Program

Symposium 203

XXIV International Bile Acid Meeting: Bile Acids in Health and Disease

June 17 – 18, 2016
Hilton Düsseldorf
Düsseldorf, Germany

General Information:

FALK FOUNDATION e.V.
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CME credits

Awarded with

8
8 credit hours (CME) have been awarded for the Symposium 203 by the European Union of Medical Specialists (UEMS) - European Board of Gastroenterology (EBG).
Preface

Since the last International Bile Acid Meeting in Freiburg in 2014, the field of bile acid research has continued to flourish and bile acid signaling represents an important area of research. New insights have been gained into the role of bile acid signaling in the liver and intestine and the role of bile acids and their receptors in the gut liver axis. The findings that the nuclear bile acid receptor FXR plays an essential role in the development of obesity and NASH as well as in the beneficial effects of bariatric surgery, offer novel aspects on the pathogenesis of these diseases as well as potential targets for future therapies. Furthermore, downstream targets of bile acid signaling, such as FGF15/FGF19 have been recently identified to control liver size and also contribute to the development of hepatocellular carcinoma.

The XXIV International Bile Acid Meeting, which will be held in Düsseldorf, will be dedicated to both, basic and clinical aspects of bile acid research with focus on the role of bile acid signaling in liver regeneration and tumor development, on bile acids and the gut liver axis as well as on intrahepatic effects of bile acids. Novel aspects of bile acid transport as well as therapeutic strategies using bile acid derivatives or bile acid receptor agonists represent another focus of this conference. The latest findings will be presented by leading scientists and clinicians in these fields.

During the symposium a poster session will also take place. In line with the tradition of the International Bile Acid Meetings some of the best poster abstracts will be selected by the scientific committee and the authors will be invited for oral presentations. The organizers of the XXIV International Bile Acid Meeting look forward to welcome you in Düsseldorf.

Dieter Häussinger
(Chairman of the Organizing Committee)
XXIV International Bile Acid Meeting:
Bile Acids in Health and Disease

June 17 – 18, 2016
Hilton Düsseldorf
Düsseldorf, Germany

Registration:
Thursday, June 16, 2016
16.00 – 21.00 h
at the congress office

Scientific Organization:
U. Beuers, Amsterdam (The Netherlands)
D. Häussinger, Düsseldorf (Germany)
V. Keitel, Düsseldorf (Germany)
M. Trauner, Vienna (Austria)

Congress Venue:
Hilton Düsseldorf
Georg-Glock-Str. 20
40474 Düsseldorf
Germany

Information:
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Official Language:
English

Posters:
For details see page 12.
Friday, June 17, 2016

8.30 Introduction and Welcome  D. Häussinger, Düsseldorf

Session I
Bile acid signaling in liver regeneration and tumor development

Chair: D. Häussinger, Düsseldorf; A.F. Hofmann, La Jolla

8.40 Bile transport analysis by intravital 2-photon microscopy  J.G. Hengstler, Dortmund

9.00 Fibroblast growth factor signaling controls liver size in mice with humanized livers  M. Grompe, Portland

9.20 The FGF15/19 signaling system FGFR4 in hepatocarcinogenesis  M.A. Avila, Pamplona

9.40 Portal vein embolization-triggered liver regeneration is accelerated by the FXR agonist obeticholic acid  F.G. Schaap, Maastricht

10.00 Coffee break with poster session

Session II
Bile acids and the gut liver axis

Chair: U. Beuers, Amsterdam; D. Keppler, Heidelberg

10.30 Role of the intestinal microbiome for cholestatic liver disease  N.F. LaRusso, Rochester

10.50 Intestinal specific actions of nuclear bile acid receptor FXR  A. Moschetta, Bari

11.10 Microbe-host interactions via microbial moderation of bile acid signatures  C. Gahan, Cork

11.30 Bile acids, ceramides and a new approach for the treatment of metabolic disease  F.J. Gonzalez, Bethesda

11.50 Gut feelings: How intestinal FXR controls fatty acid and cholesterol metabolism  R.M. Evans, La Jolla

12.10 Vertical sleeve gastrectomy (VSG) in morbidly obese adolescents results in increased fibroblast growth factor 21 (FGF21) that correlates with weight loss  R. Kohli, Cincinnati

