



Press Information

Welcome to the premier global congress on gastrointestinal cancer. As our guest, you will have the opportunity to interact with some of the world's leading authorities in the study and treatment of gastrointestinal cancers. More than 60 expert specialists from a range of disciplines will participate in the scientific program and their presentations will provide delegates with information on the latest developments in basic and clinical research. Their consensus report on the treatment of hepatocellular carcinoma will bring the Congress to a close. Members of the press are invited to attend all scientific sessions and symposia at no charge. Below you will find information that will assist you with your reporting of the Congress.

General Congress Information

The ESMO International Symposium: 10th World Congress on Gastrointestinal Cancer has been organized in partnership with ESMO (European School of Medical Oncology). The Congress is developed and managed by Imedex®, LLC.

Societies and Associations

This Congress is endorsed by the following societies and associations:

ENETS (European Neuroendocrine Tumor Society)

EONS (European Oncology Nursing Society)

EORTC (European Organisation for Research and Treatment of Cancer: Gastrointestinal Tumor Group)

ESO (European School of Oncology)

ESSO (European Society of Surgical Oncology)

Europacoln

Congress Dates

Wednesday 25 June – Saturday 28 June 2008.

Congress Location

All scientific sessions will be held at The International Convention Center of Barcelona (Centre de Convencions Internacional de Barcelona) – CCIB

Registrations

Close to 3,500 participants are expected for the scientific gathering, representing nearly 100 countries around the world.

Language

The Congress language is English. No translation is provided.

Scientific Agenda

Unless otherwise noted, all lectures are in the General Session Room, Level 0, room 041

- Clinically-focused with a multidisciplinary approach
- Overview lectures by widely recognized experts
- Poster discussion sessions, abstract presentations
- Meet the Expert sessions
- Dedicated nursing session
- Dedicated session for young medical oncologists
- Satellite Symposia

Scientific Sessions:

- Cancer of the pancreas
- Cancer of the liver

- Rare tumors: Neuroendocrine tumors and gastrointestinal stromal tumors (GIST)
- Esophageal cancer
- Gastric cancer
- Colorectal cancer: Prevention, screening and nutrition
- Colorectal cancer: Surgery
- Imaging in gastrointestinal cancer
- Metastatic colorectal cancer
- Adjuvant therapy for colon cancer
- Report on Expert Discussion: Hepatocellular carcinoma

The complete scientific agenda is available online at www.worldgicancer.com/WCGI/WGIC08/sp.html.

Abstracts

405 abstracts have been accepted for presentation at the ESMO International Symposium: 10th World Congress on Gastrointestinal Cancer®, representing the latest research from across the globe in colon, esophageal, gastric, liver, pancreatic and rectal cancer. All accepted abstracts are published in a special *Annals of Oncology* supplement. A summary of featured abstracts will be posted online during the week of 16 June. **All abstract details are embargoed until Wednesday, 25 June 2008 at 15:00.**

Abstract Poster Hours

Exhibition Hall, CCIB Level 0, Rooms 021-031

Thursday, 26 June 2008	9:00 – 19:00
Friday, 27 June 2008	9:00 – 18:15
Saturday, 28 June 2008	9:00 – 15:00

Travel Award Winners

A limited number of abstract travel awards for participants from developing nations have been awarded. A ceremony will be held on Friday, 27 June 2008 at 12:50 in the General Session to present these awards.

Exhibition

As an integral part of the Congress, companies and organizations will display their products and services. The exhibition is located on Level 0, Rooms 021-031, of the Centre de Convencions Internacional de Barcelona (CCIB).

Thursday, 26 June 2008	9:00 – 19:00
Friday, 27 June 2008	9:00 – 18:15
Saturday, 28 June 2008	9:00 – 15:00

Corporate Satellite Symposia

Thursday, 26 June 2008

12:45 - 14:15

Level 1, Rooms 111 - 112

Luncheon Symposium: Supported by Nestle Nutrition, "Proactive nutrition in GI cancer patients"

Level 1, Rooms 113 - 115

Luncheon Symposium: Supported by Sirtex Medical Europe GmbH, "Integrating radioembolisation (intra-arterial brachytherapy) into the treatment paradigms for liver dominant colorectal metastases"

19:15 - 20:45

Level 1, Rooms 116 – 117

Evening Symposium: Supported by F. Hoffmann-LaRoche, "Expanding options in GI cancer with Xeloda and Tarceva"

Friday, 27 June 2008

13:15 - 14:45

Level 1, Rooms 111-112

Luncheon Symposium: Supported by Merck Serono, "ERBITUX – a new era in metastatic colorectal cancer"

Level 1, Rooms 116 - 117

Luncheon Symposium: Supported by F. Hoffmann-LaRoche, "Avastin-based therapy: moving towards a better future in metastatic CRC management"

18:30 - 20:00

Level 1, Rooms 116 - 117

Evening Symposium: Supported by sanofi-aventis, "S-1: Updates and perspectives on a targeted designer drug"

Saturday, 28 June 2008

13:15 - 14:45

Level 1, Rooms 113 - 115

Luncheon Symposium: Supported by the European School of Oncology, "The ESO colorectal cancer observatory: A look into the future"

Level 1, Rooms 116 – 117

Luncheon Symposium: Supported by Amgen, "KRAS: The first clinical biomarker for individualized treatment in mCRC"

Onsite Resources

Press Room

International Conventional Center of Barcelona (CCIB), Entrance Hall

Hours:

13:00 – 20:00 Wednesday, 25 June

8:00 – 20:00 Thursday, 26 June – Friday, 27 June

8:00 – 17:00 Saturday, 28 June

Fax and internet access will be available. Congress materials, scientific highlights, ESMO information, information on Congress representatives, and general information on gastrointestinal cancer will be available. The press room will be staffed on an as needed basis.

Access

Press registration includes full access to all scientific sessions, satellite symposia and the exhibit hall. Registered press will receive a complete Congress bag. Social events are not included with press registration.

Contacts

Among the specialists available for interviews are the co-chairs of the Congress – Mario Dicato, MD, of the Luxembourg Medical Center in Luxembourg and Eric Van Cutsem, MD, PhD, of the University Hospital Gasthuisberg in Leuven, Belgium. Christopher Bolwell, Senior Director of Medical Affairs for Imedex is also available to speak regarding the Congress. Please let us know in advance if you are interested in speaking with any of the Congress faculty including the co-chairs. Elke Koscher, Project Manager, is available onsite and will be able to facilitate your interviews. She can be contacted by telephone at (+1) 404-446-7100.

Press Briefings

No daily press briefings are scheduled.

Guidelines for Industry/Supporters

Industry/Supporter Press Conferences:

Press briefings by industry/supporters will be allowed at the ESMO International Symposium: 10th World Congress on Gastrointestinal Cancer. In order to avoid any competing activities that could deter a significant number of attendees/faculty from participating in the program, the following policies will apply:

1. They must be narrowly targeted (i.e. less than 20 people in attendance)
2. They may not otherwise interfere with World Congress activities (i.e. noise, large camera crews, etc.) or be held during the Congress plenary sessions.
3. They may not be held in the designated Press room at the Convention Center, in order to allow unrestricted access to that room, but may be held in a separate room or offsite.
4. Imedex must be notified of the time and location of the briefing in advance of the event

Press briefings may be held during a satellite symposium if the above 4 guidelines are met. Other time slots will be considered if the same guidelines will be met.

