

POSTER PRESENTATIONS

- 7 **The role of deltaEF1 family proteins, in the regulation of TGF-beta-induced epithelial-mesenchymal transdifferentiation**
Saitoh, M, Horiguchi, K, Shirakihara, T and Miyazono, K
Molecular Pathology, Tokyo University, 7-3-1 Hongo, Bunkyo-ku, Tokyo, Tokyo 113-0033, Japan
- 8 **Activation of NF-kB by Akt upregulates Snail expression and induces epithelium mesenchyme transition.**
Julien, S*, Puig, I*, Caretti, E, Bonaventure, J*, Nelles, L***, van Roy, F****, Dargemont, C*****, Garcia de Herreros, A*****, Bellacosa, A** and Larue, L***
Developmental genetics of melanocytes, Institut Curie, Batiment 110-Centre Universitaire, Orsay, 91405, France
- 9 **Snail1 and Snail2 dependent epithelial-mesenchymal transition in breast carcinoma cells**
Hugo, HJ*, #Kokkinos, MI, \$Ackland, ML, ^Thompson, EW and Newgreen, DF
** Embryology, Murdoch Childrens Research Institute, Flemington Rd, Parkville, Melbourne, VIC 3052, Australia; #Department of Surgery, Royal Melbourne Hospital, Melbourne, Australia; \$Centre for Cellular and Molecular Biology, School of Biological and Chemical Sciences, Deakin University, Burwood Campus, Burwood, Australia; ^Department of Surgery St. Vincent's Hospital, Melbourne, Victoria, Bernard O'Brien Institute for Microsurgery, Fitzroy, Melbourne, Australia and St. Vincent's Institute of Medical Research, Fitzroy, Melbourne, Victoria*
- 10 **Periphelial blood monocytes induce a phenotypic transition and an increase of a motile activity of human pancreatic carcinoma (HPC-4) cells via secretion of tumour necrosis factor (TNF)-alpha**
Bechyne, I, Baran, B, Sroka, S, Madeja, Z, Siedlar, M* and Czyz, J
*Department of Cell Biology, Faculty of Biochemistry, Biophysics and Biotechnology, Jagiellonian University, ul. Gronostajowa 7, Cracow, 30-387, Poland; *Department of Clinical Immunology, Polish-American Children Hospital, Jagiellonian University Medical College, Cracow, Poland*
- 11 **The role of the Snail family of transcription factors in human carcinomas**
Bram De Craene *, Petra Vermassen * and Geert Berx *
** Department for Molecular Biomedical Research, VIB & Department of Molecular Biology, Ghent University, B-9052 Ghent, Belgium.*
- 12 **Genetic alterations in epithelial and stromal cells of ovarian carcinomas**
Hanna Tuhkanen 1,2,3, Maarit Anttila 1,3, Veli-Matti Kosma 1,4, Seppo Heinonen 3, Matti Juhola 4, Seppo Helisalml 5, Vesa Kataja 2 and Arto Mannermaa 1
1 Institute of Clinical Medicine, Pathology and Forensic Medicine 2 Oncology 3 Obstetrics and Gynecology, University of Kuopio and Kuopio University Hospital, 70210 Kuopio, Finland. 4 Department of Pathology, Jyvaskyla Central Hospital, 40620 Jyvaskyla, Finland. 5 Institute of Clinical Medicine, Neurology, Brain Research Unit, Clinical Research Center / Mediteknia, University of Kuopio, 70210 Kuopio, Finland.