12.30 Lunch break with poster session
Friday, June 17, 2016

14.00 Presentation of Adolf Windaus Award
D. Häussinger, Düsseldorf

14.10 Adolf Windaus Award Lecture
Progress in the molecular characterization of hepatobiliary transporters
D. Keppler, Heidelberg

Session III
Intrahepatic signaling and bile acids as endogenous toxins

Chair: V. Keitel, Düsseldorf; D.D. Moore, Houston

14.30 Nuclear receptors FXR and PPARα regulate hepatic autophagy
D.D. Moore, Houston

14.50 Bile acids and cholangiocyte autophagy
M. Sasaki, Kanazawa

15.10 Soluble adenylyl cyclase regulates bile-salt-induced adoptosis in human cholangiocytes: A link to primary biliary cirrhosis
R.P.J. Oude Elferink, Amsterdam

15.30 Mechanisms of hepatoprotection by tauroursodesoxycholate
D. Häussinger, Düsseldorf

15.50 Steroid binding to autotaxin links bile salts and lysophosphatidic acid signaling
R. Bolier, Amsterdam

16.10 Coffee break with poster session

Session III (cont.)
Intrahepatic signaling and bile acids as endogenous toxins

Chair: P. Fickert, Graz; P.L.M. Jansen, Maastricht

17.00 The role of inflammation in the mechanism of bile acid-induced liver damage
J.L. Boyer, New Haven

17.20 TGR5 and bile acid induced liver damage
V. Keitel, Düsseldorf

17.40 Activation of intestinal bile acid receptor FXR induces membrane G protein-coupled bile acid receptor TGR5 expression and stimulates GLP-1 secretion to ameliorate metabolic disorders in diabetic mice
J.Y.L. Chiang, Rootstown

18.00 Colonization of germ-free mice with a human microbiota induces FXR signaling
A. Wahlström, Gothenburg
Saturday, June 18, 2016

Session IV

Bile acid transporters: Role in health and disease

Chair: A. Moschetta, Bari; R.P.J. Oude Elferink, Amsterdam

8.30 Targeting Ntcp to treat metabolic and cholestatic diseases
S.F.J. van de Graaf, Amsterdam

8.50 Sodium taurocholate cotransporting polypeptide (SLC10A1) deficiency: A new inborn error of metabolism with a complex phenotype
F.M. Vaz, Amsterdam

9.10 Roles of ileal ASBT and OSTα-OSTβ in regulating the FXR/FGF15 pathway and bile acid-induced injury
P.A. Dawson, Atlanta

9.30 ASBT inhibitors in PBC and PSC
G. Hirschfield, Birmingham

9.50 Effect of intrahepatic cholestasis of pregnancy on maternal glucose homeostasis
E. Bellafante, London

10.10 Coffee break with poster session

Session V

Bile acid receptors and bile acid signaling as therapeutic targets

Chair: G. Hirschfield, Birmingham; M. Trauner, Vienna

10.40 Bile acids in polycystic liver disease: Triggers of disease progression and/or potential solution for treatment?
J.M. Banales, San Sebastian

11.00 NorUrsodeoxycholic acid in primary sclerosing cholangitis and non-alcoholic fatty liver disease
M. Trauner, Vienna

11.20 Role of OCA in decompensated cirrhosis
R. Mookerjee, London

11.40 Cholic acid treatment in Zellweger spectrum disorders
F.C.C. Klouwer, Amsterdam

12.00 NorUDCA reduces liver injury and improves the metabolic state in mouse models of obesity and steatosis
D. Steinacher, Vienna