Other Imedex Meetings of Interest

2nd Conference – World Congress on Gastrointestinal Cancer: Asian Perspectives

22-23 August 2008 – Bangkok, Thailand

Chairs: Eric Van Cutsem, MD, PhD and Yoon-Koo Kang, MD

9th Annual Perspectives in Colorectal Cancer

19-20 September 2008 – Miami, Florida, USA

Chairs: Richard M. Goldberg, MD and Mace L. Rothenberg, MD

Perspectives in Melanoma XII

2-4 October 2008 – Scheveningen/The Hague, The Netherlands

Chairs: Alexander M.M. Eggermont, MD, PhD and John M. Kirkwood, MD

Regional Co-Chairs: Alan Spatz, MD and Nelleke Gruis, PhD

Lymphoma & Myeloma 2008: An International Congress on Hematologic Malignancies

16-18 October 2008 – New York, New York, USA

Chair: Morton Coleman, MD

Co-Chairs: Richard R. Furman, MD, John P. Leonard, MD and Ruben Niesvisky, MD

2008 Advances in Inflammatory Bowel Diseases

Crohn's & Colitis Foundation's Clinical & Research Conference

4-7 December 2008 – Hollywood, Florida, USA

Chairs: Richard P. MacDermott, MD and Stephen Hanauer, MD

Website: www.AdvancesinIBD.com

6th European Congress: Perspectives in Gynecologic Oncology

30-31 January 2009 - Nice, France

Chairs: Andreas du Bois, MD, PhD, Peter G. Harper, MD, FRCP, Stanley B. Kaye, MD, and Ignace Vergote, MD, PhD

5th European Congress on Hematologic Malignancies: From Clinical Science to Clinical Practice

13-15 February 2009 – Munich, Germany

Chairs: Bertrand Coiffier, MD and Eva Kimby, MD, PhD

10th International Symposium on Febrile Neutropenia

20-21 February 2009 – Valencia, Spain

Chairs: Jean A. Klastersky, MD, PhD and Miguel Sanz, MD, PhD

ESMO Conference: 11th World Congress on Gastrointestinal Cancer

24-27 June 2009 – Barcelona, Spain

Chairs: Mario Dicato, MD and Eric Van Cutsem, MD

Website: www.worldgicancer.com

View a complete list of upcoming meetings at www.imedex.com.

Contact Us

For questions, please contact the Congress organizer:

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 **ESMO International Symposium**



10TH WORLD CONGRESS ON

Gastrointestinal C A N C E R

FOR IMMEDIATE RELEASE

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Congress Celebrates 10 Years with Endorsements from Leading Professional Societies and Organizations

Endorsements Demonstrate Impact of World Congress on Gastrointestinal Cancer[®]

Barcelona, Spain 16 June 2008 – As it celebrates its 10th year, the *ESMO International Symposium: 10th World Congress on Gastrointestinal Cancer[®]* has received endorsements from 6 leading professional societies and organizations, solidifying the reputation of the Congress as the premier platform for specialists in cancer research, leading oncologists and practicing clinicians to review the state-of-the-art in gastrointestinal cancer and share the latest information on its multidisciplinary management. The European Neuroendocrine Tumor Society (ENETS), European Oncology Nursing Society (EONS), European Organisation for Research and Treatment of Cancer (EORTC): Gastrointestinal Tumor Group, European School of Oncology (ESO), European Society of Surgical Oncology (ESSO), and Europacoln have indicated their support for the high quality of the scientific agenda and its educational value to oncology professionals. The European Society for Medical Oncology (ESMO) began its association with the Congress in 2005, and in 2008 brought the Congress under its educational wing as a partnership meeting.

In recognition of its 10 year history, Congress co-chair Eric Van Cutsem observed “We have much to celebrate from the past years. The number of deaths from colorectal cancer has been dropping as treatment has improved, allowing for

more effective options for people with this diagnosis. However, there is still much work to be done. We are proud of our tradition of excellence in educating the medical community and look forward to continuing the journey.”

The *ESMO International Symposium: 10th World Congress on Gastrointestinal Cancer*[®], developed and managed by Imedex, begins next week in Barcelona, Spain, running from 25-28 June 2008. More than 3,300 healthcare providers from nearly 100 countries are expected to attend. The internationally recognized faculty will include 68 experts, chaired by Mario Dicato, MD of the Luxembourg Medical Center in Luxembourg and Eric Van Cutsem, MD, PhD of the University Hospital Gasthuisberg in Leuven, Belgium.

Designed to promote a multidisciplinary approach to treatment, the scientific agenda offers targeted sessions for oncology surgeons and nurses as well as a comprehensive range of topics for researchers, gastroenterologists, and medical and radiation oncologists. Presentation of cutting edge research has become one of the highlights of the Congress, as more than 400 abstracts have been accepted for publication in a special supplement of *Annals of Oncology*.

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About Imedex

Imedex, LLC is an industry leader in providing accredited, independent continuing medical education (CME) worldwide. Imedex develops high quality, scientific programs that translate the latest research into clinically relevant information, improving patient care through education. The programs have a proven sustained impact on disease management, exceptional organization, and outstanding educational value. With 13,000 live meeting attendees and 120,000 e-learning experiences annually, Imedex educates the global healthcare community. Live, enduring, and e-learning programs focus on impacting the clinical management of patients around the world in the therapy areas of oncology, hematology, gastroenterology, infectious disease, urology, and psychiatry. Attendees consistently rate Imedex programs as excellent in terms of scientific quality, execution, and educational value.

For more information about Imedex, visit www.imedex.com.

About the European Society for Medical Oncology (ESMO)

The European Society for Medical Oncology (ESMO) is the leading European non-profit, professional organization for medical oncology with a focus on promoting multidisciplinary cancer treatment around the world. ESMO unites medical oncologists, oncology specialists, healthcare professionals, caregivers, patients and policy makers in a global alliance committed to eradicating cancer and ensuring equal access to high quality treatment for all patients. Thanks to its state-of-the-art education and training programs, ESMO plays an instrumental role in providing the oncology community with the most up-to-date scientific research and information available. Through its flagship journal, *Annals of Oncology*, ESMO publishes articles on all aspects of clinical oncology. ESMO is dedicated to educating and supporting oncologists, optimizing patient care, disseminating cancer-specific information to the public, and advocating patient rights. As an authoritative voice in the fight against cancer, ESMO provides both the platform and the consultative expertise to influence national and international organizations as well as European authorities, in order to establish common standards for a multidisciplinary approach to cancer treatment.

For more information about ESMO, visit www.esmo.org



Scientific Highlights

Travel Award Winners

Based on the scientific merit of their abstract submissions, grants for participants from developing nations have been awarded. A ceremony will be held Friday, 27 June 2008 at 12:50 in the General Session to present these awards. We would like to congratulate the following winners:

- Dilshod Egamberdiev, Uzbekistan
- Nanda Rajaneesh Karinagashetty, India
- Nataliya Kitsera, Ukraine
- Nazar Lukavetsky, Ukraine
- Subrata Saha, India
- Gagan Saini, India
- Ben Selvan, India
- Shantanu Sharma, India
- Mehrnoosh Tashakori, Iran
- Abrorjon Yusupbekov, Uzbekistan

Top Abstracts

(Note: All abstract details are embargoed until 25 June 2008 at 15:00)

The following abstracts have been selected by the Scientific Committee and the Congress Chairs as the Top Abstracts for the ESMO International Symposium: 10th World Congress on Gastrointestinal cancer[®].

