linda Kievit & Jacob Bjerrum  Panel: Jens Kelsen, Jan Fallingborg, Inge Nordgaard Lassen  Chair: Jakob Seidelin          |  |  |
| 11.35 – 11.45 | Afslutning  DSGHs formand Ebbe Langholz  |  |  |
| 11.45 – 12.15 | Sandwich to go   |  |  |



Abstract oversigt

|     | FOREDRAG                    |  |  |
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| 5   | Konstantin<br>Kazankov      | CIRRHOCARE® - A PILOT STUDY OF DIGITAL HOME MANAGEMENT TO DETERMINE FEASIBILITY AND UTILITY TO DIAGNOSE NEW DECOMPENSATION EVENTS IN ADVANCED CIRRHOSIS  |  |
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#### **Abstracts 2021**

#### Foredrag - Abstract nummer 1-7:

1)
COLLAGEN PROPORTIONATE AREA PREDICTS LONG-TERM MORTALITY IN PATIENTS WITH ALCOHOLIC HEPATITIS

Mads Israelsen<sup>1,2</sup>, Marta Guerrero Misas<sup>2</sup>, Anastasios Koutsoumourakis<sup>2</sup>, Andrew Hall<sup>3</sup>, Claudia Covelli<sup>4</sup>, Elena Buzzetti<sup>2</sup>, Laura Iogna Prat<sup>2</sup>, Davide Roccarina<sup>2</sup>, Tu Vinh Luong<sup>3</sup>, Alberto Quaglia<sup>3</sup>, Massimo Pinzani<sup>2</sup>, Emmanuel A. Tsochatzis<sup>2</sup>

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**Background & Aims**: There are several short-term prognostic scores for alcoholic hepatitis (AH) that combine demographical and biochemical parameters. The extent of liver fibrosis may also be relevant to the prognosis of AH with potential added value. We evaluated collagen proportionate area (CPA) as a predictor of short and long-term mortality in AH.

**Methods:** We retrospectively included patients with biopsy-verified AH. Clinical, laboratory and outcome data were collected. CPA and five AH scores were calculated: Maddrey's DF, MELD, GAHS, ABIC, and the Lille Model. Predictors of short and long-term all-cause mortality were assessed using Cox regression analysis.

**Results**: We included 140 patients with AH. In total, 67 (48%) patients died after a median follow-up of 66 (IQR 102) months, with 17 (12%) dying within the first 90-days. CPA was not a predictor of 90-days mortality and had no additional value to the prognostic AH scores on short-term mortality. However, CPA predicted long-term mortality independently of prognostic AH scores. Importantly, CPA and abstinence from alcohol were independent predictors of long-term mortality in patients alive 90 days after the biopsy.

**Conclusion**: CPA predicts long-term mortality in patients with AH independently of abstinence from alcohol but has no prognostic value on short-term mortality.

## HEMODYNAMICS OF THE LIVER, HEART AND KIDNEY FROM COMPENSATED CIRRHOSIS TO HEPATORENAL SYNDROME: A MAGNETIC RESONANCE STUDY

Karen Vagner Danielsen<sup>1,2</sup>, Signe Wiese<sup>1</sup>, Troels Busk<sup>3</sup>, Puria Nabilou<sup>1</sup>, Thit Kronborg<sup>1</sup>, Claus Leth Petersen<sup>2</sup>, Jens Dahlgaard Hove<sup>2,4,5</sup>, Søren Møller<sup>2,5\*</sup>, and Flemming Bendtsen<sup>1,5\*</sup>

<sup>1</sup>Centre of Gastroenterology, Medical division, Copenhagen University Hospital Hvidovre; <sup>2</sup>Dept. Clinical Physiology and Nuclear Medicine, Centre of Functional and Diagnostic Imaging and Research, Copenhagen University Hospital Hvidovre; <sup>3</sup>Centre of Gastroenterology, Medical division, Copenhagen University Hospital Herlev; <sup>4</sup>Dept. of Cardiology, Copenhagen University Hospital Hvidovre; <sup>5</sup>Institute of Clinical Medicine, Faculty of Health Sciences, University of Copenhagen

\*) Søren Møller and Flemming Bendtsen are joint senior authors

**Background and aims:** The relationship between portal pressure, hemodynamic disturbances and progressive decompensation in patients with cirrhosis is complex and not yet fully understood. In this magnetic resonance imaging (MRI) study we aimed to characterize cardiac function and splanchnic, renal and peripheral blood flow along the spectrum of disease severity from compensated cirrhosis to the advanced stages with decompensation, acute-on-chronic liver failure (ACLF) and/or hepatorenal syndrome-acute kidney injury (HRS-AKI).

**Methods:** We prospectively included 89 patients with cirrhosis and 27 healthy controls in this cohort study. The spectrum comprised patients with compensated cirrhosis (n=27), decompensated cirrhosis (n=33), refractory ascites (n=16), HRS-AKI (n=11), and ACLF (n=2). We measured the hepatic venous pressure gradient (HVPG) and performed MRIs of the heart and organ blood flow.

**Results:** Patients with compensated cirrhosis had higher azygos venous flow than healthy controls (p < 0.01), but cardiac output (CO) and the other flow parameters were not significantly different. Decompensated patients with or without refractory ascites had a significantly higher cardiac index (p < 0.01), higher superior mesenteric artery flow (p = 0.01) and lower systemic vascular resistance (p < 0.001) compared with compensated patients. Patients with HRS-AKI had the highest CO and lowest renal flow of all groups (p < 0.01 and p = 0.02, respectively).

Conclusion: Hemodynamic changes in cirrhosis are closely related to disease severity. Compensated patients show minor hemodynamic changes, whereas decompensated patients exhibit an increasingly hyperdynamic circulation. Interestingly, renal perfusion is low in patients with HRS-AKI despite a substantially higher CO. This finding challenges the prevailing pathophysiological hypothesis of cardiac dysfunction being a major causal factor in HRS-AKI.

3)
DISCONTINUATION OF INFLIXIMAB THERAPY IN PATIENTS WITH CROHN'S DISEASE IN SUSTAINED, COMBINED CLINICAL-BIOCHEMICAL-ENDOSCOPIC REMISSION: A DOUBLE-BLINDED, PLACEBO-CONTROLLED, RANDOMIZED CLINICAL TRIAL

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**Introduction:** It remains unknown whether infliximab (IFX) can be discontinued successfully, once Crohn's disease (CD) patients have attained sustained, combined clinical, biochemical, and endoscopic remission.

**Methods:** Double-blind, randomized, PBO-controlled multicenter trial enrolling patients with luminal CD treated with IFX maintenance therapy for at least 1 year, in combined clinical, biochemical and endoscopic remission at the time of inclusion. Patients were randomized 1:1 to continue IFX therapy or matching PBO for a duration of 48 weeks. Concomitant therapy with stable doses of immunomodulators was allowed. Primary endpoint was time to relapse.

**Results:** Study population comprised 115 patients (n=54 female; age median 34 years [IQR 26-50]; [3-12]; IFX treatment duration median 23 months [16-39]). All patients were in combined clinical-(CDAI median 41 [IQR 15-66]), biochemical- (CRP median 3mg/L [IQR 2-4]), and endoscopic-(Simple Endoscopic Score for CD median 0, [IQR 0-0] (n=99)) remission. Patients were randomized to continued IFX therapy (n=59) or to start PBO infusions (n=56). Time to relapse was significantly shorter among patients who discontinued IFX as compared to those continuing IFX (p<0.001). After one year, relapse-free survival was 51% in the PBO group and 100% in the IFX group (Kaplan Meier survival analysis). Concomitant immunosuppressants numerically increased time to relapse for placebo treated patients. However, also for this subgroup, time to relapse remained significantly shorter than in the IFX group.

**Conclusions:** This first double-blinded placebo-controlled RCT of IFX withdrawal in Crohn's disease patients strongly suggests that discontinuation of IFX leads to a considerable risk of relapse.

### NATURHISTORIEN VED ALKOHOLRELATERET LEVERSYGDOM I EN PROSPEKTIV BIOPTERET KOHORTE MED 422 PATIENTER OG 1.419 PATIENTÅR

Ditlev Rasmussen, Maria Kjærgaard, Katrine Prier Lindvig, Mads Israelsen, Katrine Thorhauge, Nikolaj Christian Torp, Stine Johansen, Sönke Detlefsen, Aleksander Krag, Maja Thiele. *FLASH Center for leverforskning, afdeling S, Odense Universitetshospital* 

**Introduktion:** Alkoholrelateret skrumpelever er en sygdom med høj risiko for komplikationer og død, men naturhistorien for de tidlige sygdomsstadier hos alkoholoverforbrugende patienter med fibrose er ukendt.

**Metoder:** Vi inkluderede patienter med en anamnese med alkoholoverforbrug men uden dekompenseret leversygdom. Vi inddelte patienterne i tre grupper baseret på leverbiopsier, som blev scoret for fibrose, og transient elastography. Leverraske patienter havde enten en leverbiopsi med F0-F1 fibrose eller en transient elastography på <6kPa. Patienter med betydende fibrose havde en leverbiopsi med F2 fibrose, og patienter med fremskreden fibrose havde en biopsi med F3-F4 fibrose. Vi gennemgik manuelt al dokumentation fra patientjournaler fra tiden efter inklusion.