12.20 Presentation of poster awards

12.30 Closing remarks
D. Häussinger, Düsseldorf

12.40 Farewell lunch
Adolf Windaus (1876-1959)

Adolf Windaus was born on Christmas Day in 1876 in Berlin, where his father owned a factory. Even as a young student in the Berlin gymnasium, he was fascinated by the epochal discoveries of Koch and Pasteur, and by his 18th birthday he had decided on a scientific career. He entered medical school, taking his pre-clinical year at the University of Freiburg and his clinical years in Berlin. However, he soon realized, especially during the lectures of Emil Fischer, that biological processes could be understood only when the chemical structure of organisms was known. Therefore, as soon as he had finished medical school, he returned to Freiburg to study chemistry under the supervision of Heinrich Kiliani. In 1899, he completed his first research project which dealt with the chemical composition of digitalis. He then spent two years in compulsory military service in Berlin. During this time he also worked in the laboratory of Emil Fischer, carrying out studies on derivatives of aniline. On completing his military service, Windaus returned to the University of Freiburg where he began his life-long work on the structure of cholesterol. His thesis, which qualified him for the position of docent, had the simple title „Über Cholesterin“. The choice of this research topic originated from Windaus’ logical belief that any substance which was so widely distributed in animal and plant tissues must have an important biological function, and that understanding of its structure and function might lead to unifying concepts, a hypothesis he would subsequently prove so brilliantly. In addition to initiating studies on cholesterol, he and his colleague Knoop soon discovered that an amino acid containing the imidazole ring, histidine, was present in proteins, and could be decarboxylated to give histamine. The discovery of histamine opened a vast area of pharmacological research.

In 1913, Adolf Windaus accepted a call to direct the prestigious Institute of Medical Chemistry in Innsbruck, Austria, where earlier Pregl had founded microanalytical chemistry. Two years later, in 1915, he was called to be Director of the Chemical Laboratories of the University of Göttingen, laboratories rich in tradition since the time of Wöhler. Here, he could pursue his work on elucidating the structure of cholesterol in a series of integrated investigations that were truly Herculean in scope. In the year 1919 a most significant discovery was made. Windaus found that coprostanol could be oxidized to cholic acid. With the knowledge of this transformation, came the realization of the close structural similarity of cholesterol and bile acids; one could now apply the existing knowledge of cholesterol structure to that of bile acids and that of bile acids to cholesterol. The work of elucidating the exact structure of the condensed steroid rings of steroids was extraordinarily difficult. To understand the structural isomerism of the A / B ring juncture, it was necessary to study the simplest model compounds, cis and trans decalin. This was done with Hueckel, who later became one of the world’s greatest physical chemists.

In the twenties, Adolf Windaus, with his pupils, established the relationships between cholesterol and other important steroids such as sitosterol, the saponins, and the various
classes of cardiac steroids. He showed that all shared the cyclopentanophenanthrene nucleus. Inspired by Windaus, his pupil Butenandt isolated and determined the structure of the adrenal steroids whose origins from cholesterol had not been suspected by anyone. Butenandt was able to rapidly determine the structure of estrone, androsterone, and progesterone, for which he received the Nobel Prize in 1939.

Probably the climax in the extraordinary research output of Adolf Windaus was his elucidation of the structure and biosynthesis of vitamin D. Hess in New York had made the observation that ultraviolet radiation of a lipid extract induced the formation of active vitamin D. In the next 8 years, Adolf Windaus and his students succeeded in identifying the provitamin as ergosterol and 7-dehydrocholesterol and also in clarifying the structure of vitamin D2 and vitamin D3. The complex steps in photoactivation of the vitamin were clarified, and each intermediate was crystallized and its structure determined.

Thus, the research area of the chemical structure of cholesterol, which Adolf Windaus had selected when still a young docent in Freiburg led to studies spanning over 30 years – studies which opened up a vast – almost limitless field that continues to be active today. His work has been of inestimable significance for the practice of medicine. Adolf Windaus, however, insisted that his research was not aimed at applications, but only at understanding the mysteries of nature.

Adolf Windaus had a legendary reputation among his colleagues and students. He was a man of infinite energy and extraordinary insight, who could reduce scientific problems to their essence. He had the art to ask the right question and do the definitive experiment. Nature disclosed her secrets quickly to a man of such talent. His former associates had continuous admiration for his clarity of speech, both in conversation and scientific discussion. He was a man of modesty and dignity who combined the highest scientific standards with great personal generosity.

For his many discoveries, Adolf Windaus received many honors and awards. Under his leadership, the Chemical Institute in Göttingen became known throughout the world. He was honored by being chosen to receive the Nobel Prize for chemistry in 1928, and his lecture is a masterpiece of erudition, clarity and modesty.