1. O-009. Analysis of axitinib (AG-013736) plus gemcitabine in patients with advanced pancreatic cancer: Diastolic blood pressure as biomarker of clinical benefit

Spano J¹, Maurel J², Wasan H³, Bycott P⁴, Liau K⁴, Pithavala Y⁴, Garrett M⁴, Ricart A⁴, Kim S⁴, Chodkiewicz C⁵

¹Hopital de la Pitie Salpetriere, Paris, France, ²Hospital Clinic I Provincial de Barcelona, Barcelona, Spain, ³Hammersmith Hospital, London, United Kingdom, ⁴Pfizer La Jolla, CA, United States, ⁵Moffitt Cancer Center, Tampa, FL, United States

Background: Axitinib is an oral, potent and selective inhibitor of VEGF receptors 1, 2, 3. A randomised phase 2 study of gemcitabine (GEM) + axitinib (GA) versus GEM (2:1 randomisation), was conducted in patients with advanced pancreatic cancer. This analysis explored the relationship between axitinib concentration, survival and diastolic blood pressure (dBp).

Methods: Eligibility criteria included unresectable locally advanced or metastatic pancreatic cancer, no prior systemic therapy, satisfactory bone marrow, renal and hepatic function, BP \leq 140/90 mmHg at baseline and ECOG PS 0–2. Patients (n=103) received GEM 1000 mg/m² infused over 30 minutes on days 1, 8 and 15 every 4 weeks. Axitinib 5 mg orally BID was given continuously to patients randomised to the GA arm (n=69). Full pharmacokinetic profiles were obtained (n=6) on cycle (C) 1 day (D) 1 (GEM alone), C1D14 (steady-state axitinib alone), and C1D15 (GEM + steady-state axitinib) in a phase 1 assessment that preceded phase II. Blood pressure was monitored weekly in the clinic. Antihypertensive medications and/or axitinib dose modifications were used to manage BP as pre-specified in the protocol. Median overall survival (OS) was estimated using the Kaplan-Meier method.

Results: Pharmacokinetic parameters of GEM were similar in the presence of axitinib. The mean (%CV) plasma AUC for GEM was 11348 (64) vs 12840 (20) ng.h/mL in the presence and absence of axitinib; similarly the AUC for the GEM metabolite, dFdU, was 184511 (24) vs 195051 (25) ng.h/mL respectively. Plasma profiles and PK parameters for axitinib were similar in the presence and absence of GEM; mean AUC₀₋₂₄ 270 (41) vs 252 (30) ng.h/mL. Among the 69 patients receiving GA, the median OS and 1-year survival

was 12.2 months and 51% respectively for patients experiencing dBP ≥ 90 mmHg (n=38) versus 5.2 months and 19% respectively for those without any dBP ≥ 90 mmHg (n=31). The median OS and 1-year survival in patients receiving GEM alone (n=34) was 5.6 months and 23% respectively. Elevations in dBP were mostly transient and managed with the use of antihypertensive medications and/or axitinib dose reductions.

Conclusion: GA pharmacokinetics were similar when administered in combination. Preliminary analysis of patients with advanced pancreatic cancer treated with GA indicates that patients with dBP ≥ 90 mmHg appear to be associated with longer survival.

2. O-012. Long-term survival in a phase III trial of sunitinib in imatinib-resistant/intolerant gastrointestinal stromal tumor with novel statistical analysis to account for crossover

Garrett C¹, Huang X², Casali P³, Schöffski P⁴, Blackstein M⁵, Shah M⁶, Verweij J⁷, Tassel V², Baum C², Demetri G⁸

¹H Lee Moffitt Cancer Center and Research Institute, FL, United States, ²Pfizer Global Research and Development, La Jolla, CA, United States, ³Istituto Nazionale Tumori, Milan, Italy, ⁴University Hospital Gasthuisberg, Leuven, Belgium, ⁵Mount Sinai Hospital and the University of Toronto, Toronto, Ontario, Canada, ⁶Ohio State University Comprehensive Cancer Center, Columbus, OH, United States, ⁷Erasmus University Medical Centre, Rotterdam, The Netherlands, ⁸Ludwig Center at Dana-Farber/Harvard Cancer Center, Boston, MA, United States

Background: Sunitinib malate (SUTENT[®]) is an oral multitargeted tyrosine kinase inhibitor of KIT, PDGFRs, VEGFRs, FLT3, CSF-1R and RET, approved multinationally for treatment of advanced imatinib-resistant or imatinib-intolerant gastrointestinal stromal tumor (GIST). A phase III, double-blind, placebo-controlled, multicenter trial assessed the safety and efficacy of a 50-mg starting dose of sunitinib delivered orally in 6-week cycles comprising 4 weeks on treatment followed by 2 weeks off treatment in patients with imatinib-resistant or imatinib-intolerant GIST. A planned interim analysis revealed a significant difference in overall survival (OS) between patients randomized to receive sunitinib (n=207) or placebo (n=105) in favor of sunitinib (HR: 0.49; P=0.007) without the medians having been reached. Crossover from placebo to sunitinib was allowed for patients with progressive disease (PD) and at trial unblinding. The trial continues to assess the efficacy, safety and tolerability of sunitinib, but the crossover study design results in conventional statistical methods giving rise to biased estimates of treatment-effect for mature survival data.

Methods: In addition to the log-rank test, Cox model and Kaplan–Meier method, OS was analyzed using the rank-preserving structural failure time (RPSFT) method to account for crossover.

Results: Two-hundred forty-three patients were ultimately randomized to receive sunitinib and 118 patients were randomized to receive placebo. Of the latter group, 104 crossed over to sunitinib. As of November 2007, conventional analysis demonstrated that OS converged between the two treatment groups across the entire trial (double-blind and open-label phases): median OS for sunitinib was 74.7 weeks (95% CI: 61.4–85.7), while that for placebo was 64.9 weeks (95% CI: 45.7–98.4; HR: 0.82, P=0.128), a result that was expected because of the crossover design. In contrast, RPSFT analysis revealed an estimated median OS of 36.0 weeks (95% CI: 25.9–51.0) for placebo, thus making apparent a significant treatment effect of sunitinib (HR: 0.46, P<0.0001) comparable with that of the blinded phase. Throughout the entire study, as in the blinded phase, the most common treatment-related AEs in the sunitinib arm were fatigue (38%), diarrhea (34%), nausea (28%) and skin discoloration (28%), which were mostly grade 1/2. The most common treatment-related grade 3/4 AEs were fatigue (8%), hand–foot syndrome (4%) and hypertension (4%). Treatment-related hypothyroidism (all grades) occurred in 5% of patients. The incidence of treatment-related hypertension (all grades) was 14%. Overall, the incidence of cardiac AEs was low (4%). Incidences of non-hematologic AEs increased slightly with extended duration of sunitinib therapy. Hematologic laboratory abnormalities included reduced levels of hemoglobin (57%), neutrophils (55%) and platelets (41%), were mostly grade 1/2 and were similar in frequency for shorter-term and extended sunitinib therapy.

Conclusions: The long-term OS benefit of sunitinib relative to placebo in patients with imatinib-resistant or imatinib-intolerant GIST was confirmed in this phase III trial using RPSFT analysis. This finding has implications for other crossover trials. Long-term sunitinib therapy demonstrated an acceptable and predictable safety profile.