- 13** **Role of p120ctn in cell migration and invasion**
Kuemper, S and Ridley, AJ
Cell Signalling in Invasion and Migration Laboratory, Randall Division of Cell & Molecular Biophysics, King's College London, Guy's Campus, 2nd floor New Hunt's House, London, SE1 1UL, UK
- 14** **Role of epithelium- mesenchymal transition in tissue fibrosis in asthma**
Letuve, S, Maret, M, Grandsaigne, M, Taille, C, Dombret, MC, Aubier, M and Pretolani, M
INSERM U700, Faculte de Medecine Xavier Bichat, and Service de Pneumologie, Hopital Bichat, Paris, France
- 15** **ERK MAPK and NF-kB activation control peritoneal mesothelial cell EMT**
Strippoli R, Benedicto I*, Perez Lozano ML*, Lopez Cabrera M* and Del Pozo MA
*Vascular Biology and Inflammation, CNIC Fundacion Centro Nacional de Investigaciones Cardiovasculares Carlos III, Madrid, Spain; *Unidad de Biologia Molecular, Hospital Universitario de la Princesa, Madrid, Spain*
- 16** **The Protein Tyrosine Phosphatase Pez Regulates TGF-beta, Epithelial-Mesenchymal Transition and Organ Development**
Wyatt, L*^, Wadham, C*, Crocker, LA*, Lardelli, M# and Khew-Goodall, Y*^
**Department of Human Immunology, Hanson Institute, Institute of Medical and Veterinary Science, Frome Road, Adelaide, SA, 5000, Australia; ^Discipline of Biochemistry and #Discipline of Genetics, School of Molecular and Biomedical Science, The University of Adelaide, Adelaide, SA, 5005, Australia*
- 17** **EGF-induced Epithelial-mesenchymal Transition of Human Ovarian Surface Epithelial Cells: Molecular and Signaling Profiles and Possible Role of Amphiregulin**
Huang, RYJ, Ozbun, L, Choi, JH, Salamanca, CM, Pelech, S, Birrer, MJ and Auersperg, N
Dept. Anatomy and Cell Biology, College of Medicine, National Taiwan University, Taipei, Taiwan, Dept. Obstetrics and Gynecology, University of British Columbia, Vancouver, B. C., Canada; Cell and Cancer Biology Branch, NCI, Bethesda, Maryland, USA, Dept. Medicine, University of British Columbia, Vancouver, B.C., Canada, Kinexus Bioinformatics Corp., Vancouver, B.C., Canada.
- 18** **Role of HMGA2 in TGF-beta signaling and cancer progression**
Thuault, S, Tan E-J, Peinado, H*, Cano, A*, Heldin, C-H and Moustakas, A
*TGF-beta Signaling Group, Ludwig Institute for Cancer Research, Box 595, Biomedical Center, 75124 Uppsala, Sweden. * Departamento Biologia Molecular del Cancer, Instituto de Investigaciones Biomedicas "Alberto Sols", C/ Arturo Duperier 4, 28029 Madrid, Spain.*
