ABSTRACT BOOK

17th ECCO - 38th ESMO - 32nd ESTRO
European Cancer Congress
Reinforcing multidisciplinarity
AMSTERDAM, 27 SEPTEMBER - 1 OCTOBER 2013

www.ecco-org.eu
Disclaimer

All advertising material in this publication is expected to conform to ethical (medical) standards, and does not constitute a guarantee or endorsement of the quality or value of such product or the claims made of it by its manufacturer, and is intended for prescribing healthcare professionals only.

No responsibility is assumed by the Publisher for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical sciences, in particular, independent verification of diagnoses and drug dosages should be made.
European Journal of Cancer

Editor-in-Chief: Alexander M.M. Eggermont
Institut Gustave Roussy
Villejuif, France

Editors:
Basic and Preclinical Research: Richard Marais, Manchester, UK
Giorgio Parmiani, Milan, Italy

Drug Development: Jean-Charles Soria, Villejuif, France

Early Breast Cancer: Kathleen I. Pritchard, Toronto, Canada

Advanced Breast Cancer: David Cameron, Edinburgh, UK

Gastrointestinal Cancers: Eric Van Cutsem, Leuven, Belgium
Michel D'Urso, Villejuif, France

Genitourinary Cancers: Cora Sternberg, Rome, Italy

Lung Cancer: Mary O’Brien, London, UK

Gynaecological Cancers: Ignace Vergote, Leuven, Belgium

Head and Neck Cancer: Kevin Harrington, London, UK

Sarcomas: Jean-Yves Blay, Lyon, France

Melanoma: Dirk Schadendorf, Essen, Germany

Neuro-oncology: Roger Stupp, Zurich, Switzerland

Epidemiology and Prevention: Jan Willem Coebergh, Rotterdam, The Netherlands

Paediatric Oncology: Rob Peter, Rotterdam, The Netherlands

Founding Editor: Henri Tagnon

Past Editors: Michael Peckham, London, UK; Hans-Jörg Senn, St Gallen, Switzerland; John Smyth, Edinburgh, UK

Editorial Office: Elsevier, The Boulevard, Langford Lane, Kidlington, Oxford OX5 1GB, UK
Tel: +44 (0) 1865 843590, Email: ejcancer@elsevier.com

EDITORIAL BOARD

CLINICAL ONCOLOGY

J.-P. Armand (France)  G. Ferrandina (Italy)  P. O’Dwyer (USA)
A. Ayhan (Japan)  H. Gabra (UK)  J. Overgaard (Denmark)
R. Blamey (UK)  H. Gelderblom (The Netherlands)  N. Pavlidis (Greece)
M. Bolla (France)  B. Hassan (Belgium)  J. Perry (Canada)
J. Boyages (Australia)  J.C. Horiot (Switzerland)  P. Price (UK)
N. Brünner (Denmark)  C. Huber (Germany)  D. Raghavan (USA)
F. Cardoso (Portugal)  R. Jakesz (Austria)  J. Ringash (Canada)
J. Cassidy (UK)  J. Jassim (Poland)  J. Robert (France)
M. Castiglione (Switzerland)  D. Jedrell (UK)  A. Rody (Germany)
L. Cataliotti (Italy)  V.C. Jordan (USA)  D. Sargent (USA)
L. Cheng (USA)  A. Katz (Brazil)  M. Schmidinger (Austria)
H. Cody (USA)  M. Kaufmann (Germany)  S. Sleijfer (The Netherlands)
R. Coleman (UK)  I. Kunkler (UK)  P. Sonneveld (The Netherlands)
A. Costa (Italy)  L. Lindner (Germany)  A. Sparreboom (USA)
J. De Boni (UK)  P.E. Lannning (Norway)  M. van den Bent (The Netherlands)
M.J.A. De Jong (The Netherlands)  P. Lorigan (UK)  M. van Glabbeke (Belgium)
E. de Vries (The Netherlands)  K. McDonald (Australia)  G. Velikova (UK)
A. Dicker (USA)  R. Mertelsmann (UK)  U. Veronesi (Italy)
R. Dummer (Switzerland)  F. Meunier (Belgium)  A. Vincent-Salomon (France)
F. Eisinger (France)  T. Mok (Hong Kong)  A. Voogd (The Netherlands)
S. Enridge (UK)  D. Nam (Korea)  E. Wörsching (Canada)