3. O-018. Role of KRAS mutations in predicting response and survival in irinotecan-refractory patients treated with cetuximab and irinotecan for metastatic colorectal cancer: Analysis of 281 patients with individual data

Di Fiore F¹, Van Cutsem E¹, Laurent-Puig P², Personeni N¹, Siena S³, Frattini M⁴, De Roock W¹, Lièvre A², Sartore-Bianchi A³, Bardelli A⁵, Tejpar S¹

¹Digestive Oncology Unit, University Hospital Gasthuisberg, Leuven-Belgium, ²Inserm U775, Université Paris-Descartes, Paris-France, ³Divisione Oncologia Medica Falck, Ospedale Niguarda Ca' Granda, Milan-Italy, ⁴Institute of Pathology, Locarno-Switzerland, ⁵Laboratory of Molecular Genetics Institute for Cancer Research and Treatment, University of Torino Medical School, Torino-Italy

Purpose of the study: Cetuximab, a monoclonal antibody (mAb) directed against the Epidermal Growth Factor Receptor (EGFR), is approved in combination with irinotecan (Iri) for the treatment of Iri-refractory metastatic colorectal cancer (mCRC) expressing EGFR. Recent studies have shown that *KRAS* mutation confers resistance to anti-EGFR mAbs. We extracted data of 281 patients (pts) from 7 series (Lievre 2006 and 2008, Moroni 2005, Di Fiore 2007, De Roock 2007, Benvenuti 2007, Frattini 2007) to determine the role of *KRAS* mutation in Iri-refractory mCRC patients treated with cetuximab plus Iri based-chemotherapy (CT).

Methods: The following data were collected: sex, age, previous CT lines, anti-EGFR regimen, response rate based on RECIST criteria, progression-free survival (PFS), overall survival (OS) and *KRAS* mutational status. Response rate was evaluated using the Fischer exact test. PFS and OS were calculated using the Kaplan-Meier method and compared with log-rank test. Predictive factors of response and survival were determined by logistic regression and Cox-regression model, respectively.

Results: A total of 281 pts were included, 174 men and 107 women with a mean age of 59.8 yrs. Pts received a mean of 2.4 prior CT lines. 77 pts (27.4%) responded (3 complete response (CR) and 74 partial response (PR)), 107 (38.1%) had stable disease (SD) and 97 (34.5%) had disease progression (PD). A *KRAS* mutation was detected in 98 pts including 40/107 (37.4%) pts with SD and 58/97 (59.8%) with PD. All responders were *KRAS* wild-type. *KRAS* mutation was significantly associated with PD ($p < 0.0001$). Median PFS and OS were significantly lower in mutated *KRAS* pts, 12 weeks (wks) vs 24 wks ($p < 0.0001$) and 36 wks vs 44 wks ($p < 0.0001$), respectively. The absence of *KRAS* mutation was identified as predictive factor of disease control (CR+PR+SD) ($p < 0.0001$, OR:0.17;95IC:0.10-0.30) and OS ($p < 0.0001$, OR:0.51;95IC:0.37-0.70), respectively.

Conclusions: *KRAS* mutational status plays a key role in response rate, PFS and OS in Iri-refractory mCRC pts treated with cetuximab and Iri. Our results demonstrate the potential major impact of *KRAS* mutation in pts selection.

4. O-021. Updated results of STEPP, a phase 2, open-label study of pre-emptive versus reactive skin toxicity treatment in metastatic colorectal cancer (mCRC) patients receiving panitumumab+FOLFIRI or irinotecan-only chemotherapy as second-line treatment

Mitchell, Edith P.¹ LaCouture, Mario,² Shearer, Heather,³ Iannotti, Nicholas,⁴ Piperdi, Bilal,⁵ Pillai, Madhavan V.,⁶ Xu, Feng,⁷ Yassine, Mohamed⁷

¹Kimmel Cancer Center, Thomas Jefferson University, Philadelphia, PA, USA; ²Northwestern University, Chicago, IL, USA; ³Piedmont Hematology Oncology Associates PLLC, Winston-Salem, NC, USA; ⁴Hematology Oncology Associates of Treasure Coast, Port Saint Lucie, FL, USA; ⁵University of Massachusetts Medical Center, Worcester, MA, USA; ⁶Virginia Oncology Care PC, Richlands, VA, USA; ⁷Amgen Inc., Thousand Oaks, CA, USA

Background: Panitumumab, a fully human monoclonal antibody targeting the epidermal growth factor receptor, is approved as single agent in the US for the treatment of refractory mCRC and in Europe for refractory mCRC in patients with wild-type *KRAS*. This randomized study examined differences between pre-emptive and reactive skin treatment for skin toxicities associated with panitumumab+chemotherapy.

Methods: Patients with unresectable mCRC that failed first-line administration of oxaliplatin-based chemotherapy +/- bevacizumab were enrolled. Patients received Q2W FOLFIRI+panitumumab (6.0 mg/kg) or Q3W irinotecan+panitumumab (9.0 mg/kg). Within cohorts, patients were randomized 1:1 to pre-emptive skin treatment (24 hrs prior to 1st panitumumab dose, daily through week 6) or reactive skin treatment (after skin toxicity developed). Skin toxicity treatment included use of skin moisturizers, sunscreen (PABA free, SPF ≥ 15 , UVA/UVB protection), topical steroid (1% hydrocortisone cream), and doxycycline (100 mg BID). The primary endpoint was incidence of specific \geq grade 2 skin toxicities during the 6-week skin treatment period. Secondary endpoints included safety and efficacy; responses were confirmed ≥ 4 weeks after the initial assessment. This planned interim analysis includes the results for the primary endpoint, and safety and efficacy for all enrolled patients (N=95) with the opportunity to complete the first tumor assessment.

Results: Of all 95 patients, 48 received pre-emptive and 47 received reactive skin treatment. Nearly all patients had ECOG status of 0 or 1. Median panitumumab doses were 5.0 for the pre-emptive and 6.0 for the reactive treatment group. 93% of all patients had a panitumumab treatment-related adverse event (AE); 71% of all patients had a grade 3/4 AE. Panitumumab dose reductions due to skin toxicities occurred in 8 (8%) patients. 36 (38%) patients had serious AEs. 13 (14%) patients ended treatment because of an AE. Pooled AEs of interest are shown (Table).

Conclusions: No safety issues were identified in this analysis of patients receiving panitumumab+chemotherapy. The final analysis of the primary endpoint will be presented. Interim safety and efficacy (RR, PFS, OS) by pre-emptive versus reactive skin treatment randomization, chemotherapy regimen, and *KRAS* status for all patients will be presented.

Table: Any AEs of Interest¹ (N=95)

AE	Any grade, n (%)	Grade 3, n (%)	Grade 4, n (%)
Skin Toxicity ²	85 (89)	19 (20)	1 (1)
Infections ^{2,3}	72 (76)	20 (21)	2 (2)
Diarrhea	67 (71)	22 (23)	0 (0)
Nausea	57 (60)	7 (7)	0 (0)
Neutropenia	29 (31)	11 (12)	5 (5)
Dehydration	26 (27)	16 (17)	0 (0)
Hypomagnesemia	20 (21)	3 (3)	2 (2)
Deep vein thrombosis	2 (2)	2 (2)	0 (0)
Pulmonary embolism	0 (0)	0 (0)	0 (0)

¹Per MedDRA; grading based on CTCAEv3, modified for skin toxicities.