W. Gerok
Adolf Windaus Award

The "Adolf Windaus Award" was founded by the Falk Foundation e. V. and will, for the nineteenth time, be presented on the occasion of the XXIV International Bile Acid Meeting, on June 17, 2016. The prize amounts to € 15,000 and is awarded for outstanding contributions in the field of bile acid research.

Members of the Prize Committee:
U. Beuers (Amsterdam)
D. Häussinger (Düsseldorf)
R.P.J. Oude Elferink (Amsterdam)
A. Parés (Barcelona)
R. Poupon (Paris)
M. Trauner (Vienna)

Windaus Prize Winners:
1980 - C. Einarsson (Stockholm) & K. Hellstrom (Stockholm)
1982 - E. H. Mosbach (New York) & H. Danielsson (Uppsala)
1984 - M. C. Carey (Boston)
1986 - I. Bjorkhem (Huddinge)
1988 - J. L. Boyer (New Haven)
1990 - P. B. Hylemon (Richmond) & P. J. Meier-Abt (Zurich)
1992 - K. Okuda (Hiroshima)
1994 - Z. R. Vlahcevic (Richmond)
1996 - W. Kramer (Frankfurt)
1998 - P. A. Dawson (Winston-Salem)
2000 - D. J. Mangelsdorf (Dallas)
2002 - D. W. Russell (Dallas)
2004 - K. D. R. Setchell (Cincinnati)
2006 - R. Poupon (Paris)
2008 - N. Ballatori (Rochester)
2010 - J. Auwerx & K. Schoonjans (Lausanne)
2012 - G. Paumgartner (Munich)
2014 - S. Kliewer (Dallas)
2016 - D. Keppler (Heidelberg)

Coordinator of the Prize Committee:
Prof. Dr. Dieter Häussinger
Department of Internal Medicine
Clinic of Gastroenterology, Hepatology and Infectious Diseases
Heinrich-Heine-University Düsseldorf
Moorenstr. 5
40225 Düsseldorf
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Poster Session

Posters will be exhibited June 17-18, 2016 in the Hilton Düsseldorf. The authors will be in attendance during coffee and lunch breaks on both days.

1. Pregnancy alters the liver transcriptome to engage gestational metabolic and inflammatory pathways
   S. Abu-Hayyeh, C. Williamson (London, GB)

2. The role of necroptosis in acute and chronic cholestasis

3. Cyp3a11 is dispensable for the formation of murine bile acid
   S. Al-Dury, A. Wahlström, M. Ståhlman, F. Bäckhed, H.-U. Marschall
   (Gothenburg, SE; Copenhagen, DK)

4. Cafestol but not resveratrol stimulates FGF19 expression in human ileal explants
   (London, GB; Dublin, IE)

5. Bile acids are key regulators of testicular physiology and male fertility
   M. Baptissart, E. Martinot, A. Vega, L. Sédes, B. Rouaisnel, K. Schoonjans, D.H. Volle
   (Aubiere Cedex, FR; Lausanne, CH)

6. Effect of intrahepatic cholestasis of pregnancy on maternal glucose homeostasis
   E. Bellafante, V. Nikolova, J. Chambers, M. Martineau, C. Williamson
   (London, GB)

7. α5β1 integrins are receptors for bile acids with a (nor-)ursodeoxycholane scaffold
   M. Bonus, A. Sommerfeld, D. Häussinger, H. Gohlke (Düsseldorf, DE)

8. Autoimmune BSEP disease is curable with hematopoietic stem cell transplantation
   F. Brinkert, A. Briem-Richter, V. Keitel, I. Müller, E. Grabhorn (Hamburg, Düsseldorf, DE)

9. Selective targeting of fxr isoform α1–4 by novel bile acid derivatives and lipotoxicity protection in HepG2 cells
   H. Brito, S. Batista, J.A. Salvador, R.E. Castro, C.M. Rodrigues
   (Lisbon, Coimbra, PT)

10. Metformin protects rat hepatocytes against bile acid-induced apoptosis
    M. Buist-Homan, T. Woudenberg-Vrenken, L. Conde de la Rosa, K.N. Faber, H. Moshage (Groningen, NL)