- 19** **Mechanisms of palatal seam EMT by Transforming Growth Factor (TGF) beta 3**
Shaheen Ahmed, ChangChih Liu and Ali Nawshad
Department of Oral Biology, University of Nebraska Medical Center, 40th and Holdrege, Lincoln, NE 68583, USA
- 20** **Downregulation of polarity factor LGL2 at the invasion front of colorectal carcinomas - a region exhibiting high amounts of nuclear b-catenin**
Simone Spaderna*, Otto Schmalhofer*, Mandy Wahlbuhl#, Dennis Strand∞, Andreas Eger##, Jorgen Behrens§ and Thomas Brabletz*
**Department of Surgery, University of Freiburg, Hugstetter Str. 55, Freiburg, 79095, Germany; #Dept. of Pathology and §Nikolaus-Fiebiger-Center, University of Erlangen; ∞First Dept. of Internal Medicine, Univ. of Mainz; ##Max F. Perutz Laboratories, Medical University Vienna*
- 21** **Genes associated with metastasis and epithelial-mesenchymal transition (EMT)-like phenotype in human colon cancer cells**
Tay, Puei Nam, Tan, Tze Chin, Laban, Mirtha, Leung, Carol Ho-Wing and Hooi, Shing Chuan
Physiology, National University of Singapore, Yong Loo Lin School of Medicine, Blk MD9, 2 Medical Drive, Singapore, Singapore 117597, Singapore
- 22** **Inhibition of TGFbeta-mediated effects by BMPs across the spectrum of breast cancer progression**
Van Overveld, PGM (1), Henriquez, NV (2) and Van der Pluijm, G (1, 2)
(1)Department of Urology, Leiden University Medical Center, Albinusdreef 2, PO Box 9600, 2300 RC, Leiden, The Netherlands; (2) Department of Endocrinology and Metabolic Diseases, Leiden University Medical Center, Albinusdreef 2, PO box 9600, 2300 RC, Leiden, The Netherlands
- 23** **Functional characterization of Snail2 repression complex**
Molina-Ortiz P, McPherson M, Cano A and Portillo F
Universidad Autonoma de Madrid. Dept. Bioquimica, Instituto de Investigaciones Biomedicas "Alberto Sols" (CSIC-UAM), Arturo Duperier 4, Madrid, 28029, Spain
- 24** **Regulation of transcription factor Snail1 through Phosphorylation.**
Matthew MacPherson*, Ihor Yakomovych#, Serhiy Souchelnytskyi#, Paco Portillo* and Amparo Cano*
**Departamento de Bioquimica, Alberto Sols, Universidad Autonoma de Madrid, Calle Arturo Duperier 4, Madrid 28029, Spain; #Institutionen for Onkologi-Patologi, Karolinska institute, Stockholm, Sweden.*