²Represents system organ class

³Includes rash pustular: 32 (34%) pts with any grade and 10 (11%) pts with ≥grade 3, and paronychia: 24 (25%) pts with any grade and 5 (5%) pts with ≥grade 3

5. O-031. The CRYSTAL study: Assessment of the predictive value of *KRAS* status on clinical outcome in patients with mCRC receiving first-line treatment with cetuximab or cetuximab plus FOLFIRI

Van Cutsem E¹, Lang I², D'haens G³, Moiseyenko V⁴, Zaluski J⁵, Köhne C⁶, Folprecht G⁷, Tejpar S¹, Shparyk Y⁸, Schlichting M⁹, Kisker O⁹, Stroh C⁹, Rougier P¹⁰

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Background: The magnitude of the treatment effect of cetuximab alone or in combination with standard chemotherapy has recently been shown to be associated with *KRAS* mutation status in patients with metastatic colorectal cancer (mCRC). In the randomized controlled phase III CRYSTAL study, the addition of cetuximab to FOLFIRI significantly reduced the risk of progression (HR= 0.85; 95% CI [0.726, 0.998]) and significantly enhanced the chance of a tumor response (odds ratio of best overall response 1.40; 95% CI [1.115, 1.766]). Additional evaluation of this study has been conducted to assess the influence of *KRAS* mutation status on the outcome of treatment with FOLFIRI ± cetuximab.

Methods: Of the 1198 randomized patients, 578 provided archived tumor material for analysis. Genomic DNA was isolated and *KRAS* mutation status determined on codons 12/13 using a mutation-specific, quantitative PCR-based assay. The relationship between *KRAS* mutation status (wild-type [wt] or mutant [mt]) and outcome was assessed in the 540 *KRAS*-evaluable patients for PFS (primary endpoint; Independent Review Committee evaluation) by means of a stratified log-rank test and the stratified CMH test for best overall response.

Results: Generally, the *KRAS*-evaluable patient population was representative of the overall intent-to-treat (ITT) population. *KRAS* mutations were detected in 35.6% of patients (192/540) with evaluable samples. The treatment effect in terms of PFS and best overall response was substantially enhanced in patients with *KRAS* wt treated with cetuximab and FOLFIRI compared with the overall ITT population. Instead of a 15% risk reduction for progression, there was a 32% reduction in risk of progression (p=0.017) in patients with *KRAS* wt treated with cetuximab and FOLFIRI compared with FOLFIRI alone. Addition of cetuximab to FOLFIRI in patients with *KRAS* mt showed no significant differences between the groups for either PFS or ORR (Table).

	ITT		KRAS wt		KRAS mt	
	Cetuximab+FOLFIRI	FOLFIRI	Cetuximab+FOLFIRI	FOLFIRI	Cetuximab+FOLFIRI	FOLFIRI
N	599	599	172	176	105	87
ORR (%)	47	39	59	43	36	40
Odds ratio	1.40		1.91		0.80	

[95% CI]	[1.115, 1.766]	[1.245, 2.929]	[0.440, 1.443]			
p-value	0.0038	0.0025	0.46			
PFS (months)	8.9	8.0	9.9	8.7	7.6	8.1
HR [95% CI]	0.85 [0.726, 0.998]	0.68 [0.501, 0.934]	1.07 [0.710, 1.610]			
p-value	0.048	0.017	0.75			

Conclusions: KRAS mutation status can be considered as a predictive marker for clinical outcome in terms of PFS and ORR in patients with mCRC receiving first-line treatment with cetuximab plus FOLFIRI. There was a 32% reduction in risk of progression ($p=0.017$) in patients treated with cetuximab plus FOLFIRI in the KRAS wt population.

6. O-034. Multivariate analysis of prognosis prediction for stage II and III colorectal cancer patients using genomic profiling, clinical parameters and K-ras and PI3-Kinase mutation status

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Background: The TNM staging system is the current standard for determining the prognosis and treatment of colorectal cancer (CRC) patients. Chemotherapy is usually recommended for stage III patients but the treatment of stage II patients is still controversial. Various biomarkers and clinical parameters are therefore investigated to improve the diagnosis and predict response to therapy. In this study, we report the validation of a prognostic genomic profile and its interaction with other prognostic factors.

Patients and Methods: Using microarray technology and tumor classification methods, we recently identified a subset of genes, the ColoPrint prognostic signature, that are predictive for the prognosis of recurrence of stage II and III CRC patients. We now validated the signature on independent samples from 300 patients treated with radical surgery at two Spanish hospitals. The signature was converted into a robust diagnostic tool and control systems were established to monitor reproducibility and accuracy. Uni- and multi-variate analyses are used to evaluate the significance of risk stratification obtained using the prognostic profile in relation to existing clinic-pathological parameters. Additionally, the *K-Ras* and *PI3-Kinase* mutation status was determined in a subset of samples to correlate mutation with prognosis.

Results: Tumor samples for the Validation study were collected at different hospitals. Patients had a median follow-up time for 56 months (1-130 months). Most patients had colon cancer (79%) and more than half of the patients had stage II cancer (57%). Applying the prognostic profile on the stage II subset, 61% of patients were classified as low risk and 39% as high risk. Patients with a high risk score had a significant higher risk of developing distant metastasis within the next five years (HR 3.9, $p=0.01$). In the multivariate analysis, including profile, grade, number of assessed lymph nodes (12) and stage, the profile and T-stage were identified as the strongest independent prognostic factors. Patients classified as high risk by both gene expression and ASCO recommendation risk factors have the worst prognosis. Notably, those individuals discordantly classified as low risk by ASCO recommendation but high risk by gene expression analysis, also experienced poor survival. The correlation of prognostic profile, clinic-pathological parameters and *PI3-Kinase* and *K-Ras* mutation status is currently being analyzed. The results of this analysis will be available at the meeting.

Conclusion: Microarray gene expression profiling is able to identify subsets of stage II colon cancer patients most likely to benefit from adjuvant chemotherapy. A combination of various biomarkers and clinical parameters will allow the best characterization of tumors and the most tailored treatment of patients.

7. O-035. Clinical Benefit of Bevacizumab (BV) in Metastatic Colorectal Cancer (mCRC) is Independent of K-ras Mutation Status: Exploratory Analyses in a Large, Placebo-Controlled Phase III Study of Previously Untreated Patients

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¹Genentech, Inc., South San Francisco, California, United States, ²Duke University, Durham, North Carolina, United States

Background: Mutation of the K-ras gene has been identified as a prognostic marker in mCRC. In addition, there is emerging data suggesting that K-ras mutation is a negative predictor of clinical benefit from anti-EGFR treatment in mCRC. Previously reported data

also suggest that improvement in overall survival (OS) observed with BV treatment in mCRC is independent of alterations in the Ras/Raf/Mek/Erk pathway. We conducted additional analyses to better describe parameters of clinical benefit of BV treatment in mCRC relative to K-ras mutation status.

Methods: Microdissected tumor samples from 230 patients (pts), who were enrolled on a placebo-controlled randomized phase III trial of irinotecan/fluorouracil/leucovorin (IFL) ± BV, were subject to DNA sequence analysis for k-ras mutation. Progression-free survival (PFS), overall survival (OS), and objective response (ORR) were done retrospectively on an intent-to-treat basis. Safety analysis was based on reports of targeted adverse events (AE) in treated pts.

Results: K-ras status was assessed in 129 patients (56.1% of eligible population) treated in combination with BV and 101 patients (43.9%) in the placebo arm. In wild-type (wt) and mutant (m) K-ras tumors similar efficacy was observed (Table). The odds ratio for ORR in wt k-ras was 2.52 (60.0 vs 37.3%; P = .0055) and 1.09 (43.2 vs 41.2%; P = .86) in m K-ras. The incidence of BV associated AEs is comparable for the two k-ras subgroups. However, due to the small sample size and the rarity of the events, no conclusion could be made on the small difference observed.