**Resultater:** Vi fulgte 422 patienter igennem 1.419 patientår. Den mediane alder ved baseline var 57 år og 75% var mænd. Den mediane opfølgningstid var 43 måneder, og 53 patienter døde. 51 patienter fik en dekompenserende event. Sandsynligheden for at være i live uden at have fået en dekompenserende event afhang af sygdomsstadiet: 13/225 (5,8%) for de leverraske, 22/104 (21%) ved betydende fibrose og 42/93 (45%) ved fremskreden fibrose. Dødeligheden efter 3 år var 3% for de leverraske, 12% ved betydende fibrose og 22% ved fremskreden fibrose. De hyppigste enkelte dekompenserende events var ascites og overt hepatisk encefalopati efterfulgt af variceblødning. Patienter som havde et alkoholoverforbrug i løbet af opfølgningsperioden havde en højere sandsynlighed for at dekompensere eller dø.

**Konklusion:** Patienter med betydende eller fremskreden alkoholrelateret fibrose har en høj risiko for dekompensation og død på kort sigt. Disse patienter bør følges i et ambulatorium.

### 5) CIRRHOCARE® - A PILOT STUDY OF DIGITAL HOME MANAGEMENT TO DETERMINE FEASIBILITY AND UTILITY TO DIAGNOSE NEW DECOMPENSATION

Konstantin Kazankov<sup>1,2</sup>, Devnandan Amor Chatterjee<sup>1</sup>, Simone Novelli<sup>1</sup>, Alexandra Phillips<sup>1</sup>, Anu Balaji<sup>3</sup>, Maruthi Raja<sup>3</sup>, Rajiv Jalan<sup>1,3</sup>, Ravan Boddu<sup>3</sup>, Ravi Kumar<sup>3</sup>, Rajeshwar P. Mookerjee<sup>1,2</sup>

**EVENTS IN ADVANCED CIRRHOSIS** 

**Introduction:** Patients with acute cirrhosis decompensation are at high risk of new complications and need close follow-up, limited by the growing prevalence of cirrhosis and the challenges of COVID-19. Specialist hepatology care in the community is an unmet need.

**Methods:** We included 20 patients with recent acute cirrhosis decompensation. Monitoring devices were linked to CirrhoCare®-App on a smartphone, issued to patients for daily recording of ECG, blood pressure, weight, % body-water, cognitive function, self-reported well-being and food/fluid/alcohol intake, and 2-way patient-physician communication. Twenty control decompensated cirrhosis patients were observed in parallel.

**Results:** Mean follow-up was 10.1±2.4 weeks. Fifteen patients showed good (≥4 readings/week), 2 moderate (2-4/week), and 3 poor engagement (<2/week). In a usability questionnaire scored 1-10, the median score was ≥9 for all questions. Five CirrhoCare®-managed patients had 8 readmissions. The median readmission lasted 5 (IQR 3.5–11) days, with none >14 days. Based on early signs of decompensation, we contacted patients on 16 other occasions, guiding intervention, e.g. advice on fluids, diuretics and laxatives, possibly preventing more readmissions, confirmed by two independent hepatologists. Controls had 13 readmissions in 8 patients, lasting median 7 (IQR 3–15) days with four admissions >14 days; and 6 unplanned paracenteses vs. 1 in CirrhoCare®-managed patients.

**Conclusions:** CirrhoCare<sup>®</sup>'s novel, multimodal, home management in patients with cirrhosis decompensation, is feasible, with excellent patient engagement, prompting *early* diagnosis of new decompensating events and their intervention; hospital readmissions are fewer and shorter than in controls. We propose CirrhoCare<sup>®</sup> for assisted, specialist community management of advanced cirrhosis.

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### 6) INFLIXIMAB USE FOR SEVERE IMMUNE MEDIATED COLITIS

Emilie Dahl<sup>1</sup>, Jacob Bjerrum<sup>1</sup>, Anders Dige<sup>2</sup>, Jørgen Agnholt<sup>2</sup>, Jakob Seidelin<sup>1</sup>

<sup>1</sup>Herlev Hospital, dept. of gastroenterology; <sup>2</sup>Aarhus University Hospital, dept. of gastroenterology

**Introduction:** We assessed the efficacy of infliximab and observed the risk of thromboembolic events, infections and re-hospitalizations in patients with severe immune mediated colitis (IMC) due to check point inhibitors.

**Method:** A double center retrospective cohort study was performed with cancer patients treated with infliximab due to immune mediated colitis at Aarhus and Herlev Hospitals from 2011 to 2021.

**Results:** One hundred patients were included in the study. Infliximab was effective after the first infusion in 48% of patients; after two or more infusions in 75%, 15% had a partly response, 9% had no effect, 1% uncertain. Nine patients needed additional rescue vedolizumab treatment.

On average, patients received an accumulative dose of 5.450 mg (mean, 1.050 - 24.460 mg) of prednisolone and the diarrhea lasted 111 days (mean, 30 - 656 days). Duration of diarrhea associated hospitalizations were 14 days (mean, 0 - 38) split on 1.5 hospitalizations. Thirteen percent of patients had a thromboembolism event within the first 3 months of treatment with infliximab.

During concomitant treatment with steroids and infliximab we registered 100 infections requiring treatment, 30 of which needed hospitalization. Eighteen patients had infections in the gastrointestinal tract, of which 11 patients had *Clostridium difficile* and three needed fecal transplantation.

One year after the first infliximab infusion 38% patients died, and at the time of the trial, a total of 52 patients had died.

**Conclusions:** Severe IMC can be difficult to treat, and heavy use of steroids combined with IFX makes patients prone to infections and prolonged hospitalizations. Treatment algorithms resulting in lowered glucocorticoid exposure should be considered. Due to the high occurrence of embolism, we suggest that thromboprophylaxis treatment is considered during hospitalization for IMC.

## RANDOMISED CLINICAL STUDY: ACUTE EFFECTS OF METFORMIN VERSUS PLACEBO ON PORTAL PRESSURE IN PATIENTS WITH CIRRHOSIS AND PORTAL HYPERTENSION

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**Background:** Portal hypertension is the main determinant of clinical decompensation in patients with liver cirrhosis. In preclinical data metformin lowers portal pressure, but there are no clinical data for this beneficial effect. We aimed to investigate the acute effects of metformin on hepatic venous pressure gradient (HVPG) and liver perfusion.

**Methods:** In a randomised, double-blinded study design, we investigated 32 patients with cirrhosis before and 90 minutes after ingestion of 1000-mg metformin (n = 16) or placebo (n = 16). Liver vein catherisation was performed to evaluate HVPG and indocyanine green (ICG) infusion for investigation of hepatic blood flow.

**Results:** The mean relative change in HVPG was -16% (95% CI: -28% to -4%) in the metformin group compared with 4% (95% CI: -6% to 14%) in the placebo group (time X group interaction, P = 0.008). In patients with baseline HVPG  $\geq 12$  mm Hg clinically significant improvements in HVPG (HVPG <12 mm Hg or a >20% reduction in HVPG) were observed in 46% (6/13) of metformintreated and in 8% (1/13) of placebo-treated patients (P = 0.07). There were no changes or differences in systemic blood pressure, heart rate, hepatic plasma and blood flow, hepatic ICG clearance, hepatic O2 uptake or inflammation markers between groups.

Conclusions: A single oral metformin dose acutely reduces HVPG in patients with portal hypertension without affecting systemic or liver hemodynamics or inflammatory biomarkers. This offers a promising perspective of a safe and inexpensive treatment option that should be investigated in larger-scale clinical studies with long-term outcomes in patients with cirrhosis and portal hypertension.

#### **POSTER + FLASH TALK (Abstract 8-30)**

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## THE INCIDENCE OF EOSINOPHILIC OESOPHAGITIS AMONG CHILDREN IN THE NORTH DENMARK REGION (2007-2017)

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**Introduction:** In the North Denmark Region (NDR), no data is available on the incidence of Eosinophilic Oesophagitis (EoE) among children. The aims of this study were to describe the incidence of EoE in children aged 0-17 in the NDR, and describe diagnostic delay, clinical manifestations, treatment and complications.

**Methods:** This retrospective, registry based, DanEoE cohort study included 18 children diagnosed with EoE between 2007-2017 in NDR. The medical records of the included children were reviewed with attention to symptoms, referring cause, progress of disease, treatment, symptomatic and histological remission and diagnostic delay.

**Results:** The median incidence was 0.86/100000 children annually. The median diagnostic delay was 4 years and 6 months. Sixty percent presented with food impaction at first hospital visit. After initial treatment, one of 18 children achieved complete remission and had a long-term treatment plan.

Conclusions: The calculated incidence was lower than that of comparable studies. Combined with poor remission rates and lack of follow-up, this indicates that EoE is an underdiagnosed and insufficiently treated disease among children in the NDR, especially among girls, infants and toddlers. Our findings suggest that more knowledge among doctors concerning EoE in children could lead to greater incidence, shorter diagnostic delay and more effective treatment.