- 25 **New insights in the regulation of E-cadherin and EMT: the role of bHLH factors E2-2 and their relationship with other EMT inducers**
Sobrado, VR, Holt H, Moreno-Bueno G, Portillo F and Cano A
Departamento de Bioquímica, Instituto de Investigaciones Biomedicas Alberto Sols (CSIC-UAM), Arturo Duperier 4, (28029)Madrid, Spain
- 26 **Chronic Allograft Nephropathy: Can TGFbeta-expressing intraepithelial T cells induce EMT?**
Pekalski, M, Robertson, H, Rygiel, K, Al-Hamidi, AH, Burt, AD and Kirby, JA
Applied Immunobiology and Transplantation Research Group, Institute of Cellular Medicine, Newcastle University, Framlington Pl., Newcastle upon Tyne, Tyne and Wear NE2 4HH, UK
- 27 **The Transition of Intrahepatic Biliary Epithelium to Mesenchymal Cells during Chronic Inflammatory Liver Disease.**
Rygiel, KA, Robertson, H, Pekalski, M, Burt, AD*, Jones, DEJ and Kirby, JA**
*Institute of Cellular Medicine, Applied Immunobiology and Transplantation Research Group, Newcastle University, Framlington pl, Newcastle upon Tyne, NE2 4HH, England; *Institute of Cellular Medicine, Applied Immunobiology and Transplantation Research Group, Royal Victoria Infirmary, Department of Pathology, Newcastle University; **Institute of Cellular Medicine, Liver Research Group, Newcastle University*
- 28 **Regulation of EMT during Avian Gastrulation by FGF Signaling and the Receptor Tyrosine Kinase EPHA1.**
Hardy, KM*, Yatskievych, TA*, Konieczka, JH[^] and Antin, PB*[^]
Depts of Cell Biology and Anatomy and Molecular & Cellular Biology[^], University of Arizona, Tucson, AZ, USA*
- 29 **MUC1 and MUC4 epithelial mucins: actors of epithelial-mesenchymal transition?**
Perrais, M, Aubert, S, Hemon, B, Porchet, N, Leroy, X and Van Seuningen, I
INSERM U837 - Jean-Pierre Aubert Research Center, Batiment Biserte, Place de Verdun, Lille cedex, 59045, France
- 30 **Oxidative stress drives Epithelial To Mesenchymal Transition (EMT) in human lung epithelial cells via the TGF-beta pathway**
Nazarowicz, MR, Parker, S, Borthwick, L, Saretzki, GC, Kirby, JA, Corris, PA and Fisher, AJ
Applied Immunobiology and Transplantation Research Group, Institute of Cellular Medicine, Newcastle University, UK
- 31 **Snail and Smad act as co-repressors of coxsackie- and adenovirus receptor in TGF-beta-induced EMT**
Neve, EPA*, Vincent, T*, Kukalev, A#, Moustakas, A, †, Pettersson, RF* and Fuxe, JB
**Ludwig Institute for Cancer Research, Stockholm Branch, Nobels vag 3, Stockholm, SE-17177, Sweden; #Department of Cell and Molecular Biology, Karolinska Institute, SE-17177 Stockholm, Sweden; †Ludwig Institute for Cancer Research Uppsala Branch, SE-75124 Uppsala, Sweden; βDepartment of Anatomy and Cardiovascular Research Institute (CVRI), University of California San Francisco, San Francisco, CA 94143, United States*
- 32 **MUC1 and MUC4 epithelial mucins: actors of epithelial-mesenchymal transition?**
Perrais, M, Aubert, S, Hémon, B, Porchet, N, Leroy, X and Van Seuningen, I
Inserm U837, Place de Verdun, Lille cedex, 59045, FRANCE
- 33 **HLH transcription factors E47 and Id1 in E-cadherin promoter repression and EMT**
Cubillo, E (1), Peinado, H (1), Moreno-Bueno, G (1), Palacios, J (2) and Cano, A (1)
Instituto de Investigaciones Biomedicas (UAM-CSIC), C/ Arturo Duperier, 4, Madrid, 28029, Spain
- 34 **Involvement of NF-kB in embryonic vascular remodelling and endothelial-mesenchymal transition process**
Arciniegas, E, Carrillo, LM and DeSanctis, JB
Lab Estructura y Biología Celular, Servicio Autonomo Instituto de Biomedicina. Universidad Central de Venezuela. , Esq San Nicolas a esq Providencia, San Jose, Caracas, Dto Capital 1010-A, Venezuela
- 35 **EMT of hepatocyte through increase of TGF-b1 and 2 contributes to hepatocyte dysfunction and liver fibrosis**
Wang, JH, Han, JH, Kim, JH, Kim, MD, Lee, JH and Kim, WH
Department of surgery, Ajou University School of Medicine, San5 Wonchun-Dong , Yeongtong-Gu, Suwon, 443-749, Korea
- 36 **Dissecting regulatory networks controlling EMT and mesoderm formation by in vivo RNAi**
Morkel M, Brouwer-Lehmitz A, Liu F, Leushacke M, Werber M and Herrmann BG
Developmental Biology, Max-Planck-Institute for Molecular Genetics, Ihnestr. 73, Berlin 14195, Germany