Conclusion: BV provides similar clinical benefit in pts with mCRC expressing either wt or m K-ras.

	Wild Type K-ras	Mutant K-ras	Eligible population
	IFL ± BV (n=152)	IFL ± BV (n=78)	IFL ± BV (n=230)
HR PFS (95% CI)	0.44 (0.29, 0.67)	0.41 (0.24, 0.70)	0.44 (0.32, 0.61)
mPFS (mo)	7.4 vs 13.5 (p<.0001)	5.5 vs 9.3 (p=.0008)	6.3 vs 11.3 (p<.0001)
HR OS (95% CI)	0.58 (0.34, 0.99)	0.69 (0.37, 1.31)	0.60 (0.40, 0.91)
mOS (mo)	17.6 vs 27.7(p=.043)	13.6 vs 19.9 (p=.26)	17.5 vs 25.1 (p=.014)

8. O-037. KRAS Mutation status is a predictive biomarker for cetuximab benefit in the treatment of advanced colorectal cancer – Results from NCIC CTG CO.17: A phase III trial of cetuximab versus best supportive care.

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¹Australasian Gastrointestinal Trials Group, National Health and Medical Research Council Clinical Trials Centre, University of Sydney, Sydney, NSW, Australia; ²Bristol-Myers-Squibb Company, Princeton NJ & Wallingford CT, USA; ³National Cancer Institute of Canada Clinical Trials Group, Queen’s University, Kingston, ON, Canada.

ABSTRACT

Background: Cetuximab, a monoclonal antibody that targets the epidermal growth factor receptor (EGFR), improves overall survival (OS) and progression free survival (PFS) and preserves quality of life in patients with advanced colorectal cancer (CRC) that has progressed after chemotherapy. The presence of wild type K-Ras (WT) may predict which patients will optimally benefit from cetuximab in this setting. In addition, the prognostic significance of K-Ras mutation status is unclear.

Methods: CRC tumour samples were collected and analysed as part of a phase III clinical trial of cetuximab plus best supportive care (BSC) versus BSC alone (NEJM 2007; 357(20): 2040-8). Activating mutations in exon 2 of the K-Ras gene were detected in tumour-derived genomic DNA by direct gene sequencing without knowledge of clinical outcome. The predictive effect of K-Ras mutation status on OS and PFS was examined using a Cox model with tests for treatment-biomarker interaction.

Results: K-Ras mutation status was ascertained in 394 (69%) of the total study population (198 cetuximab, 196 BSC). Mutant K-Ras was detected in 164 (42%) patients. Within the mutant K-Ras group the median PFS was the same (1.8 months) for both groups (HR, 0.99; 95% CI, 0.73 to 1.35; p=0.96), while median OS was 4.6 months with cetuximab and 4.5 months with BSC (HR, 0.98; 95% CI, 0.70 to 1.37; p=0.89). In the 230 (58%) WT patients, median PFS was 3.8 months for the cetuximab treated group and 1.9 months with BSC (HR, 0.40; 95% CI, 0.30 to 0.54; p < 0.0001). The survival of patients with WT K-Ras was longer when they were treated with cetuximab, with a median OS of 9.5 months with cetuximab vs. 4.8 months with BSC (HR, 0.55; 95% CI, 0.41 to 0.74; p<0.0001). The test for interaction between K-Ras mutation status and cetuximab treatment demonstrates that the effect of cetuximab on OS (p=0.01) and PFS (p=0.0001) is significantly greater in the K-Ras WT group than mutant group. The difference in the OS of patients with either WT or mutant K-Ras in the BSC arm was not significant (HR, 1.01; 95% CI, 0.74 to 1.37; p=0.97).

Conclusions: In the setting of pre-treated advanced CRC, there is an almost doubling of median overall and progression free survival in patients with WT K-Ras tumours while no significant benefit is observed in patients with mutant K-Ras. K-Ras mutation status did not

demonstrate a prognostic effect within a 'no treatment' group. In this population, K-Ras mutation status is a strong predictive biomarker and K-Ras mutation analysis may now be considered a new standard of care in the selection of patients for EGFR targeted therapy.



Gastrointestinal Cancers Disease Information

Gastrointestinal Cancers include:

- Anal Cancer
- Colorectal Cancer
- Esophageal Cancer
- Gallbladder Cancer
- Gastric Cancer
- Liver Cancer (Hepatoma)
- Pancreatic Cancer
- Small Intestine Cancers

“Colorectal cancer is a major public health problem in western countries, with the highest incidence rates in North America, Western Europe, Australia and New Zealand. It is the third most common cancer in both men and women, and the third most common cause of cancer death in both sexes. Colon cancer is 2.5 times more common than rectal cancer, and both have different natural histories and thus separate treatment strategies. About 90-95% of all colorectal cancers are adenocarcinomas, with the remainder comprised of squamous cell, neuroendocrine or undifferentiated carcinomas.”
(www.oncolink.com)

“The American Cancer Society estimates that about 112,340 new cases of colon cancer (55,290 men and 57,050 women) and 41,420 new cases of rectal cancer (23,840 men and 17,580 women) will be diagnosed in 2008.

Colorectal cancer is the second leading cause of cancer-related deaths in the United States and is expected to cause about 52,180 deaths (26,000 men and 26,180 women) during 2008.

The number of deaths from colorectal cancer has been dropping for the past 15 years. There are a number of likely reasons for this. One probable reason is that polyps are being found by screening and removed before they can develop into cancers. Screening is also allowing more colorectal cancers to be found earlier when the disease is easier to cure. In addition, treatment for colorectal cancer has improved over the last 10 years, allowing for more effective options for people with this diagnosis. Because of this, there are around 1 million survivors of colorectal cancer in the United States.

The 5-year relative survival rate for people whose colorectal cancer is treated in an early stage, before it has spread, is greater than 90%. But only 39% of colorectal cancers are found at that early stage. Once the cancer has spread to nearby organs or lymph nodes, the 5-year relative survival rate goes down, and if cancer has spread to distant organs (i.e., the liver or lung) the 5-year survival is less than 10%.” (www.cancer.org)

"Patient Profile for Colorectal cancer: It is found most often in people aged 50 and older but any age possible.

Gender Profile for Colorectal cancer: It is a myth that this cancer affects mostly men. Women get colorectal cancer as often as men do (about a 1 in 20 chance).

Survival rate for Colorectal cancer: 47% survival rate for colon cancer in the UK 2001 (National Statistics – UK Government Census, 2001)

Average life years lost for Colorectal cancer: 13.4 years for colon/rectum cancer (SEER)¹

Deaths for Colorectal cancer: 57,200 deaths in USA 1999 (CDC)" (www.wrongdiagnosis.com)

[Types of Colon Cancer](#)

The type of colon cancer is usually defined by what kind of cell or tissue (collection of cells) it originates in. For example, the most common type of colon cancer is adenocarcinoma (literally, "cancer of a gland"). But colon cancer can begin in other types of cells, too. Colon lymphoma, which is rare but does occur, begins in a lymphocyte (lymph cell). Leiomyosarcoma of the colon, which is also rare, is a cancerous tumor that begins in one of the muscle layers of the colon.