### ARE CURATIVE-INTENT TREATMENTS FOR HEPATOCELLULAR CARCINOMA TRULY CURATIVE? A NATIONWIDE STUDY

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**Introduction:** The aim of curative-intent hepatocellular carcinoma (HCC) treatments is to restore the patients' survival to what it would have been, had they not developed HCC. We examined the chances of being cured by resection or ablation for HCC in patients with cirrhosis due to alcohol-related liver disease (ALD cirrhosis).

**Methods:** Using the Danish healthcare registries, all patients with ALD cirrhosis who were treated for HCC in 2004-2018 were identified and included in treatment cohorts based on initial HCC treatment. We used cure fraction analyses to estimate the chance of being cured by each HCC treatment.

**Results:** We included 1,087 patients with HCC due to ALD cirrhosis, of whom 51 were treated with resection, 215 with ablation. The cure fraction, i.e. the fraction of patients who experienced no excess mortality from HCC, was 31.8% (95% CI: 0.0–67.5) following resection and 22.9% (95% CI: 2.6–43.2) following ablation. There was a high level of agreement between the different cure models used. In patients who were still alive five years after the initial HCC treatment, the likelihood of having been cured was 69.0% after resection and 60.2% after ablation. For both treatments, a 90% chance of having been cured was reached after seven years.

Conclusions: Based on cure fraction analyses, resection for HCC cures 31.8% of patients with HCC and underlying ALD cirrhosis, while ablation cures 22.9% of patients. Seven years after curative-intent treatments for HCC, surviving patients are 90% likely to be cured of HCC.

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## ACTIVATION AND FUNCTIONAL PRIMING OF BLOOD NEUTROPHILS IN NON-ALCOHOLIC FATTY LIVER DISEASE INCREASING IN NON-ALCOHOLIC STEATOHEPATITIS

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**Introduction:** In non-alcoholic fatty liver disease (NAFLD), neutrophils in liver infiltrates are activated, which may contribute to disease progression towards non-alcoholic steatohepatitis (NASH). However, the functional status of the blood neutrophils remains unknown and their role in the disease mechanisms thus uncertain. We therefore characterised activation and function of blood neutrophils in patients with NAFLD in relation to clinical disease markers and the NAFLD plasma milieu.

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**Methods:** We studied 20 patients with NAFLD, among which 6 patients with NASH, and 14 healthy persons. Neutrophil activation, interleukin (IL)-8 production and oxidative burst were measured by flow cytometry on participants' neutrophils and on healthy neutrophils exposed in vitro to plasma from the study participants.

**Results:** Blood neutrophils from the NASH patients showed a doubling in the expression of the activation marker CD62L. Also, all NAFLD patients had 50-100% increased expression of CD11b. Functionally, NASH neutrophils had 30% elevated IL-8 production and more than doubled spontaneous oxidative burst. In all NAFLD patients, higher spontaneous oxidative burst was associated with worse liver function. Incubation of healthy neutrophils with NAFLD plasma paradoxically slightly reduced CD62L and CD11b expression, and NASH plasma also reduced the frequency of IL-8-producing neutrophils.

Conclusions: In NAFLD, blood neutrophils are activated, and in NASH also functionally primed. This suggests a progressive neutrophil aggressiveness already present with liver fat infiltration. However, NAFLD plasma in vitro, if anything, had the opposite effect on healthy neutrophils so the NAFLD related neutrophil activation cannot be attributed to humoral factors and remains unexplained.

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## BILE ACID MALABSORPTION IN PATIENTS WITH AND WITHOUT CHRONIC DIARRHOEA FOLLOWING RIGHT-SIDED HEMICOLECTOMY FOR COLON CANCER

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**Introduction:** A proportion of colon cancer patients treated with right-sided hemicolectomy have documented long-term bowel dysfunction, including chronic diarrhoea, urgency and faecal incontinence, affecting their quality of life. The underlying causes are unknown. The aim of this study was to investigate the aetiology of chronic diarrhoea among right-sided hemicolectomy patients curatively operated for cancer in the right colon.

**Methods:** Cases with chronic diarrhoea (Bristol stool type 6-7) after right-sided hemicolectomy were compared to a control group of right-sided hemicolectomy patients without diarrhoea. All participants underwent a selenium-75 homocholic acid taurine (SeHCAT) scan to diagnose bile acid malabsorption (BAM). A glucose breath test was performed to diagnose small intestinal bacterial overgrowth (SIBO). Fibroblast Growth Factor (FGF) 19 was measured in fasting blood. In addition, gastrointestinal transit time (GITT) was measured in all participants.

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**Results:** In total, 45 cases and 19 controls were included. In the case group, 82% had BAM, compared with 37% in the control group, P <0.001. SIBO was diagnosed in 73% of cases and in 74% of controls. No association between BAM and SIBO was observed. There was no association between FGF19 and chronic diarrhoea or between FGF19 and SeHCAT retention. GITT was similar in cases and controls.

**Conclusions:** Right-sided hemicolectomy patients with chronic diarrhoea had a higher frequency of BAM than controls, indicating that BAM plays an important role in the bowel dysfunction seen after colonic resection for right-sided colon cancer. Since BAM was frequently found in patients without diarrhoea, further studies are needed.

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## IMPAIRED COGNITION IN MORBID OBESITY IS NOT LINKED TO FATTY LIVER INFLAMMATION BUT TO ADVANCED FIBROSIS

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**Background:** Morbid obesity and the metabolic syndrome have been linked to cognitive problems and it has been speculated that liver injury caused by NAFDL might cause pre-cirrhosis hepatic encephalopathy. The aim of our study was to characterize brain function in pre-cirrhosis NAFLD and access any correlation to NAFLD severity.

**Method:** We examined brain function in 160 morbidly obese (BMI 42±6.1, 122 women, 45 yrs) using Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), StroopEncephal App, portosystemic hepatic encephalopathy test (PSE), and continuous reaction times (CRT). On liver biopsy 38 had no NAFLD, 84 had NAFLD and 38 had NASH. Fibrosis > Kleiner 2 was present in 34. All medications and comorbid diseases were recorded.

**Results:** One in 3 were cognitively impaired in  $\geq 2$  of the psychometric tests. Verbal abilities seemed the least affected while attention, memory, and visuospatial functions were more severely impaired. Age, hypertension, hepatic lobular inflammation, APRI, and Fib-4 were associated with more abnormal psychometric tests, but on multivariate analysis, only lower age remained significant (p<.0001). Poor performance on RBANS was associated with male gender, low age, and sleep apnoea (p<0.005) while diabetes and higher fib-4 negatively affected Stroop (p<0.03). Low PHES was associated with the male gender (p=0.01) and the use of antidepressants and antipsychotics (p=0.006). Reaction time variability was not directly correlated to any of the clinical variables, but very high variability was more prevalent in patients who were on antidepressants and antipsychotics (10vs25%p=0.012).

**Conclusion:** A mixed—etiology cognitive dysfunction is prevalent in the morbidly obese and is worst in the youngest, but no close relation to hepatic damage can be established in this cohort.

## INABILITY TO NAME MORE THAN 20 ANIMALS IN ONE MINUTE SEEMS TO RULE IN MINIMAL HEPATIC ENCEPHALOPATHY IN DANISH PATIENTS WITH LIVER CIRRHOSIS

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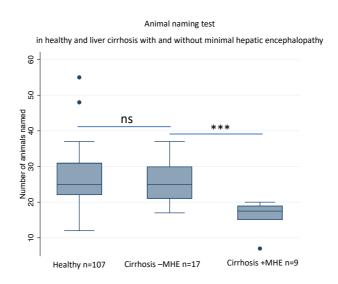
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**Background:** The animal naming test (ANT) is an analogue, one-minute psychometric test measuring semantic fluency. ANT has been proposed as a fast, screening test to identify liver cirrhosis patients needing further psychometric testing to diagnose minimal hepatic encephalopathy (MHE). The main advantaged of the ANT is that it is a one-minute test while other validated tests take up 10–20 minutes. Our aim was to determine the Danish ANT normal interval and identify a preliminary diagnostic cut off for MHE.

**Methods:** 107 healthy controls and 26 with cirrhosis, whereof 9 had MHE, were asked how many animals they could name in one minute. MHE was diagnosed by abnormal continuous reaction time and portosystemic encephalopathy test.

**Results:** Healthy controls mentioned 27 animals on average (16–48, SD 6.1) and there was no effect of gender (p=0.9), age (r=0.052, p=0.05) or education (r=0.01, p=0.001). Patients with cirrhosis and no MHE named 24 animals (15–36, SD 5.9) whereas those with MHE named 16 animals (7–20, SD 4.7, p=0.0013) (see figure). An ANT ≤ 20 identify all +MHE patients (Sens 100%, NPV=100%, AUROC 0.96) but falsely include 12% of healthy and 17% of no-MHE cirrhotics (specificity 82%).

**Conclusion:** ANT above 20 could rule out MHE in the cirrhotic cohort while ANT ≥ 20 seems to identify patients with severe MHE (abnormal PSE and CRT) without extensive psychometric testing. The presented data are preliminary. We will include more patients and the ANT's ability to detect a response to treatment will be evaluated.