- 37 **Snail genes are key factors controlling epithelial plasticity and EMT during embryonic development and in the adult.**
Acloque, H
Developmental Neurobiology, Instituto de Neurociencias de Alicante CSIC-UMH, Campus de San Juan, apartado de correo 18, San Juan de Alicante, 03550, Spain
- 38 **Identification of novel genes and signaling networks involved in tumor associated EMT**
Marc Leushacke*, Ralf Spoerle*, Lorenz Neidhardt*, Anne-Kristin Heninger#, Mirko Theis#, Frank Buchholz#, Bernhard G. Herrmann* and Markus Morkel*
**Max Planck Institute for Molecular Genetics, Ihnestr. 73, Berlin 14195, Germany; #Max-Planck-Institute of Molecular Cell Biology and Genetics, Pfotenhauerstr. 108, Dresden 01307, Germany*
- 39 **The small GTPase RhoV is an essential regulator of neural crest induction in Xenopus**
Vignal, E., Guemar, L., de Santa Barbara, P., Donnay, JM., Fort, P. and Faure, S.
CRBM, CNRS UMR 5237, 1919 Route de Mende, Montpellier cedex 5, 34293, France
- 40 **Phosphoproteomic identification of effectors of the breast tumour suppressor Syk**
Larive, RM, Mascre, G, Poncet, J*, Urbach, S*, Jouin, P*, Mangeat, PH, Coopman, PJ and Bettache, N
*CRBM, CNRS-UMR5237, IFR 122, Univ. Montpellier II, Place Eugene Bataillon, CC107, Montpellier Cedex05, 34095, France; *IGF, CNRS-UMR5203, INSERM-U661, IFR 3, Univ. Montpellier I, Univ. Montpellier II, 141 rue de la Cardonille, 34094 Montpellier Cedex 5 FRANCE*
- 41 **Distinct bone morphogenetic protein expression profiles and Smad pathway activation in different phenotypes of experimental canine mammary tumors**
Helena Wensman*, Nils-Erik Heldin**, Gunnar Pejler* and Eva Hellmen*
**Department of Anatomy, Physiology and Biochemistry, Swedish University of Agricultural Sciences, Uppsala, Sweden; **Department of Genetics and Pathology, Uppsala University, Uppsala, Sweden*
- 43 **Overexpression of thioredoxin reductase 1 inhibits Protein Kinase C-dependent induction of phenotypic transition and motility of HEK-293 cells**
Sroka, J*, Antosik, A*, Czyz, J*, Nalvarte, I**, Ollson, JM#, Spyrou, G§ and Madeja, Z*
**Department of Cell Biology, Faculty of Biochemistry, Biophysics and Biotechnology, Jagiellonian University, Gronostajowa 7, Cracow, 30-387, Poland; **Department of Biosciences at Novum, Center for Biotechnology, Karolinska Institute, Huddinge, Sweden; #Department of Molecular Medicine and Surgery, Karolinska Institutet, Karolinska University Hospital, Solna, Stockholm, Sweden; §Foundation for Biomedical Research, Academy of Athens, Athens, Greece*
- 44 **Smad3 is a key mediator of TGFβ-induced transcriptional responses and EMT in mouse mammary epithelial cells**
Joanna Dzwonek, Lena Preobrazhenska, Ann Schellens, Silvia Cazzola, Maarten van Dinther, Anke Klippel, Peter ten Dijke and Kristin Verschuere
Department of Molecular and Developmental Genetics, VIB, K.U.Leuven, Herestraat 49, Box 812, Leuven, 3000, Belgium
- 45 **Snai1 and Snai2 silencing effectively suppresses tumor growth and invasiveness.**
Olmeda, D, Peinado, H, Montes, A, Santos, V, Fabra, A and Cano, A
Instituto de Investigaciones Biomedicas "Alberto Sols", C/Arturo Duperier 4., Madrid, Madrid 28029, Spain
- 46 **The E-cadherin-repressed hNanos1 gene induces tumor cell invasion by upregulating MT1-MMP expression**
Arnaud Bonnomet*,β, Béatrice Nawrocki-Raby*, Kristin Strumane§, #, Christine Gillesβ, Geert Berx§, Frans van Roy§, Myriam Polette* and Philippe Birembaut*
**INSERM UMRS 514, Laboratory of Histology, IFR 53, CHU Maison Blanche, Reims, France; βLaboratory of Developmental and Tumor Biology, University of Liège, CHU Sart-Tilman, B23, Liège, Belgium; §Department for Molecular Biomedical Research, VIB-Ghent University, Ghent, Belgium; #The Netherlands Cancer Institute, Amsterdam, the Netherlands*
- 47 **Regulation of CXCL8/IL-8 by Zonula Occludens-1 during breast tumor-associated epithelial-to-mesenchymal transitions**
Mestdagt M*, Polette M**, Bindels S*, Hunziker W§, Buendia M#, Birembaut P**, Foidart JM* and Gilles C*
**University of Liege, Laboratory of Tumor and Developmental Biology, Liege, 4000, Belgium; **Unité I.N.S.E.R.M. U514, Laboratoire Pol Bouin, I. F. R. 53, C.H.U. Maison Blanche, 51100 Reims, France; §Institute of Molecular and Cell Biology, Epithelial Cell Biology Laboratory, Singapore, Singapore; #Unité I.N.S.E.R.M. U163, Department of Molecular Medicine, Institut Pasteur, 75015 Paris, France.*