[Stages of Colon Cancer](#)

There are five colon cancer stages (0-4). Colon cancer used to be rated using the Duke's system. For example, Duke's A cancer was the equivalent of stage 1 cancer. Sometimes you'll still hear people refer to their tumors that way, which can cause some confusion. This staging system reflects where the cancer went when no one was looking. In general, the earlier the stage, the easier the cancer is to treat.

[Colon Cancer Treatment](#)

There are three standard colon cancer treatments: surgery, chemotherapy, and radiation therapy. Another potential option for some patients is immunotherapy. But, surgery is the most common treatment and is often combined with chemo/radiation to treat later-stage cancers." (www.about.com)

(The information above is provided for reference only. The source of the information is indicated following each section. Imedex does not claim any responsibility for incorrect or misleading information.)



The World Congress on Gastrointestinal Cancer

The ESMO International Symposium: World Congress on Gastrointestinal Cancer continues to serve as a forum at which leading oncologists, practicing clinicians and specialists in cancer research review the state of the art and share the newest information on the management of gastrointestinal cancer. The Congress is built around a multi-disciplinary approach provided in plenary lectures by widely recognized experts, complemented with presentations of selected controversial topics, meet the expert sessions, case discussions, and satellite symposia on specific topics.

Congress chairs Mario Dicato and Eric Van Cutsem have been with the Congress since its inception in 1999 as *Perspectives in Colorectal Cancer*, along with Jacques Wils, of Laurentius Hospital, Roermond, The Netherlands, who retired as chair following the 2003 Congress. At the end of 2004, an agreement was reached between the organizers and the European Society for Medical Oncology (ESMO) to collaborate on the development of the Congress, further increasing the educational value of this event and increasing exposure to a wider audience. In 2007, the Congress was host to more than 3000 attendees from around the world with 89 countries represented.

Since its inception in 1999, the Congress has strived to increase its educational quality and scope for attendees. It has grown into an event that encompasses malignancies affecting every component of the gastrointestinal tract and aspects related to the care of all gastrointestinal cancer patients, including screening, diagnosis, and the latest management options (prevention, treatment, and supportive care) for common and uncommon tumors.

Congress Chairs

Mario Dicato, MD

Luxembourg Medical Center,
Luxembourg



Eric Van Cutsem, MD, PhD

University Hospital Gasthuisberg,
Leuven, Belgium



Mario Dicato, MD
Luxembourg Medical Center, Luxembourg

Mario Dicato is Professor and Head of Internal Medicine in the Department of Haematology-Oncology at Luxembourg Medical Centre, Luxembourg. He did his postgraduate training at Yale and Harvard University.

Professor Dicato is a member of several scientific societies, including the European Haematology Association, the American Society of Hematology, the American Society of Clinical Oncology, and the European Society for Medical Oncology. He is currently serving as Chair of the ESMO Symposia Working Group and as a member of the ESMO Educational Steering Committee. Professor Dicato has published more than 100 scientific papers and numerous book chapters. He is a member of the editorial board of several oncology journals.

Eric Van Cutsem, MD, PhD
University Hospital Gasthuisberg, Leuven, Belgium

Eric Van Cutsem is currently Professor of Internal Medicine at the University of Leuven, Belgium. He is responsible for the division of Digestive Oncology at the University Hospital Gasthuisberg in Leuven, where he sees many patients with gastrointestinal cancer. Professor Van Cutsem obtained the degree of medical doctor in 1983 at the University of Leuven, specialised in internal medicine and obtained his PhD degree in 1994. He is holder of the special chair Digestive Oncology at the University of Leuven and has a mandate as clinical researcher of the Fund for Scientific Research (FWO). During his training he spent several months in England, Switzerland, the USA and the Netherlands involved in clinical and research projects.

Professor Van Cutsem's main research interest is the treatment of gastrointestinal tumours and he has published more than 185 peer-reviewed articles and more than 300 other texts or chapters in books. His work has appeared in respected publications such as the *New England Journal of Medicine*, *The Lancet*, *JAMA*, *Journal of Clinical Oncology*, *Annals of Oncology*, *European Journal of Cancer*, *British Journal of Cancer* and *Gastroenterology*. He also co-ordinates several European and worldwide trials investigating new drugs for gastrointestinal cancer and serves on many steering committee and advisory boards.

Professor Van Cutsem is a member of several scientific organisations, including the American Society of Clinical Oncology (ASCO), the European Society of Medical Oncology (ESMO), the American Association of Cancer Research (AACR), the European NeuroEndocrine Tumor Society (ENETS) and many national organisations. He was a member of the programme committee of ASCO 2003-2004, ASCO GI programme committee 2004, 2006-2007, and is a member of the ASCO Cancer Education Committee Committee 2005-2008. Professor Van Cutsem is a member of the ESMO faculty and of the strategic ESMO multidisciplinary oncology committee and was chairman of the ECCO 2005 colon cancer committee. He is president of the Belgian FAPA (Familial Adenomatous Polyposis Association), chairs the ministerial commission on colon cancer prevention in Flanders, is vice-president of the Belgian Group of Digestive Oncology and is vice-president of IDCA (International Digestive Cancer Alliance). He served as secretary of the European Organization for Research and Treatment of Cancer – Gastrointestinal Cancer Group (EORTC-GI group) from January 2000 - February 2003. He became chairman of the EORTC GI group in March 2003. He is a member of the general assembly of PETACC (PanEuropean Trials on Adjuvant Colon Cancer).

Professor Van Cutsem is/was a member of the editorial board of *Journal of Clinical Oncology*, *Annals of Oncology*, *European Journal Cancer*, *Japanese Journal Clinical Oncology*, *Critical Reviews in Oncology/Hematology*, *Clinical Colorectal Cancer*, *European Journal of Cancer Prevention*, *Journal of Chemotherapy*, *Expert Opinion of Pharmacotherapy* as well as several others, and is associate editor of *Targeted Oncology*.

2008 Scientific Committee

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About ESMO

The European Society for Medical Oncology (ESMO) is the leading European non-profit, professional organization for medical oncology with a focus on promoting multidisciplinary cancer treatment around the world.

ESMO unites medical oncologists, oncology specialists, healthcare professionals, caregivers, patients and policy makers in a global alliance committed to eradicating cancer and ensuring equal access to high quality treatment for all patients. Thanks to its state-of-the-art education and training programs, ESMO plays an instrumental role in providing the oncology community with the most up-to-date scientific research and information available. Through its flagship journal, *Annals of Oncology*, ESMO publishes articles on all aspects of clinical oncology. ESMO is dedicated to educating and supporting oncologists, optimizing patient care, disseminating cancer-specific information to the public, and advocating patient rights. As an authoritative voice in the fight against cancer, ESMO provides both the platform and the consultative expertise to influence national and international organizations as well as European authorities, in order to establish common standards for a multidisciplinary approach to cancer treatment.

For more information about ESMO, please visit www.esmo.org or the ESMO booth.