### NATRIURETIC PEPTIDES AS TREATMENT OF CIRRHOTIC ASCITES: A SYSTEMATIC REVIEW WITH META-ANALYSIS ON EFFECTS AND SAFETY

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**Introduction:** Natriuretic peptides are elevated in patients with cirrhosis and portal hypertension to counterbalance anti-natriuretic factors and vasoconstriction. The effects of natriuretic peptides as treatment of cirrhotic ascites have been investigated in only small studies, and definitive results are lacking. We systematically reviewed the literature and meta-analysed results to conclude on the effects and safety of natriuretic peptides as treatment for cirrhotic ascites.

**Methods:** We searched MEDLINE, Scopus, Web of Science, Embase, and Cochrane Library for all studies investigating an intravenous administration of atrial natriuretic peptide (ANP), B-type natriuretic peptide, or Urodilatin to cirrhosis patients with ascites. The primary outcome was change in renal sodium excretion. Secondary outcomes comprised safety measures and changes in renal water excretion, plasma aldosterone concentration (PAC), and plasma renin activity (PRA).

**Results:** 22 studies were included. ANP was the only intensively studied treatment. Sodium excretion increased as response to continuous ANP infusion and was most pronounced when infusion rates >30 ng/kg/min were applied and in study subgroups with mild/moderate ascites. PAC and PRA were significantly elevated at baseline in subgroups achieving a negative sodium balance compared with treatment non-responders. Blood pressure drops occurred less frequently with doses ≤30 ng/kg/min. The quality of evidence for a natriuretic response to ANP was low, mainly due to small sample sizes and considerable between-study heterogeneity.

**Conclusions:** ANP infusions increase renal sodium excretion in patients with cirrhotic ascites with the largest effect for infusion rates >30 ng/kg/min. However, infusion rates  $\le 30$  ng/kg/min are less likely to affect blood pressure.

# 15) DIAGNOSTIC ACCURACY OF THE HEPATORENAL B-MODE RATIO TO DIAGNOSE HEPATIC STEATOSIS COMPARED TO IMAGING IN ALCOHOL RELATED AND NON-ALCOHOLIC FATTY LIVER DISEASE

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**Introduction:** We aimed to evaluate the diagnostic accuracy of the hepatorenal B-mode ratio to assess hepatic steatosis and compare it with regular ultrasound steatosis scoring, and Controlled Attenuation Parameter (CAP).

<sup>&</sup>lt;sup>4</sup>Lise Lotte Gluud and Henning Grønbæk contributed equally to the work and share last authorship.

**Methods:** We prospectively included participants at risk of alcohol related and non-alcoholic fatty liver disease for same day B-mode ratio, ultrasound, CAP, and liver biopsy. Histological steatosis grades were used as reference.

**Results:** Of the 137 participants 72% were male, median age was 60 years, and median BMI was 32 kg/m². On histology 80% had steatosis. B-mode ratio diagnosed any steatosis (≥S1), moderate-severe steatosis (≥S2), and severe steatosis (=S3) with AUROC 0.789 (95% CI 0.701-0.877), 0.756 (0.663-0.848), and 0.737 (0.619-0.855) respectively. The cut-off values to rule-out and rule-in any steatosis (≥S1) was 1.09 and 1.45 respectively. There was no difference in the diagnostic accuracy depending on disease aetiology (alcohol related versus non-alcoholic fatty liver disease). The diagnostic accuracy of ultrasound steatosis scoring, and CAP did not differ from B-mode ratio. B-mode ratio had a failure rate of 12% (1% for ultrasound and 2% for CAP) and failed measurements were predicted by a large skin to capsule distance (OR 8.4, p=0.012).

**Conclusion:** The hepatorenal B-mode ratio has a moderate diagnostic accuracy and does not outperform regular B-mode ultrasound steatosis scoring or CAP to diagnose hepatic steatosis in patients with fatty liver disease. B-mode ratio has a higher failure rate which limits its clinical use.

## 16) STUDY PROTOCOL: EFFECTS OF PERIPHERALLY ACTING $\mu$ -OPIOID RECEPTOR ANTAGONISTS ON ACUTE PANCREATITIS

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**Introduction**: Acute pancreatitis (AP) is associated with a high rate of complications and increased mortality, yet no targeted pharmacologic treatment currently exists. As pain is a dominant symptom in AP, patients are exposed to excess levels of both endo- and exogenous opioids, which may worsen the course of AP. We wish to investigate the effects of the peripherally acting  $\mu$ -opioid receptor antagonist (PAMORA) methylnaltrexone on potential harmful effects of opioids on the gastrointestinal tract and the immune system in patients with AP.

Methods: PAMORA in AP is a multicentre, investigator-initiated, double-blind, randomized-controlled, parallel-group trial, which will be conducted at four referral centres for pancreatitis in Denmark. Ninety patients with early-onset AP (pain onset within 48 hours) and predicted moderate-to-severe disease (based on two or more systemic inflammatory response syndrome (SIRS) criteria) will be prospectively included and randomized (1:1) to intravenous treatment with methylnaltrexone or matching placebo (Ringer-lactate) during 5 days of admission. The primary endpoint is the group difference in disease severity documented by the Pancreatitis Activity Scoring System (PASS) score 48 hours after randomization. Secondary endpoints include measures of pancreatic complications, gut motility, intestinal permeability, circulating pro- and anti-inflammatory cytokines as well as patient symptoms and clinical outcomes.

Results: Recruitment has begun in May 2021.

**Conclusions:** This study has potential to document effects of a novel targeted pharmacotherapy in patients with AP with the potential benefit of improved patient outcomes.

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## ATYPISK TILFÆLDE AF HEPATITS A INFEKTION HOS 4 KVINDER MED KLINISK PRÆSENTATION SOM AUTOIMMUN HEPATITIS

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**Formål:** Gennemgang af ophobede tilfælde med prolongeret akut viral hepatitis A infektion (HAV) med en klinisk præsentation identisk med autoimmun hepatitis (AIH).

**Metode:** Systematisk journal evaluering incl hepatitis serologi, genotype, autoimmunmarkører, comorbiditet, evaluering af leverbiopsi, og respons på behandling.

**Resultat:** Vi fandt 4 kvinder (48-67 år) med kliniske tegn på akut HAV infektion indenfor 4 mdr i 2020. Der var ingen mistanke om fælles smittekilde eller generelt udbrud af HAV infektion. HAV verificeret med pos HAV-RNA genotype 1a(2) eller 1b(2). Initial ALAT Stigning (1000-3000 u/L). Efterfølgende Spontan fald til 2-ciftrede værdier initialt, men med fornyet ALAT stigning median 40 dage (45-27). Alle havde forhøjet IgG (15-35), 1 af 4 pos ANA og SMA antistof.

Leverbiopsi hos 4/4 i fase 2 af sygdom med histologiske forekomst af interfase hepatitis, øget antal plasmaceller og histologiske forandringer foreneligt med viral hepatitis men histologi såvel med AIH præg hos alle. 3/4 patienter med Alvarez-score foreneligt med AIH (11-12). 2/4 prednisolon behandlet i median 80 dage (45-120 dage). 4/4 opnåede langvarig normalisering af levertal efter 1 måned i prednisolone gruppe og ca 4 mnd i konservativ gruppe. Ingen har haft tegn på relaps eller udvikling af kronisk leverskade.

**Konklusion:** HAV infektion er beskrevet som sjælden initiator af AIH. Vores serie af patienter med atypisk langstrakt HAV infektion med AIH præg bekræfter vigtighed af leverbiopsi og kritisk evaluering af steroid behandling ved atypiske HAV infektioner. Langvarig immundæmpende behandling frarådes hvis der ikke findes klassiske AIH forandringer histologisk og bekræfter vigtighed af kontrol af levertal til langvarig normalisering ved HAV infektion.

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## ALCOHOL CONSUMPTION AND THE INTEREST IN REDUCING IT AMONG PATIENTS WITH ALCOHOL-RELATED LIVER DISEASE: A NATIONAL HEALTH SURVEY

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**Introductions:** Patients with alcohol-related liver disease (ALD) should stop drinking alcohol. We aimed to identify clinical characteristics associated with interest in reducing alcohol consumption in patients with ALD.

**Methods:** We linked information from the Danish National Health Surveys 2010, 2013, and 2017 with national health registries to identify participants diagnosed with ALD and their subsequent engagement in alcohol misuse treatment. We estimated adjusted prevalence ratios (PR) of being interested in reducing alcohol consumption according to hospital contacts and alcohol problems. The cumulative incidence of engagement in alcohol misuse treatment was calculated with competing risk method.

**Results:** Out of 674 ALD patients, 58% of them with cirrhosis, 35% were abstainers and 41% drank more than 14 units per week. Among the 436 alcohol drinkers, 30% were interested in reducing alcohol consumption. A recent hospital admission and high severity of alcohol problems were associated with being interested in reducing alcohol consumption. The PR was 1.5 (95% CI, 1.2-2.0) for being interested in reducing alcohol consumption in those with a recent hospital admission vs. those without. Patients with interest in reducing alcohol consumption were more likely to engage in alcohol misuse treatment during two years of follow-up (29% [95% CI, 23-39]) than patients without such interest (6.5% [95% CI, 3.9-11]).

**Conclusions:** Among patients with ALD, those who had recently been admitted to the hospital and had severe alcohol problems were more motivated to reduce alcohol consumption. These findings help us identify patients who should be targeted for interventions to reduce alcohol consumption.