- 48 **Positive and Negative Cooperation of p38 MAPK with TGF beta in Epithelial to Mesenchymal Transition (EMT)**
Wang,B and Zhu,HJ
Department of Surgery (RMH), The University of Melbourne, Level 5, Clinical Sciences Building, The Royal Melbourne Hospital, Parkville, Melbourne, VIC 3050, Australia
- 49 **Epithelial-Mesenchymal Transition Induces Migratory/Invasive Phenotype, While Inhibiting Tumor Cell Growth, To Promote Metastasis in Lung Cancer.**
Jun Chen, Rork Kuick, Shalini Anthwal, Gilbert S. Omenn , Theodore J. Standiford and Venkateshwar G. Keshamouni
Internal Medicine/ Pulmonary Division, University of Michigan, 109 Zina Pitcher Place, Ann Arbor, Michigan 48109, USA
- 50 **Mesenchymal to epithelial transition in the establishment of secondary tumours: insights from a bladder carcinoma progression series**
Williams, ED, Chaffer, CL, Blick, T*, Thompson, EW#
*Monash Institute of Medical Research, Monash University, Clayton, Australia; *Department of Surgery (St. Vincent's Hospital), University of Melbourne, Parkville, Australia; #St. Vincent's Institute, Fitzroy, Australia*
- 51 **PTEN repression: a novel mechanism involved in Snail1 induced resistance to apoptosis**
Escriva, MJ, Peiro, S and Garcia de Herreros, A
Unidad de Biología Molecular y Celular; Instituto Municipal de Investigación Médica (IMIM) Barcelona, SPAIN
- 52 **A natural antisense transcript regulates Zeb2/Sip1 gene expression during Snail1-induced epithelial-mesenchymal transition**
Beltran, M*, Puig, I ,#, Alvarez, B* and Gracia de Herreros, A*
** Unitat de Recerca Biologia Celular i Molecular, Institut Municipal de Investigacions Mèdiques (IMIM), Dr Aiguader 83, Barcelona, E-08003, SPAIN; #Developmental Genetics of Melanocytes, UMR 146 , CNRS Institut Curie, Orsay Cedex, France*
- 53 **Snail1 and 1alpha,25-dihydroxyvitamin D3 have opposite effects on Wnt/beta-catenin signaling and gene expression profile in human colon cancer cells**
Larriba, MJ*, Valle, N*, Palmer, HG*, Ordonez-Moran, P*, Alvarez-Diaz, S*, Garcia de Herreros, A^, Gonzalez-Sancho, JM* and Munoz, A*
**Instituto de Investigaciones Biomedicas, Consejo Superior de Investigaciones Cientificas - Universidad Autonoma de Madrid, Madrid, Spain; ^Institut Municipal d'Investigacio Medica - Universitat Pompeu-Fabra, Barcelona, Spain*
- 54 **The Role of Interleukin-like EMT Inducer (ILEI) in Liver Carcinoma Progression**
Lahsnig, C, Mikula, M, Huber, H, Beug, H and Mikulits, W
Department of Medicine I, Division: Institute of Cancer Research, Medical University of Vienna, Borschkegasse 8a, Vienna, Vienna 1090, Austria
- 55 **Modulation of EMT and TGFbeta signal by extracellular matrix components in vivo and in vitro**
Shizuya Saika (1), Toshimitu Uede (2), Shigeyuki Kon (2), Susan R. Rittling (3), David T. Denhardt (3) and Winston Kao (4)
1: Ophthalmology, Wakayama Medical University School of Medicine, 811-1 Kimiidera, Wakayama, 641-0012, Japan; 2: Hokkaido Univ; 3: Rutgers Univ., Piscataway, NJ; 4: Univ. of Cincinnati Med Ctr, Cincinnati, OH.
- 56 **Study of gene activation mediated by Snail1: requirements of the Fibronectin promoter**
Porta de la Riva, M and Agusti, C and Baulida, J
Unitat de Biologia Celular i Molecular, Institut Municipal d'Investigacio Medica, Doctor Aiguader 88, Barcelona, 08003, Spain
- 57 **Oral administration of GW788388, a kinase inhibitor of the TGF beta type I and type II receptors, reduces renal fibrosis in db/db mice**
Maj Petersen
Molecular Cell Biology, LUMC, Einthovenweg 20, Leiden, 2333 RC, The Netherlands;
- 58 **Genipin suppresses fibrogenic behaviors of C₆TN4 lens epithelial cell line.**
Ai Kitano, Osamu Yamanaka, Yuka Okada, Kumi Shirai and Shizuya Saika
Ophthalmology, Wakayama Medical University School of Medicine, 811-1 Kimiidera, Wakayama, 641-0012, Japan