11. Soluble adenylyl cyclase regulates bile salt-induced apoptosis in human cholangiocytes
12. Activation of intestinal bile acid receptor FXR induces membrane G protein-coupled bile acid receptor TGR5 expression and stimulates GLP-1 secretion to ameliorate metabolic disorders in diabetic mice
J.Y.L. Chiang, P. Pathak, H. Liu, S. Boehme (Rootstown, US)

13. Prevalence, clinical characteristics and outcomes of antimitochondrial type 2 seropositive patients with non-established primary biliary cholangitis

P.H. Dixon, L. Wu, C. Williamson (London, GB; Chengdu, CN)

15. Drug-drug interactions related to inhibition of the sodium taurocholate co-transporting polypeptide (NTCP) by a novel anti-HBV peptide

16. FIC1, BSEP, and MDR3 sequencing disclosed 139 genetic variants including 63 new ones in 389 unrelated patients with suspected intrahepatic cholestasis
C. Dröge, M. Bonus, S. Kluge, H. Gohlke, L. Schmitt, R. Kubitz, D. Häussinger, V. Keitel (Düsseldorf, DE)

17. Chronic central infusion of taurolithocholate decreases fat mass and increases brown adipose tissue triglyceride derived fatty acid uptake

18. Hormesis in cholestatic liver disease; preconditioning with low bile acid concentrations protects against bile acid-induced toxicity
K.N. Faber, M. Buist-Homan, M. Koehorst, A.K. Groen, H. Moshage, E.M. Verhaag (Groningen, NL)

19. Absence of BSEP/ABCB11 protects from cholestatic liver injury in mice

20. Effects of ursodeoxycholic acid on FXR-mediated stimulation of FGF19 in human ileal explants

21. An experimentally validated binding mode model of TGR5 agonists
C.G.W. Gertzen, L. Spomer, S.H.J. Smits, D. Häussinger, V. Keitel, H. Gohlke (Düsseldorf, DE)

22. A novel fluorescent analogue of TUDCA reveals new mechanistic insights into TUDCA cytoprotection
J.F. Gilmer, J. Gavin, F. Quilty, G. Radics (Dublin, IE)
23. A novel protocol enables the differentiation of human pluripotent stem cell derived bipotential hepatoblasts into hepatocyte or cholangiocyte like cells
N. Graffmann, W. Wruck, J. Adjaye (Düsseldorf, DE)

24. Bile acid biosynthesis avoiding cholesterol
(London, Swansea, Manchester, GB; Oakland, US; Stockholm, SE)

25. Genetic analysis of spontaneous (non-toxic) liver fibrosis in a congenic mouse model
R.A. Hall, K. Hochrath, F. Lammert, F. Grünhage (Homburg, DE)

26. Enhanced ileal bile acid uptake may prevent vitamin A and/or D deficiency in Dutch Crohn’s disease patients
J. Heegsma, L. Wymenga, M. Hoekstra, T. Blokzijl, L. Groen, H. Groen, G. Dijkstra, K.N. Faber (Groningen, NL)

27. Differences in TGR5-mediated responses to bile acids and INT777 in neonatal and adult cardiomyocytes
E. Ibrahim, I. Diakonov, C. Williamson, J. Gorelik (London, GB)

28. Cholic acid promotes gut epithelial proliferation in rats exposed to gamma-radiation

29. Vitamin D improves liver histology and hepatic gene expression in a murine obesity/NASH model independently of intestinal Fgf15 expression
D. Jahn, D. Dorbath, S. Kircher, H.M. Hermanns, A. Geier (Würzburg, DE)

30. Characterisation of bile acid pathways in steroidogenic tissues

31. Steroid binding to autotaxin links bile salts and lysophosphatidic acid signalling

32. Vertical sleeve gastrectomy (VSG) in morbidly obese adolescents results in increased fibroblast growth factor 21 (FGF21) that correlates with weight loss

33. Protective role of TGR5 in LCA induced toxic liver damage
C. Klindt, K. Deutschmann, M. Reich, D. Herebian, E. Mayatepek, R. Deenen, K. Köhrer, D. Häussinger, V. Keitel (Düsseldorf, DE)