José Baselga, MD, Spain

ESMO President



Education

- **Degrees:** MD Universidad Autonoma of Barcelona (1982)
- **Residency:** Internal Medicine at Vall d'Hebron University Hospital, Barcelona, Spain and State University of New York
Fellowship in Medical Oncology at Memorial Sloan-Kettering Cancer Center, New York, USA
- **Specialization:** Internal Medicine Medical Oncology, Hematology

Professional Experience

- Chairman, Medical Oncology Service and Director of the Division of Medical Oncology, Hematology, and Radiation Oncology, Vall d'Hebron University Hospital, Barcelona, Spain
- Professor of Medicine, Universidad Autonoma de Barcelona, Spain
- Staff, Breast Medicine service, Memorial Sloan-Kettering Cancer Center (1992-1996)

Special Recognition

- **Main areas of research interest:** Clinical Breast Cancer and in Translational and Early Clinical Research in the area of Growth Factor Receptors and Downstream Molecules as Targets for Breast Cancer Therapy
- **Awards:** Young Investigator Award from ASCO (1992); Career Development Award from ASCO (1994); Bristol-Myers Squibb Unrestricted Cancer Grant Award (2002-2006); Honorary Membership Award from The European Society for Therapeutic Radiology and Oncology (ESTRO) (2004); Waun Ki Hong Visiting Professorship at U.T.M.D. Anderson Cancer Center in Houston, TX (2002); named Distinguished Alumnus from Memorial Sloan Kettering Cancer Center in New York, U.S.A. (2004); Elected member of the American Society of Clinical Investigation (2004); Annual Award from ESMO (2005); San Salvatore Prize (2006); American-Italian Cancer Foundation Prize for Scientific Excellence in Medicine (2007) and AACR-Rosenthal Family Foundation Award (2008)
- **Editorial Board:** Annals of Oncology, Cancer Cell, Clinical Cancer Research and Journal of Clinical Oncology
- **Faculty member:** ESMO Basic cancer research
- **Committee member:** ESMO President (2008-2009), ESMO Board of Directors and Executive Committee (since 2006), ESMO Scientific Committee Chair 29th ESMO Congress, Scientific Chair ECCO 13, Vice-Chair ESMO Young Oncologist Working Group, ESMO Symposium Working Group
- **Society member:** ESMO, ASCO, AACR, EORTC
- **Languages:** Spanish, English, Catalan
- **Other:** Involved in the clinical development of several new agents including: gefitinib, erlotinib, lapatinib, pertuzumab, m-TOR inhibitors, PI3K inhibitors, TGF β inhibitors, SRC inhibitors, Insulin-like Growth Factor Receptor inhibitors and a variety of Anti-Angiogenic agents. Conducted the initial clinical trials with the monoclonal antibodies cetuximab and trastuzumab.

David J Kerr, CBE, MA (OXON), MD, DSc,
FRCP, FRCGP, FMedSci., United Kingdom

Editor in Chief of *Annals of Oncology*,
ESMO President-Elect



Education

- **Degrees:**
 - MBChB (1980)
 - MRCP (1983)
 - MD (Aspects of Cytotoxic Drug Penetration 1987)
 - FRCP (Fellow of Royal College of Physicians and Surgeons, Glasgow, 1995; Royal College of Physicians London, 1996)
 - BSc (First class honors in Biochemistry 1977)
 - MSc (Clinical Pharmacology 1990)
 - PhD (Regulation of proliferation of breast cancer cells by growth factors 1990)
 - DSc (The molecular and clinical pharmacology of anticancer therapy 1996)
 - MA (OXON) (2002)
 - FRCGP (2007)
- **Residency:** Department of Clinical Pharmacology, University of Oxford, Old Road Research Campus Building, Old Road Campus, off Roosevelt Drive, Headington, Oxford, OX3 7DQ
- **Specialization:** Medical Oncology
- **Board Certified:** General Medical Council (GMC)

Professional Experience

- Rhodes Professor of Clinical Pharmacology and Cancer Therapeutics at the University of Oxford

Special Recognition

- **Main areas of research interest:** Treatment and research in colorectal cancer and gene therapy

- **Principal investigators:** QUASAR 2 Trial, VICTOR Trial
- **Awards:** European School of Oncology International Award for outstanding contribution to chemotherapy research (1987), 2nd International Prize for Excellence in the field of Colorectal Cancer Research and Treatment (1999), 1st Nye-Bevan Award for Innovation (2000), Fellow of the UK Academy of Medical Oncology (2000), Commander of British Empire (CBE 2002), Distinguished Medeval Lecture (2006), ESMO Award for Medical Oncology (2006)
- **Editorial Board:** Editor-in-Chief of *Annals of Oncology* (since 2000)
- **Faculty member:** University of Oxford, ESMO Faculty Basic Cancer Research
- **Committee member:** ESMO President-Elect (2008-2009), ESMO Board of Directors and Executive Committee, Chair *Annals of Oncology* Editorial Board, International Atomic Energy Agency, International Agency for Research on Cancer, Translational Research Working Group (NCI), 1998 ESMO Congress Scientific Committee Chair (Athens)
- **Society member:** ESMO, United Kingdom National Cancer Task Force
- **Languages:** English
- **Other:** Established INDOX (www.indox.org.uk) trials network with India's leading Oncologists; is building AfrOx (www.afrox.org) to improve cancer prevention and control in Africa. Published the "Kerr Report", 2006, a 20 year plan for Scotland's NHS.

Roberto Labianca, MD, Italy

**ESMO Representative in World Congress on
Gastrointestinal Cancer Scientific Committee**



Education

- **Degrees:** Degree in Medicine and Surgery on 1975
- **Specialization:** Specialization in Medical Oncology on June 1978; specialisation in Allergology and Clinical Immunology on July 1981

Professional Experience

- Head of Unit of Medical Oncology – Ospedali Riuniti di Bergamo
- Responsible of the Group on Gastrointestinal Cancer of Medical Oncology Department S.Carlo Borromeo Hospital (Milan) (1995-1997)
- Deputy Director of Medical Oncology Department S.Carlo Borromeo Hospital (1988-1997)
- Assistant of Medical Oncology Department S. Carlo Borromeo Hospital (1980-1988)
- Research fellow of Medical Oncology Department S. Carlo Borromeo Hospital (1975-1980)

Special Recognition

- **Committee member:** GISCAD (Gruppo Italiano per lo studio dei Carcinomi dell'Apparato Digerente), Scientific Secretary from 1990; ITMO (Italian Trials in Medical Oncology), Member of National Executive Council from 1991; GIVIO (Gruppo Italiano per la valutazione degli interventi in Oncologia) from 1989; EORTC (European Organization for the Research and Treatment of Cancer) as Active Member of Gastrointestinal Cooperative Group from 1987; GITCLO (Gastrointestinal Tumour Cooperative Groups Liaison Office) as national representative for Italy from 1995; IWGCRC (International Working Group for Colorectal Cancer) from 1995
- **Membership in Scientific Societies:** AIOM (Associazione Italiana di Oncologia Medica)
 - Foundation President (2005-2007)
 - President (2003-2005)
 - National Treasurer (1999-2001)
 - National Secretary (1995-1999)
 - National Councillor (1993-1995)

- Co-ordinator in Lombardia (1989/1993)
- Regional Councillor (1986-1988)
- CIOM (Collegio Italiano degli Oncologi Medici) from 1985
- SIC (Società Italiana di Cancerologia) from 1984
- ESMO (European Society for Medical Oncology):
 - National Representative for Italy (1994-1997)
 - Medical Oncology Status in Europe Survey (MOSES) Task Force Chair (2003-today)
- ASCO (American Society of Clinical Oncology) from 1994
- MASCC (Multinational Association for Supportive Care of Cancer) from 1994
- SICP (Società Italiana di Cure Palliative) from 1989
- EAPC (European Association of Palliative Care) from 1992
- SITILLO (Società Italiana per le Terapie Integrate Locoregionali in Oncologia) from 1995
- **Editorial Board:** Tumori, Quaderni Cure Palliative, Progress in Colorectal Cancer, Tekve (Update in Oncology and Hematology), Annals of Oncology, Journal of Chemotherapy