- 59 **NF- κ B mediates matrix metalloproteinase-induced EMT in mammary epithelial cells**
Przybylo, JA, Obr, AE and Radisky, DC
Mayo Clinic Jacksonville, 4500 San Pablo Rd. Griffin Bldg., Rm. 351b Jacksonville, FL 32224
- 60 **Epithelial to Mesenchymal Transition of Mesothelial cells: Pathologic Significance in Peritoneal Dialysis Patients and Potential Therapeutic Interventions**
Lopez-Cabrera, M
Unidad de Biología Molecular, Hospital de la Princesa, Diego de Leon, 62, Madrid-28006, Spain
- 61 **Analysis of the BRCA1 breast tumors identifies a novel oncogene and EMT inducer ~ YAP.**
Smolen, GA, Overholtzer, M, Zhang, J, Muir, B, Sgroi, DC, Deng, CX, Brugge, JS, and Haber, DA
Cancer Center, Massachusetts General Hospital, Harvard Medical School, Bld. 149, 13th Street, Charlestown, MA 02139, USA
- 62 **TRIP6, a novel molecular partner of the MAGI-1 scaffolding molecule, promotes invasiveness**
Larissa Kotelevets¹, Alexey Kruglov¹, Erik Bruyneel², Marc Bracke², Yolande Di Gioia¹, Mary C Beckerle³, Frans van Roy^{4,5} and Eric Chastre¹
¹INSERM, U773, Centre de Recherche Biomedicale Bichat Beaujon CRB3, BP 416, F-75018, Paris, the Université Paris 7 Denis Diderot, site Bichat, BP 416, F-75018, Paris, France; ²Laboratory of Experimental Cancerology, Ghent University Hospital, B-9000 Ghent; ³Huntsman Cancer Institute, Departments of Biology and Oncological Sciences, University of Utah, Salt Lake City, Utah 84102, USA; ⁴Department for Molecular Biomedical Research, VIB, B-9052 Ghent, Belgium; ⁵Department of Molecular Biology, Ghent University; B-9052 Ghent, Belgium
- 63 **Following Snail Trails: A screen for EMT regulators in vivo**
Mary Y.W. Wu and Caroline S. Hill
Developmental Signalling Laboratory, Cancer Research UK, 44 Lincoln's Inn Fields, London, WC2A 3PX, UK
- 64 **Control of TGF β signalling by Smad4 ubiquitination**
Mamidi, A, Dupont, S, Morsut, L, Cordenonsi, M and Piccolo, S
Department of Medical Biotechnologies - University of Padua - Italy
- 65 **ILEI, a novel cytokine essential for tumor progression: does proteolytic processing matter?**
Csiszar, A, Goepfert, A, Waerner, T, Alacakaptan, M, Gal, A and Beug, H
Research Institute of Molecular Pathology, Dr. Bohr-Gasse 7, Vienna, 1030, Austria
- 66 **An inducible system to study gene function in EMT and tumor progression - on the example of ILEI overexpression**
Soelch, S, Beug, H and Csiszar, A
Research Institute of Molekular Pathologie, Dr. Bohr Gasse 7, Vienna, Austria 1030, Austria
- 67 **Chemokines: Key players in function of the Interleukine-like EMT Inducer (ILED)?**
Gal, A, Alacakaptan, A, Csiszar, A and Beug, H
Radiation Oncology and Biology, Radiobiology Research Institute, Churchill Hospital, Headington, Oxford, OX3 7LJ, United Kingdom
- 96 **Characterization of signal transduction pathways regulating Snail in primary human endometrial carcinoma**
Hipp, S, Becker, KF
Insitut fuer Pathologie, Technische Universitaet Muenchen, Trogerstrasse 18, Muenchen, Germany 81675
- 97 **Ha-ras induced EMT in human colon cells: microarray analysis and transcriptional regulation of vimentin and S100A4 genes**
Andreolas, V, Kalogeropoulou, M, Voulgari, A, Madziar, B, Makrodouli, E, Roberts, M, Joyce, T, Pintzas, A
Lab Signal Mediated Gene Expression, Inst Biol Res Biotech, National Hellenic Research Foundation, 48, Vas Constantinou Avenue, Athens, 116 35, Greece
- 98 **A Snail-Smad transcriptional repressor complex promotes TGF-beta-mediated epithelial-mesenchymal transition**
Vincent, T, Neve, EPA, Kukalev, A, Pietras, K, Moustakas, A, Virtanen, I, Pettersson, RF
Ludwig Institute for Cancer Research, Stockholm Branch, Karolinska Institute, Nobels vag 3, Stockholm, 171 77
- 99 **p53 is a novel regulator of EMT**
Laureline Roger, Veronique Gire and Pierre Roux
CRBM, CNRS, Montpellier, France
- 100 **Control of Rho/ROCK signalling by p53:Consequences on cell migration and invasion.**
Gadea Gilles, Roger Laureline, Anguille Christelle, Vinot Stephanie and Roux Pierre
CRBM, CNRS, 1919, route de Mende, Montpellier, France 34293, France