34. Cholic acid treatment in Zellweger spectrum disorders
F.C.C. Klouwer, K. Berendse, B.G.P. Koot, E.M. Kemper, F. Schaap, H.R. Waterham, F.M. Vaz, M. Engelen, P.M.L. Jansen, R.J.A. Wanders, B.T. Poll-The (Amsterdam, Maastricht, NL)
35. Bile salt and FGF19 signaling in the early phase after liver resection in patients with colorectal liver metastasis

36. Hepatocyte- but not enterocyte-specific FXR deficiency accelerated non-alcoholic steatohepatitis development in mice

37. Bile acid-mediated hepatic differentiation of mesenchymal stem cells
C. Kordes, I. Sawitza, S. Götzte, D. Herebian, M. Castoldi, D. Häussinger (Düsseldorf, DE)

38. The frequent polymorphism PNPLA3 rs738409 increases hepatic steatosis but might protect against gallstone disease
M. Krawczyk, R. Jiménez-Agüero, M.J. Perugorria, L. Gallego, L. Bujanda, F. Lammert, J.M. Banales (Homburg, DE; Warsaw, PL; San Sebastian, ES)

39. Bile acids regulate intestinal wound healing by FXR mediated inhibition of CFTR expression in human colonic epithelial cells
N.K. Lajczak, M.S. Mroz, V. Saint-Criq, S.J. Keely (Dublin, IE)

40. Bile acids regulate colonic epithelial defensin secretion: Implications for pathogenesis and therapy of inflammatory bowel disease
N.K. Lajczak, V. Saint-Criq, M.S. Mroz, A. Perino, F. Murray, K. Schoonjans, S.J. Keely (Dublin, IE; Lausanne, CH)

41. Feedback regulation of autotaxin in mice: Why cholestatic mice don’t scratch
J. Langedijk, D. Tolenaars, R. Bolier, U. Beuers, C. Paulusma, R.P.J. Oude Elferink (Amsterdam, NL)

42. Molecular regulation of adrenal function by bile acids
L. Liu, A. Zaufel, J. Gumhold, E. Krones, G. Zollner, P. Fickert (Graz, AT)

43. Taurocholate induces cyclooxygenase-2 expression via the sphingosine 1-phosphate receptor 2 in a human cholangiocarcinoma cell line
R. Liu, X. Li, L. Zhang, P.B. Hylemon, H. Zhou (Richmond, US; Nanjing, CN)

44. Deletions in the cytoplasmic domain of iRhom1 and iRhom2 promote shedding of the TNF receptor by the protease ADAM17
S.K. Maney, P. Lang (Düsseldorf, DE)

45. ACOX2 deficiency: A new inborn error of bile acid biosynthesis causing persistent hypertransaminasemia

46. TGR5 activation inhibits muscular BCAA catabolism via thyroid hormone activation
T. Miyazaki, A. Honda, T. Ikegami, Y. Matsuzaki (Ibaraki, JP)
47. Raw extract from the Chinese herb Ipomoea stolonifera and its purified components are anti-inflammatory and protect against bile acid-induced apoptosis of rat hepatocytes
H. Moshage, X. Bai, Y. Chen, M. Buist-Homan, G. Shi, K.N. Faber (Groningen, NL; Shantou, CN)

48. Bile acid-dependent regulation of lysosomal biogenesis and function
T. Moustafa, T. Eichmann, K.A. Zierler, H. Wolinski, D. Kolb, J. Gumhold, P. Fickert, M. Trauner (Graz, Vienna, AT)

49. Characterization of bile acid homeostasis during liver regeneration under normal and pathological conditions
M. Mueller, S. Schultze, N. Auer, F. Pauler, M. Trauner (Vienna, AT)

50. Gestational cholestasis is associated with white adipose tissue dysfunction
V. Nikolova, G. Papacleovoulou, E. Bellafante, C. Williamson (London, GB)

51. Bile acid malabsorption patient-reported experiences: Results of an online survey

52. Extrahepatic cholestasis induces large scale alterations in the human liver transcriptome

53. The autophagy inhibitor Rubicon is a direct FXR target in human liver and is induced in human cholestasis
K. Panzitt, H.-U. Marschall, M. Trauner, P. Fickert, M. Wagner (Graz, AT; Gothenburg, SE; Vienna, AT)