# 19) RISK OF RECTAL CANCER AFTER COLECTOMY IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE – A NATIONWIDE POPULATION-BASED DANISH COHORH STUDY 1978-2018

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**Introduction:** We performed a nationwide population-based cohort study to estimate the risk of developing rectal cancer (RC) in patients with inflammatory bowel disease (IBD) after subtotal colectomy.

**Methods:** Through the Danish Civil Registration System, a source population of all individuals 15 years or older living in Denmark between 1978 and 2018 was retrieved.

The risk of RC in patients with diverted rectum was assessed using Cox regression analyses, as compared to the background population as well as to subjects with IBD without subtotal colectomy.

**Results:** RC occurred in 42 (0.9%) of 4931 patients after subtotal colectomy with diverted rectum, compared to 209 (0.4%) of 49,251 in the matched IBD cohort with no colectomy and 941 (0.4%) of 246,550 in the matched background population.

The hazard ratio (HR) for RC in IBD patients with diverted rectum vs. matched IBD patients without colectomy (adjusted for IBD type and sex) was 0.80 (95% CI 0.29, 2.17) during the first 10 years and 7.93 (95% CI 5.48, 11.48) 10 years after colectomy. Likewise, the HR for RC in IBD patients with diverted rectum compared to the matched background population was 0.85 (95% CI 0.32, 2.28) during the first 10 years and 10.25 (95% CI 7.36, 14.28) 10 years after colectomy.

**Conclusions:** In our nationwide population-based Danish cohort study, we found risk of RC in the diverted rectum to be markedly increased 10 years post collectomy.

#### 20)

### EXTRAHEPATIC AUTOIMMUNE DISEASES IN AUTOIMMUNE HEPATITIS: EFFECT ON MORTALITY

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**Introduction:** Autoimmune hepatitis (AIH) is a chronic liver disease associated with an increased prevalence of extrahepatic autoimmune diseases and an increased mortality compared with the general population. The contributing effect of extrahepatic autoimmune diseases on the increased mortality has not been clarified. Our aim was to determine the effect of extrahepatic autoimmune diseases on mortality in patients with AIH.

**Methods:** This was a nationwide register-based cohort study including Danish patients diagnosed with AIH between 1995 and 2019. We examined the concurrency of extrahepatic autoimmune diseases at the time of AIH-diagnosis in the included patients. We followed the patients until April 2021 and compared the cumulative mortality based on Kaplan-Meier estimator in patients with or without extrahepatic autoimmune diseases. We estimated the effect of extrahepatic autoimmune diseases on mortality using cox regression adjusted for age, sex and number of hospital visits.

**Results:** Our study included 2568 patients with AIH of whom 20.7% had concurrent extrahepatic autoimmune diseases. The 10-year cumulative mortality was 27.0% (95% Confidence Interval [CI]: 25.0-29.1) in patients with and 22.1% (95% CI: 20.4-24.0) in patients without extrahepatic autoimmune diseases. The adjusted hazard ratio of death was 1.12 (95% CI: 0.92-1.36) in AIH patients with vs. without concurrent extrahepatic autoimmune diseases; it was 1.07 (95% CI: 0.86-1.32) for patients with one extrahepatic autoimmune disease, and 1.47 (95% CI: 0.95-2.26) for those with more than one.

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**Conclusions:** Extrahepatic autoimmune diseases increase the mortality in patients with AIH. The effect increases especially when having multiple extrahepatic autoimmune diseases concurrent with AIH.

#### 21)

## DISEASE SPECIFIC LIPIDOMIC TRAJECTORIES CHARACTERIZE DELAYED MUCOSAL WOUND HEALING IN QUIESCENT ULCERATIVE COLITIS

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**Introduction:** Changing the long-term prognosis of ulcerative colitis (UC) requires sustained deep mucosal colonic healing with histologic remission and complete wound healing, making the study of colonic tissue regeneration essential. In experimental colitis models, lipid metabolites are increasingly recognized as pivotal components of this process. This study aimed to describe the kinetics of wound healing and reveal potential mechanisms of regeneration and thus new therapeutic targets using a lipidomic approach in humans with or without UC.

**Methods:** Experimental colonic wounds were created endoscopically in patients with quiescent UC (n=21) and healthy controls (n=9) and surveilled by serial endoscopies to calculate an endoscopic wound healing score, i.e., a high wound score associated with compromised wound healing, and cross-sectional wound biopsies at day two and seven post-wounding. These biopsies were analyzed by liquid chromatography coupled with mass spectrometry focusing on lipids.

**Results:** Endoscopic wound scores were significantly higher in UC at day two (p=0.001) and seven (p<0.0001), demonstrating a prolonged wound healing process. The wound scores were correlated with lipid mediators crucial for normal human intestinal regeneration and revealed sustained UC-specific changes in key phospholipids and eicosanoids, i.e., lysophosphatidylcholine, phosphatidylcholine, lysophosphatidic acid, phosphatidylglycerol, phosphatidylinositol, prostaglandin  $D_2$ , and prostaglandin  $E_1$ .

**Conclusions:** A prolonged and untimely wound healing process is identified in patients with quiescent UC with underlying altered disease specific lipidomic trajectories providing a rationale for devising novel therapeutic avenues for stimulating mucosal regeneration and thus challenging the traditional immune suppression treatment dogma.

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### TIDLIGERE HOSPITALSKONTAKTER FOR ALKOHOLMISBRUG KAN IKKE FORUDSIGE GRADEN AF FIBROSE VED ALKOHOLRELATERET LEVERSYGDOM

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**Baggrund:** Patienter, der har en hospitalskontakt for alkoholrelaterede problemer, er i risiko for senere at få skrumpelever. Måske er det muligt at intervenere på disse hospitalskontakter og forebygge udvikling af cirrose.

**Metode:** Leverraske patienter havde en leverstivhed på under 6 kPA vurderet ved transient elastography eller en biopsi med F0-F1 fibrose. Patienter med betydende fibrose og fremskreden fibrose havde en leverbiopsi med hhv. F2 og F3-F4 fibrose. Vi koblede patienternes fibrosegrad til ICD-10 diagnose koder fra Landspatientregisteret for patienternes hospitalskontakter i de 18 år, der gik forud for leverbiopsi. Vi skelnede mellem alkoholintoksikation (F10.0), skadelig brug af alkohol (F10.1) og alkoholafhængighed (F10.3-F10.5)

**Resultater:** Vi studerede den medicinske forhistorie for 462 patienter; 261 leverraske, 107 med betydende fibrose og 94 med fremskreden fibrose. I løbet af de 18 år havde 45% af patienterne en eller mere alkoholrelateret hospitalkontakt, og der var ikke nogen forskel mellem grupperne (hhv, 43%, 50% og 44%, P=0,57 for forskel). Andelen af patienter, der havde mindst en alkoholrelateret kontakt var ens for de tre grupper igennem hele den 18-årsperioden. Ingen af grupperne havde et stabilt mønster af alkohol-overforbrug over tid. En forudgående diagnose med polyneuropati var fire gange så almindelig for patienter med fremskreden fibrose og ulcera var dobbelt så almindelige.

**Konklusion:** Mere end halvdelen af de patienter, der udvikler fremskreden fibrose, har aldrig haft en hospitalskontakt med en diagnosekode for alkoholoverforbrug, og hyppigheden af alkoholrelaterede kontakter kan ikke bruges til at forudsige, hvem der vil udvikle fremskreden fibrose i løbet af de næste to årtier.

## COMPREHENSIVE LIPIDOMICS REVEALS PHENOTYPIC DIFFERENCES IN HEPATIC LIPID TURNOVER IN ALD AND NAFLD DURING ALCOHOL INTOXICATION

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**Introduction:** In experimental models, alcohol induces acute changes in lipid metabolism that cause hepatocyte lipoapoptosis and inflammation. Here we study human hepatic lipid turnover during controlled alcohol intoxication.

**Methods:** We studied 39 participants with three distinct hepatic phenotypes: alcohol-related liver disease (ALD), non-alcoholic fatty liver disease (NAFLD), and healthy controls. Alcohol was administrated via nasogastric tube over 30 minutes. Hepatic and systemic venous blood were sampled simultaneously at three time points: baseline, 60 and 180 min after alcohol intervention. Liver biopsies were sampled 240 minutes after alcohol intervention. We used ultra-high-performance liquid chromatograph mass spectrometry to measure levels of more than 250 lipid species from the blood and liver samples.

**Results:** After alcohol intervention, the levels of blood free fatty acid (FFA) and lysophosphatidylcholine (LPC) decreased while triglyceride (TG) increased. FFA was the only lipid class to decrease in NAFLD after alcohol intervention, while LPC and FFA decreased and TG increased after intervention in ALD and healthy controls. Fatty acid chain uptake preference in FFAs and LPCs were oleic acid, linoleic acid, arachidonic acid, and docosahexaenoic acid. Hepatic venous blood FFA and LPC levels were lower when compared to systemic venous blood levels throughout the intervention. After alcohol intoxication, liver lipidome in ALD was similar to that in NAFLD.

**Conclusions:** Alcohol intoxication induces rapid changes in circulating lipids including hepatic turnaround from FFA and LPC, potentially leading to lipoapoptosis and steatohepatitis. TG clearance was suppressed in NAFLD, possibly explaining why alcohol and NAFLD are synergistic risk factors for disease progression. These effects may be central to the pathogenesis of ALD.