END OF POSTER PRESENTATIONS

~ LATE-BREAKING POSTERS ~

The 3rd Epithelial-Mesenchymal Transition (EMT) Meeting

An EMBO Workshop



Co-organized by TEMTIA and the
Marie-Curie Epiplasticarcinoma EU-RTN Network

101 INTERPLAY BETWEEN EGFR-TKI RESISTANCE MECHANISMS, EMT BIOLOGY AND RATIONALE FOR MULTI-TARGETING IN CANCER

Kan, JLC, Epstein, D, Thomson, S, Miglarese, M, Buck, E and Eyzaguirre, A

Cancer Biology, OSI Pharmaceuticals, 1 Bioscience Park Drive, Farmingdale, New York 11735, USA

The receptor for epidermal growth factor (EGFR) is overexpressed in a wide range of cancers. EGFR can utilize both the MAPK and Akt signaling pathways to confer increased proliferation and cell survival. EGFR inhibitors such as erlotinib can downregulate the activities of MAPK and Akt pathways only in tumor cells that are sensitive to growth inhibition. Biomarkers have been identified that differentiate erlotinib responsive from non-responsive pancreatic and NSCLC tumors. These studies established a strong correlation between the expression of epithelial markers and sensitivity to growth inhibition by erlotinib; tumor cells that have undergone the epithelial to mesenchymal transition being less sensitive to erlotinib.

With the ability to trigger Akt signaling by multiple mechanisms, identifying cooperative relationships to augment cell growth would have clinical utility. A cooperative relationship between EGFR and IGF-1R toward regulation of cell growth has been previously described, and mTOR signaling downstream of Akt has been shown to stimulate cell proliferation, suggesting the potential for both IGF-1R and mTOR to modulate the sensitivity toward EGFR antagonists. We observed synergistic growth inhibition of tumor cells treated with the combination of erlotinib and either an IGF-1R or mTOR inhibitor. These results support the potential clinical utility for combining an IGF-1R inhibitor or rapamycin with erlotinib, especially for NSCLC and pancreatic cancer.

102 SNAIL AND SMAD ACT AS CO-REPRESSORS OF COXSACKIE- AND ADENOVIRUS RECEPTOR INTGF-BETA-INDUCED EMT

Neve, EPA, Vincent, T, Kukalev, A, Moustakas, A, Pettersson, RF and Fuxe, J

Department of Cell and Molecular Biology (CMB), Karolinska Institute, von Eulers v. 3, Stockholm, 17177, Sweden

103 ERK5 CONTROLS SLUG EXPRESSION DURING WOUND HEALING.

V. Arnoux, M. Nassour, R. Hipskind and P. Savagner.

INSERM U868, CRCM, CRLC Val d'Aurelle, Montpellier, France.

Reepithelialization during cutaneous wound healing involves numerous signals resulting in basal keratinocyte activation, spreading and migration linked to a loosening of cell-cell adhesion structures. The transcription factor Slug is required for this process, and EGF treatment of humankeratinocytes induced activating phosphorylation of Erk5 that coincides with slug transcriptional activation. Accordingly, ectopic activation of Erk5 led to increased Slug mRNA levels and faster wound healing, while keratinocyte migration was totally blocked by inhibition of the Erk5 pathway. Transfection of a vector expressing a shRNA specific for Erk5 strongly diminished Erk5 levels in keratinocytes and blocked their mitogenic response to EGF, along with induction of Slug expression. These Erk5-deprived keratinocytes showed an altered, more compact morphology. Consistent with this, cellular actin was found primarily in the subcortical region, linked to adherens junctions, instead of in lamellipodia and stress fibers. Similarly, we found that embryonic fibroblasts from Erk5- and Mek5-knockout mice show a dramatic rearrangement of the actin cytoskeleton with a loss of thick actin cables. In isolated cells, focal contacts remained sparse and failed to aggregate in cytoplasmic extensions. These results implicate a novel EGFR/Erk5/Slug pathway in controlling cytoskeleton organization and cell motility in keratinocytes treated with EGF.