54. Antimicrobial remodelling of gut microbiota differentially affects bile acid profile, signalling and enteroprotective response in the ileum and colon of pigs

55. Impact of male cholestasis on the sperm epigenome and consequences for the health of the offspring
V. Pataia, G. Papacleovoulou, L. Poston, C. Williamson (London, GB)

56. Recurrence of progressive familial intrahepatic cholestasis type 2 after liver transplantation with a detection of anti-BSEP antibodies
B. Prusinskas, S. Kathemann, D. Pilic, B. Hegen, P. Küster, V. Keitel, D. Häussinger, R. Büscher, H.A. Baba, P.F. Hoyer, E. Lainka (Essen, Münster, Düsseldorf, DE)

57. Analysis of the bile salt export pump (ABCB11) interactome employing complementary approaches
58. Inactivation of the apical sodium-dependent bile acid transporter (Asbt; Slc10a2) protects against hepatic steatosis in high fat diet-fed mice
   A. Rao, C. Ferrebee, J. Haywood, G. Wynn, W. Zhang, K.D.R. Setchell,
   S.J. Karpen, P.A. Dawson (Atlanta, Winston-Salem, Cincinnati, US)

59. ER-stress regulates bile acid uptake via downregulation of NTCP
   M. Robin, S.F.J. van de Graaf (Amsterdam, NL)

60. Pharmacokinetics, biodistribution and metabolism of obeticholic acid in rats with CCl4-induced decompensated liver cirrhosis
   A. Roda, R. Aldini, S. Spinozzi, P. Franco, M. Cont, A. D'Errico, F. Vasuri,
   A. Degiovanni, L. Adorini (Bologna, IT; San Diego, US)

61. Dual targeting of nuclear receptors ameliorates NAFLD pathogenesis in different dietary murine models
   P.M. Rodrigues, M.B. Afonso, A.L. Simão, M. Caridade, C.C. Carvalho,
   A. Trindade, A. Duarte, P.M. Borralho, M.V. Machado, H. Cortez-Pinto,
   C.M.P. Rodrigues, R.E. Castro (Lisbon, PT)

62. Altered bile acid homeostasis by treatment with glucocorticoids is mediated by interference with FXR/FGF19 ileum-liver crosstalk
   M.R. Romero, F.A. Al-Aqil, M.J. Monte, E. Herraez, R. Rosales, M.A. Serrano,
   F. Jimenez, L. Sanz-Ortega, R. Gonzales, C. Pizarro, J.C. Aranda, B. Ocon,
   I. Uriarte, F. Sanchez de Medina, O. Martinez-Augustin, M.A. Avila,
   J.J.G. Marin (Salamanca, Granada, Pamplona, ES)

63. Inhibiting hepatic bile acid uptake in DDC-induced cholestasis reduces serum biomarkers of liver injury
   R.L.P. Roscam Abbing, D. Slijepcevic, L. Haazen, U. Beuers,
   R.P.J. Oude Elferink, S.F.J. van de Graaf (Amsterdam, NL)

64. Vitamin A deficiency leads to mild cholestasis and a "humanized" bile acid profile in rats
   A. Saeed, M.O. Hoeke, M. Hoekstra, J. Heegsma, H. Moshage, K.N. Faber
   (Groningen, NL)

65. Portal vein embolization-triggered liver regeneration is accelerated by the FXR agonist obeticholic acid
   F.G. Schaap, P.B. Olthof, C. van Himbeeck, F. Huisman, K.P. van Lienden,
   R.F. van Golen, M. Heger, J. Verheij, I.A. Leclercq, P.L.M. Jansen,
   T.M. van Gulik, S.W.M. Olde Damink
   (Maastricht, Amsterdam, NL; Brussels, BE)

66. Oncostatin M contributes to non-alcoholic fatty liver disease (NAFLD) progression in hypercholesterolemic mice
   (Würzburg, DE)

67. Cholestasis reduce immune induction after viral infection
   A.-K. Schupp, S. Rattay, A. Kislat, B. Homey, D. Häussinger, A. Zimmermann,
   D. Graf (Düsseldorf, DE)
68. FXR agonist PX20606 reduces liver damage, fibrosis and portal hypertension in CCl4 cirrhotic rats