### BINGE DRINKING INDUCES AN ACUTE RELEASE OF MARKERS OF HEPATIC FIBROGENESIS

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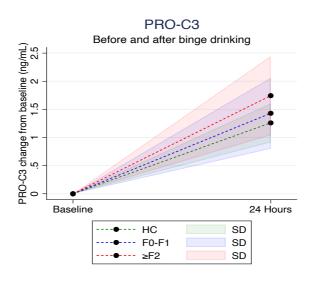
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**Introduction:** Binge drinking increases the risk of alcohol-related cirrhosis. The fibrogenesis, leading to cirrhosis, is a long-standing process of collagen deposition in the hepatic extracellular matrix (ECM). However, fibrogenic activity is highly dynamic and the acute effects of binge drinking on ECM turnover are largely unknown. We aimed to investigate hepatic fibrogenesis and fibrolysis following a binge drinking episode.

Method: We performed a pathophysiological intervention study in 39 participants (16 with ≥F2 fibrosis, 13 with F0-F1, and 10 healthy controls). To mimic binge drinking, participants with alcohol-related liver disease, non-alcohol related liver disease and healthy controls received 2.5 mL of 40% ethanol per kg body weight, infused over 30 minutes via a nasogastric tube. We sampled venous blood simultaneously from the hepatic and right external jugular vein, while peripheral venous blood was sampled 24 hours after alcohol infusion. Markers of hepatic fibrogenesis (PRO-C3) and fibrolysis (C3M) were measured with competitive ELISA assays.

**Results:** Baseline blood alcohol concentration was  $0 \pm 0$  mmol/L. Baseline level of peripheral PROC3 was 12.6 ng/mL (IQR 9.8; 16.2) and increased by +1.2 ng/mL (p < 0.001) after 24 hours, with no difference between the groups (p = 0.661). Increasing baseline PRO-C3 was associated with a hepatic PRO-C3 increase of +0.03 ng/mL/h (95% CI 0.01; 0.05, p = 0.005) and a systemic PRO-C3 decrease of -0.04 ng/mL/h (95% CI -0.05; -0.02, p < 0.001) during the first three hours. No association between peripheral C3M and PRO-C3 was observed 24 hours after binge drinking (p = 0.911).

**Conclusion:** Binge drinking induces an acute burst release of fibrogenic markers in healthy individuals and in patients with fatty liver disease. Fibrosis degradation C3M is not correlated to fibrosis formation PRO-C3, indicating that even a single episode of binge drinking promotes hepatic fibrogenesis.



## TOLV UGERS STYRKETRÆNING NEDSÆTTER RISIKOEN FOR INDLÆGGELSE OG BEDRER OVERLEVELSEN BLANDT CIRROSEPATIENTER – TREÅRSOPFØLGNING EFTER RANDOMISERET FORSØG

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**Baggrund:** Levercirrosepatienters sygdomsforløb er ofte alvorlige og præget af mange hospitalsindlæggelser. Hyppigt ender forløbene med en alt for tidlig død.

Fysisk aktivitet gavner helbredet og øger muligheden for et længere liv, selv hos patienter med kroniske sygdomme.

Formålet med dette studie var at undersøge, om styrketræning påvirker risikoen for indlæggelse, samt overlevelsen blandt patienter med cirrose.

**Metode:** 39 personer med cirrose Child-Pugh A/B deltog oprindelig i et klinisk forsøg, hvor 10 deltagere ad gangen blev blokrandomiseret til en kontrolgruppe eller en træningsgruppe. I 12 uger styrketrænede træningsgruppen 3x1 time ugentligt i 5-mands grupper.

Den første træningsdag lå maksimalt en uge efter randomiseringen og var startdato for hver blok/10-mands gruppe. Alle blev fulgt i 3 år fra startdato. Datoen for den første akutte hospitalsindlæggelse efter startdatoen (ikke genindlæggelser) samt eventuel død blev uanset årsagen registreret.

Fine and Gray-regression blev anvendt til at analysere risikoen for indlæggelse med død som competing risk. Overlevelse blev estimeret ved Cox-regression. Analyserne blev justeret for Child-Pugh ved randomisering.

**Resultater:** 9 fra træningsgruppen og 15 fra kontrolgruppen blev akut indlagt minimum én gang. Risikoen for første indlæggelse var efter 3 år lavere i træningsgruppen end kontrolgruppen: adjusted subdistribution hazard ratio: 0,40 (95% CI: 0,17-0,92; p = 0,032).

En fra træningsgruppen og 6 fra kontrolgruppen døde. Vi fandt en signifikant forskel i overlevelsen til fordel for træningsgruppen i 3-års opfølgningsperioden: adjusted hazard ratio 0.12 (95% CI: 0.01-0.96; p = 0.046).

**Konklusion:** 12 ugers styrketræning mindsker risikoen for indlæggelse og bedrer overlevelsen blandt kompenserede cirrosepatienter tre år efter træningsstart.

## FATIGUE AND QUALITY OF LIFE IN PATIENTS WITH NEUROENDOCRINE NEOPLASIA

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**Introduction:** Neuroendocrine Neoplasms (NEN) are rare cancers most often arising in the gastro-intestinal tract or lungs and with an increasing incidence. Poor health related quality of life (HRQoL) may be caused by the carcinoid syndrome caused by some NENs. Further, cancer related fatigue is one of the most common and distressing side effects of cancer and its treatments and may persist for years after treatment completion - even in healthy survivors. We aimed to quantify HRQoL and fatigue in all out-patients at our ENETS Center of Excellence at Aarhus University Hospital.

**Methods:** In a cross-sectional study, we included 231 patients with NEN (G1-G3). We distributed the pre-validated questionnaires MFI-20 and EQ5D-5L along with a baseline questionnaire. Moreover, we collected clinical, biochemical, imaging and pathology data from the Electronic Patient files. 85% of patients provided full responses.

#### **Results:**

At inclusion patients median age was 62 (range 21-9) and 51% were female. Primary tumor location was found in the small intestine: n=95 (41%), pancreas: n=52 (22.5%), lung n=38 (16.5%), colon n=13 (5.5%) rectum n=6 (2.5%), and other n=27 (12%). 47% of patients had a G1 tumor with KI-67 index <2%, 33% were G2 with Ki67-index 3-20% and 20% were G3 with Ki67-index>20%.

Cured patients reported higher self-reported quality of life than patients with current disease; 61.3 versus 69.5 (scale of 0-100, 100 being best, p=0.01). Patients with high grade neoplasms reported more anxiety and depression (p<0.00) and women reported higher levels of general fatigue than men (p=0.01). Patients receiving somatostatin analogues reported lower general fatigue scores than other patients (p<0.01). Self-reported health seemed higher among patients receiving somatostatin analogues compared to those receiving peptide receptor radionuclide therapy-treatment (64.6 vs 57.8, p 0.06). Likewise, self-reported health tended to be worse in patients who had received any form of chemotherapy compared to patients who had not (51.2 vs. 65, p=0.11).

Conclusion: In general, cured patients report better QoL than patients with active disease and QoL and fatigue differs more in patients with aggressive and disseminated disease than in patients with localized disease. HRQoL is of relevance in patient's assessment and should be taken into consideration for treatment decisions.

27)

## ALBUMIN-CORRECTED ZINC AND AVAILABLE FREE ZINC-BINDING CAPACITY AS INDICATORS OF ZINC STATUS IN PATIENTS WITH CIRRHOSIS AND SHORT-BOWEL SYNDROME – POTENTIAL FOR CLINICAL IMPLEMENTATION

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Introduction: Globally, zinc deficiency constitutes a nutritional problem associated with increased morbidity and mortality. Deficiency develops due to malnutrition, malabsorption and/or increased endogenous losses. Two patient groups at risk of deficiency are patients with cirrhosis and patients with short-bowel syndrome (SBS). Plasma zinc is currently used to assess zinc status, however, circulating zinc is influenced by several factors unrelated to actual zinc status, especially hypoalbuminemia, and may be a poor indicator of deficiency. The aim of this study was to examine whether albumin-corrected plasma zinc and/or available free zinc-binding capacity provide a more accurate diagnostic assessments of zinc status.

**Methods:** Reference intervals for albumin-corrected plasma zinc and available free zinc-binding capacity were established using blood from 200 blood donors. Clinical applicability to detect zinc deficiency was tested in 21 SBS and 109 cirrhotic patients and 31 healthy controls using plasma zinc as the standardized reference measurement.

**Results:** For SBS patients, nine were zinc-deficient based on plasma zinc, while one and two were deficient according to albumin-corrected zinc (p=0.009) and zinc-binding capacity (p=0.02). For cirrhotic patients, 89 were zinc-deficient according to plasma zinc, while this number was reduced to four and 22 according to albumin-corrected zinc (p<0.0001) and zinc-binding capacity (p<0.0001). The healthy controls were zinc-sufficient regardless zinc evaluation methods.

**Conclusion:** Patients' zinc deficiency may be overestimated by standard measurement of plasma zinc, possibly resulting in overuse of oral zinc supplements. Albumin-corrected zinc and zinc-binding capacity could be useful methods for more accurate assessment of zinc status in the clinical setting.