BASIC, PRECLINICAL AND TRANSLATIONAL RESEARCH

A. Albini (Italy)  A. Gescher (UK)  A. Puisieux (France)
P. Allavena (Italy)  R. Giavazzi (Italy)  V. Rotter (Israel)
F. Balkwill (UK)  I. Hart (UK)  M. Schmitt (Germany)
M. Barbacid (Spain)  W. Keih (UK)  C.G. Sweep (The Netherlands)
M. Broggiini (Italy)  L.A. Kiemeney (The Netherlands)  G. Taraboletti (Italy)
C. Catapano (Switzerland)  J. Lunec (UK)  P. Vineis (UK)
J. Collard (The Netherlands)  D.R. Newell (UK)  N. Zaffaroni (Italy)
E. Garattini (Italy)  G.J. Peters (The Netherlands)

EPIDEMIOLOGY AND PREVENTION

B. Armstrong (Australia)  A. Green (Australia)  S. Sanjose (Spain)
P. Autier (France)  K. Hemminki (Germany)  M.K. Schmidt (The Netherlands)
J.M. Borrás (Spain)  C. Johansen (Denmark)  H. Storm (Denmark)
C. Bosetti (Italy)  L.A. Kiemeney (The Netherlands)  L.Y. van der Poll-Franse (The Netherlands)
J. Faire (France)  M. Maynadie (France)  H.M. Verkooijen (The Netherlands)
S. Franceschi (France)  H. Møller (UK)  R. Zanetti (Italy)
D. Forman (France)  P. Feeters (The Netherlands)

PAEDIATRIC ONCOLOGY

C. Bergeron (France)  G. Chantada (Argentina)  L. Sung (Canada)
A. Biondi (Italy)  F. D’Oz (France)  M. van den Heuvel-Eibrink (The Netherlands)
E. Bouffet (Canada)  A. Ferrari (Italy)  M. van Noesel (The Netherlands)
M. Cairo (USA)  M.A. Grootenhuis (The Netherlands)
H. Caron (The Netherlands)  K. Pritchard-Jones (UK)
Acidic conditions as well as hyperthermia were identified as
we generated mouse strains with combined
Here we study the role of MCs in the gp130
2
Endometrial cancer cell lines were cultured
Moreover, the significant differences between methylation
1
Endometrial cancer represents the most common gynecological
and
1
ESR1, CDH1, RASSF1A, SYK, BRMS1
Mast cells (MC) are innate immune cells, which are
DNA methylation profiles observed in our group of breast
genes. Moreover, the significant differences between methylation
1
The obtained results clearly indicate the regulatory effects
results were determined by Western blot and immunocytochemistry.

Aim: To assess whether NFATc1 controls transcription of EMT genes and stemness in PDAC, particularly upon p53 inactivation.

Materials and Methods: We generated mouse strains with combined pancreas-specific expression of NFATc1, p53(−/−) and Kras(−/−) using Cre-Lox technology. These mice showed a highly aggressive tumor growth (median survival of <50 days). Mouse primary tumour cells were used to identify NFATc1 targets by gene expression profiling and pathway analyses (ChIP seq, miRNA analyses and GSEA). NFATc1 mediated EMT and stemness were assessed in human and murine pancreatic cancer models using migration and spheroid assay as well as xenograft mouse models.

Results: Here, we identified antitumor NFATc1 and p53 signaling pathways in transcriptional control over EMT and stemness. We show that p53 activation prevents cells from EMT in a miR200 dependent manner. However, disruption of the tumor suppressor pathway enables NFATc1/Sox2 chromatin complex formation and transcription of EMT programmes, resulting in highly invasive and metastatic PDACs. Finally, re-expression of miR200c or NFATc1 inactivation suppresses EMT/stemness genes and re-sensitizes PDAC to chemotherapy.

Conclusion: Antitumor NFATc1 and p53 signaling pathways control features of cellular plasticity and tumor progression at the level of gene transcription. These findings implicate key roles for NFATc1 in transcriptional regulation of differentiation and self-renewal in PDAC.

No conflict of interest.