69. A mathematical model of the intestinal transit and enterohepatic circulation of bile acids

70. Combined activity of NTCP and OATPs governs hepatic uptake of conjugated bile acids in vivo

71. Postprandial transorgan bile acid kinetics: Implications for TGR5 agonism

72. Novel role for lymphotoxin beta receptor in bile acid homeostasis after partial hepatectomy
   U.R. Sorg, K. Behnke, D. Herebian, M. Reich, E. Mayatepek, V. Keitel, D. Häussinger, K. Pfeffer (Düsseldorf, DE)

73. Generation of liver buds by self-condensation of human iPSC-derived MSCs, HLCs and endothelial cells
   L.-S. Spitzhorn, N. Graffmann, J. Adjaye (Düsseldorf, DE)

74. TGR5 protein expression is reduced in livers of Mdr2-/- (Abcb4 -/-) mice and of patients with primary sclerosing cholangitis (PSC)
   L. Spomer, M. Reich, J. Höhne, J. Hov, T. Karlsen, D. Nierhoff, D. Häussinger, V. Keitel (Düsseldorf, DE; Oslo, NO; Cologne, DE)

75. NorUDCA reduces liver injury and improves the metabolic state in mouse models of obesity and steatosis
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76. Functional studies on monoclonal, patient-derived BSEP-reactive antibodies causing antibody-induced BSEP deficiency (AIBD)
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77. Porphyran, a functional ingredient of “Nori” improves visceral obesity and non-alcoholic fatty liver via inhibition of intestinal FXR activation
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H. Taoka, T. Tanigaki, Y. Takashina, M. Watanabe (Fujisawa, JP)

79. BAs regulate host weight gain and metabolism through gut microbiota modification
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81. Post-hepatectomy dietary challenge with cholic acid to mimic post-resectional liver failure
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K.M.C. van Mierlo, V. Lebrun, C. van Himbeeck, P.L.M. Jansen, F.G. Schaap, S.W.M. Olde Damink, I.A. Leclercq (Maastricht, NL; Brussels, BE)

85. Glycodeoxycholic acid administration increases GLP-1 secretion in healthy humans
F.S. van Nierop, F.G. Schaap, F.M. Vaz, J.A. Romijn, S.W.M. Olde Damink, M.R. Soeters (Amsterdam, Maastricht, NL)

86. Colonization of germ-free mice with a human microbiota induces FXR signaling
A. Wahlström, P. Kovatcheva-Datchary, M. Ståhlman, H.-U. Marschall, F. Bäckhed (Gothenburg, SE; Copenhagen, DK)

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88. Bile acid-modulated transcript-expression in human macrophages validated by transcriptome analysis
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M. Watanabe, K. Ishihara, N. Shimada, M. Kobayashi, Y. Takashina (Fujisawa, Yokohama, JP)

92. UDCA administration in cholestatic pregnancy can ameliorate dysregulated metabolic profile of the fetus and offspring

93. rs10488631 polymorphism of IRF5-TNPO3 confers susceptibility to primary biliary cholangitis (PBC) and is associated with abnormal liver biochemistry indexes: A single centre association study
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## List of Speakers, Moderators and Scientific Organizers

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Opening Hours:
Thursday, June 16, 2016  16.00 – 21.00 h
Friday, June 17, 2016    7.00 – 18.00 h
Saturday, June 18, 2016 8.00 – 12.00 h

Admission to Scientific Events

For admission to scientific events your name badge should be clearly visible.

Congress Report

The official congress report of the Symposium 203 “XXIV International Bile Acid Meeting: Bile Acids in Health and Disease” will be published in English in the second half of 2016 by Karger, Switzerland. Orders for this book at a reduced subscription price of EUR 35,- can be placed at the Congress Office during the congress in Düsseldorf.

Airport

Düsseldorf Airport is about 5 km (10 min) from the Hilton Hotel.
There is also a bus line 729 (towards city center) going from the Airport to the city center. Please get off at station: Theodor-Heuss-Brücke. From here you need to walk about 300 m to the hotel.
Symposium 203

XXIV International Bile Acid Meeting: Bile Acids in Health and Disease

June 17 – 18, 2016
Hilton Düsseldorf
Düsseldorf, Germany

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