## 28) EFFECTIVENESS OF INFLIXIMAB TREATMENT OF COMPLEX CRYPTOGLANDULAR ANAL FISTULAS

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**Introduction**: Anti-TNF $\alpha$  has shown efficacy in the treatment of anal fistulas in patients with Crohn's disease. The role of anti-TNF $\alpha$  in patients with complex cryptoglandular fistulas is so far unexplored

**Methods**: We retrospectively evaluated the effects of infliximab treatment in patients with complex idiopathic anal fistulas refractory to standard surgical intervention. The primary outcome was achievement of substantial clinical improvement defined as sustained, reduced inflammatory activity at perioperative evaluation, i.e. only minimal-to-moderate secretion and induration and a reduction of fistula size of a magnitude that made a lay-open or sphincter-sparring closure procedure possible. Secondary outcomes were symptom improvement, adverse treatment events and fistula healing after the surgical procedure in those achieving the primary outcome.

**Results:** Twenty-two patients were included (18 high transsphincteric, 3 complex low transsphincteric, 1 suprasphincteric fistula). Fistulas had been present for a median of 24 [interquartile range, IQR:12-33] months. In total, 16 patients (73%) achieved the primary outcome of substantial clinical improvement. Median time from infliximab initiation to patients achieved the primary outcome was 11 [IQR: 8-22] months. Sixteen of the patients responding to infliximab received subsequent lay-open or sphincter-sparring closure procedure surgery. Of these, ten (63%) achieved fistula healing. No serious infectious complications to infliximab treatment were seen. One patient developed a new abscess. One patient developed psoriasis (pustolosis palmoplantaris).

**Conclusions:** Infliximab treatment may be considered a supplement to repeated curettage and seton drainage in the management of selected, complex idiopathic anal fistulas. Such combined treatment may make otherwise refractory fistulas amenable to definitive closure attempts.

#### 29)

## CONVOLUTIONAL NEURAL NETWORKS FOR COMPUTER-AIDED EVALUATION OF ENDOSCOPIC SEVERITY IN ULCERATIVE COLITIS

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**Introduction:** Evaluation of endoscopic disease severity is a key component in the management of patients with ulcerative colitis (UC). However, endoscopic assessment suffers from substantial intra-and inter-observer variation, limiting the reliability of individual assessments. Our aim was to develop a deep learning (DL) model capable of distinguishing active from healed mucosa, as well as to differentiate between different degrees of endoscopic disease severity.

**Methods:** 1,484 unique endoscopic images from 467 patients were extracted for classification. Two experts classified all images independently of one another according to the Mayo endoscopic subscore (MES). In cases of disagreement, a third expert classified the images. Convolutional neural networks were considered in the development of our DL model. Five-fold cross-validation was used to develop and select the best model. Unseen test datasets were used to evaluate the models.

**Results:** In the most difficult task – distinguishing between all categories of MES – our final model achieved a mean accuracy of 0.82, a mean AUC of 0.99, test accuracy of 0.84, sensitivity of 0.88, specificity of 0.81 and a weighted Cohen's kappa of 0.83 (p<0.001 compared to the experts).

Conclusions: We propose a new, standardised way of evaluating endoscopic images from UC patients for both clinical and academic purposes. We demonstrate how our DL model is highly capable of distinguishing between all four MES levels of activity. This model will optimize and standardize the evaluation of disease severity measured by the MES across all centres and hospitals, no matter their level of medical expertise.

## OPTIMERET SYGEPLEJE TIL PATIENTER MED INDKAPSLET PANKREASNEKROSE – MULTIMODAL TILGANG

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**Introduktion**: Indkapslet pankreasnekrose er en komplikation, der opstår sent i forløbet af svær pancreatitis. Der er stigende evidens for, at brug af minimalt invasive teknikker, især endoskopi, resulterer i lave mortalitets- og morbiditetsrater. Derimod vides meget lidt om effekten af den understøttende behandling, herunder specialiseret sygepleje, til de svært syge patienter.

**Metode**: Vi har gennem systematisk indsamling af data, identificeret fem sygeplejefaglige fokusområder: forståelse for egen sygdom, ernæring, mobilisering og træning, kommunikation med pårørende, samt ambulant opfølgning med fokus på livskvalitet.

**Resultater**: De fleste af vores patienter har været indlagt i en anden afdeling i adskillige uger, måske endda måneder. Nyt behandlingsforløb venter hos os, derfor er der et udtalt behov for forventningsafstemning, forståelse for egen sygdom og forståelse for de behandlingstiltag der venter. Derfor har vi udarbejdet en kombineret håndbog og logbog for patienter og pårørende.

Vores data har vist, at patienter med indkapslet pankreasnekrose udvikler massivt vægttab, specielt tab af muskelmasse, under sygdomsforløbet. Vi har i samarbejde med vores fysioterapeuter kunne vise, at både muskelmassen og muskelstyrken aftager signifikant under indlæggelsesforløbet. Disse fund har resulteret i øget fokus på ernæringstiltag, bl.a. brug af naso-jejunal ernæring og specialiseret fysioterapeutisk træning.

Opfølgningsdata har vist, at 80-90% af vores patienter vender tilbage til samme sociale og erhvervsstatus som før sygdommen og med god livskvalitet til følge.

**Konklusion**: Vellykket behandling af patienter med indkapslet pankreasnekrose fordrer tæt samarbejde på tværs af lægelige specialer og på tværs af faggrupper – alle bidrager med hver deres ekspertise.

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| Nadia Stokbæk OUH, Svendborg Nikolaj Torp OUH Nina Kimer Bispebjerg Hospital Ninna Drivsholm Herlev Hospital, Gastroenhenden Nurcan Aydemir SUH, Køge Nynne Hummelshøj AUH, Afd. V, Forskningsårsstud. Ole Hamberg Sjællands Universitets Hospital Ove B Schaffalitzky de Muckadell Peter Holland-Fischer Aalborg Universitetshospital Peter Nørregaard Bispebjerg Abdominalcenter Prabh Gill Rigshospital Rasmine Birn-Rydder Aarhus universitets hospital Rasmus Gantzel Aarhus Universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe wildt SUH, Køge Signe Wiese Hvidovre hospital Søren Møller Hvidovre Hospital Stine Sloth Herlev Hospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Deleuran Aalborg Universitetshospital, Skejby Torben Knudsen Sydvestiysk Sygehus Ulrich Bang Mavetarmklinikken Faxe Ulrich Rodes   | Mirabella       | Zhao                | Hvidovre Hospital/Københavns Universitet          |
| Nikolaj Torp OUH Nina Kimer Bispebjerg Hospital Nina Drivsholm Herlev Hospital, Gastroenhenden Nurcan Aydemir SUH, Køge Nynne Hummelshøj AUH, Afd. V, Forskningsårsstud. Ole Hamberg Sjællands Universitets Hospital Ove B Schaffalitzky de Muckadell Peter Holland-Fischer Aalborg Universitetshospital Peter Nørregaard Bispebjerg Abdominalcenter Prabh Gill Rigshospital Rasmine Birn-Rydder Aarhus universitets hospital Rasmus Gantzel Aarhus Universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe wildt SUH, Køge Signe Wiese Hvidovre Hospital Søren Møller Hvidovre Hospital Stine Sloth Herlev Hospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Deleuran Aalborg Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Ulrich Bang Mavetarmklinikken Faxe Ulrich Rodes Mordsållands Hospital Ulrich Rodes Mordsållands Hospital Ulrich Rodes Mordsållands Hospital   | Monika          | Madsen              | Sydvestjysk sygehus, Esbjerg                      |
| Nina Kimer Bispebjerg Hospital Ninna Drivsholm Herlev Hospital, Gastroenhenden Nurcan Aydemir SUH, Køge Nynne Hummelshøj AUH, Afd. V, Forskningsårsstud. Ole Hamberg Sjællands Universitets Hospital Ove B Schaffalitzky de Muckadell Peter Holland-Fischer Aalborg Universitetshospital Peter Nørregaard Bispebjerg Abdominalcenter Prabh Gill Rigshospital Rasmine Birn-Rydder Aarhus universitets hospital Rasmus Gantzel Aarhus Universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe Wildt SUH, Køge Signe Wiese Hvidovre Hospital Søren Møller Hvidovre Hospital Stine Sloth Herlev Hospital Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Sandahl Aarhus Universitetshospital Sydvestjysk Sygehus Troels Busk Herlev Hospital   | Nadia           | Stokbæk             | OUH, Svendborg                                    |
| Ninna Drivsholm Herlev Hospital, Gastroenhenden Nurcan Aydemir SUH, Køge Nynne Hummelshøj AUH, Afd. V, Forskningsårsstud. Ole Hamberg Sjællands Universitets Hospital Ove B Schaffalitzky de Muckadell Peter Holland-Fischer Aalborg Universitetshospital Peter Nørregaard Bispebjerg Abdominalcenter Prabh Gill Rigshospital Rasmine Birn-Rydder Aarhus universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe Wildt SUH, Køge Signe Wiese Hvidovre Hospital Steffan Østergaard Vejle Sygehus Stine Sloth Herlev Hospital Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Damgaard Sandahl Aarhus Universitetshospital Stroels Suffundarie Hvidovre Hospital, Københavns Universitet Stroels Havelund OUH Troels Busk Herlev Hospital Ulrich Rohde Nordsjællands Hospital  | Nikolaj         | Torp                | OUH   |
| Nurcan         Aydemir         SUH, Køge           Nynne         Hummelshøj         AUH, Afd. V, Forskningsårsstud.           Ole         Hamberg         Sjællands Universitets Hospital           Ove B         Schaffalitzky de Muckadell         Odense Universitetshospital           Peter         Holland-Fischer         Aalborg Universitetshospital           Peter         Nørregaard         Bispebjerg Abdominalcenter           Prabh         Gill         Rigshospital           Rasmine         Birn-Rydder         Aarhus universitets hospital           Rasmus         Gantzel         Aarhus Universitetshospital           Rozeta         Abazi         OUH           Sabine         Becker         Regionshospitalet Silkeborg           Sidsel         Støy         Lever-, Mave-, Tarmsygdomme, Aarhus           Signe         Wildt         SUH, Køge           Signe         Wiese         Hvidovre hospital           Søren         Møller         Hvidovre Hospital           Steffan         Østergaard         Vejle Sygehus           Stine         Sloth         Herlev Hospital           Stine         Johansen         Odense Universitetshospital           Thit         Kronborg         Hvidovre Hospital, Københavns U  | Nina            | Kimer               | Bispebjerg Hospital                               |
| Nynne Hummelshøj AUH, Afd. V, Forskningsårsstud.  Ole Hamberg Sjællands Universitets Hospital  Ove B Schaffalitzky de Muckadell  Peter Holland-Fischer Aalborg Universitetshospital  Peter Nørregaard Bispebjerg Abdominalcenter  Prabh Gill Rigshospital  Rasmine Birn-Rydder Aarhus universitets hospital  Rasmus Gantzel Aarhus Universitetshospital  Rozeta Abazi OUH  Sabine Becker Regionshospitalet Silkeborg  Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe wildt SUH, Køge  Signe Wiese Hvidovre hospital  Søren Møller Hvidovre Hospital  Steffan Østergaard Vejle Sygehus  Stine Sloth Herlev Hospital  Stine Johansen Odense Universitetshospital  Thit Kronborg Hvidovre Hospital, Københavns Universitet  Thomas Deleuran Aalborg Universitetshospital  Thomas Damgaard Sandahl Aarhus Universitetshospital  Troels Havelund OUH  Troels Busk Herlev Hospital  Ulrich Rohde Nordsjællands Hospital   | Ninna           | Drivsholm           | Herlev Hospital, Gastroenhenden                   |
| Ole Hamberg Sjællands Universitets Hospital Ove B Schaffalitzky de Muckadell Peter Holland-Fischer Aalborg Universitetshospital Peter Nørregaard Bispebjerg Abdominalcenter Prabh Gill Rigshospital Rasmine Birn-Rydder Aarhus universitets hospital Rasmus Gantzel Aarhus Universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe wildt SUH, Køge Signe Wiese Hvidovre hospital Søren Møller Hvidovre Hospital Steffan Østergaard Vejle Sygehus Stine Sloth Herlev Hospital Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Rohde Nordsjællands Hospital  | Nurcan          | Aydemir             | SUH, Køge   |
| Ove B Schaffalitzky de Muckadell  Peter Holland-Fischer Aalborg Universitetshospital  Peter Nørregaard Bispebjerg Abdominalcenter  Prabh Gill Rigshospital  Rasmine Birn-Rydder Aarhus universitets hospital  Rasmus Gantzel Aarhus Universitetshospital  Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe Wildt SUH, Køge Signe Wiese Hvidovre hospital  Steffan Østergaard Vejle Sygehus  Stine Sloth Herlev Hospital  Stine Johansen Odense Universitetshospital  Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital  Nordsjællands Hospital  Nordsjællands Hospital  Nordsjællands Hospital  Nordsjællands Hospital  | Nynne           | Hummelshøj          | AUH, Afd. V, Forskningsårsstud.                   |
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| PeterHolland-FischerAalborg UniversitetshospitalPeterNørregaardBispebjerg AbdominalcenterPrabhGillRigshospitalRasmineBirn-RydderAarhus universitets hospitalRasmusGantzelAarhus UniversitetshospitalRozetaAbaziOUHSabineBeckerRegionshospitalet SilkeborgSidselStøyLever-, Mave-, Tarmsygdomme, AarhussignewildtSUH, KøgeSigneWieseHvidovre hospitalSørenMøllerHvidovre HospitalSteffanØstergaardVejle SygehusStineSlothHerlev HospitalStineJohansenOdense UniversitetshospitalThitKronborgHvidovre Hospital, Københavns UniversitetThomasBlixtSUH KøgeThomasDeleuranAalborg UniversitetshospitalThomasDeleuranAalborg Universitetshospital, SkejbyTorbenKnudsenSydvestjysk SygehusTroelsHavelundOUHTroelsBuskHerlev HospitalUlrichBangMavetarmklinikken FaxeUlrichRohdeNordsjællands Hospital   | Ove B           | Schaffalitzky de    | Odense Universitetshospital                       |
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| Rasmine Birn-Rydder Aarhus universitets hospital Rasmus Gantzel Aarhus Universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus Signe Wildt SUH, Køge Signe Wiese Hvidovre hospital Søren Møller Hvidovre Hospital Steffan Østergaard Vejle Sygehus Stine Sloth Herlev Hospital Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Rohde Nordsjællands Hospital  | Peter           | Nørregaard          | Bispebjerg Abdominalcenter                        |
| Rasmus Gantzel Aarhus Universitetshospital Rozeta Abazi OUH Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe wildt SUH, Køge Signe Wiese Hvidovre hospital Søren Møller Hvidovre Hospital Steffan Østergaard Vejle Sygehus Stine Sloth Herlev Hospital Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital   | Prabh           | Gill                | Rigshospital                                      |
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| Sabine Becker Regionshospitalet Silkeborg Sidsel Støy Lever-, Mave-, Tarmsygdomme, Aarhus signe wildt SUH, Køge Signe Wiese Hvidovre hospital Søren Møller Hvidovre Hospital Steffan Østergaard Vejle Sygehus Stine Sloth Herlev Hospital Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Thiomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Rohde Nordsjællands Hospital  | Rasmus          | Gantzel             | Aarhus Universitetshospital                       |
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| Signe Wiese Hvidovre hospital  Søren Møller Hvidovre Hospital  Steffan Østergaard Vejle Sygehus  Stine Sloth Herlev Hospital  Stine Johansen Odense Universitetshospital  Thit Kronborg Hvidovre Hospital, Københavns Universitet  Thomas Blixt SUH Køge  Thomas Deleuran Aalborg Universitetshospital  Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby  Torben Knudsen Sydvestjysk Sygehus  Troels Havelund OUH  Troels Busk Herlev Hospital  Ulrich Bang Mavetarmklinikken Faxe  Ulrich Rohde Nordsjællands Hospital   | Sidsel          | Støy                | Lever-, Mave-, Tarmsygdomme, Aarhus               |
| SørenMøllerHvidovre HospitalSteffanØstergaardVejle SygehusStineSlothHerlev HospitalStineJohansenOdense UniversitetshospitalThitKronborgHvidovre Hospital, Københavns UniversitetThomasBlixtSUH KøgeThomasDeleuranAalborg UniversitetshospitalThomas DamgaardSandahlAarhus Universitetshospital, SkejbyTorbenKnudsenSydvestjysk SygehusTroelsHavelundOUHTroelsBuskHerlev HospitalUlrichBangMavetarmklinikken FaxeUlrichRohdeNordsjællands Hospital  | signe           | wildt               | SUH, Køge   |
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| Stine Johansen Odense Universitetshospital Thit Kronborg Hvidovre Hospital, Københavns Universitet Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital  | Steffan         | Østergaard          | Vejle Sygehus                                     |
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| Thomas Blixt SUH Køge Thomas Deleuran Aalborg Universitetshospital Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital   | Stine           | Johansen            | Odense Universitetshospital                       |
| Thomas Deleuran Aalborg Universitetshospital Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital   | Thit            | Kronborg            | Hvidovre Hospital, Københavns Universitet         |
| Thomas Damgaard Sandahl Aarhus Universitetshospital, Skejby Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital  | Thomas          | Blixt               | SUH Køge  |
| Torben Knudsen Sydvestjysk Sygehus Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital  | Thomas          | Deleuran            | Aalborg Universitetshospital                      |
| Troels Havelund OUH Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital   | Thomas Damgaard | Sandahl             | Aarhus Universitetshospital, Skejby               |
| Troels Busk Herlev Hospital Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital   | Torben          | Knudsen             | Sydvestjysk Sygehus                               |
| Ulrich Bang Mavetarmklinikken Faxe Ulrich Rohde Nordsjællands Hospital   | Troels          | Havelund            | OUH   |
| Ulrich Rohde Nordsjællands Hospital  | Troels          | Busk                | Herlev Hospital                                   |
| Ulrich Rohde Nordsjællands Hospital  | Ulrich          | Bang                | Mavetarmklinikken Faxe                            |
|  | Ulrich          | Rohde               | Nordsjællands Hospital                            |
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