Welcome to

Advances in the Management of Colorectal Cancer:
A Latin-American Perspective

Dear Delegate,
Welcome to São Paulo!

The “Advances in the Management of Colorectal Cancer: A Latin-American Perspective” symposium, organised by Springer Healthcare, is an important opportunity for Latin-American oncologists, gastroenterologists and gastric surgeons to meet and discuss the latest developments in the field of colorectal cancer, focusing in particular on the most up-to-date research presented recently at ESMO 2014 in Madrid, Spain, in September this year.

We have selected the key presentations from this year’s ESMO programme and, with the help of Professor Alberto Sobrero (Genova, Italy), have summarised these to inform and educate the audience on topics such as (i) the newest developments in classifying the subclasses of colorectal cancer; (ii) state-of-the-art tools available for directing adjuvant chemotherapy for colorectal cancer; (iii) recent guidelines for identifying the correct chemotherapy agent when treating metastatic colorectal cancer; and (iv) the latest findings from key clinical trials into new treatment options for colorectal cancer.

Each session will feature a Panel Discussion focusing on the implications of the findings for the Latin-American region and placing the results into context with respect to current local colorectal cancer management protocols and guidelines. As there will be an emphasis on informality, discussion and interaction between delegates and the Panel, I encourage you to get involved by asking questions or commenting on what you have heard, or writing your questions or comments on the Question Cards, which you will find on your table during the meeting. This is an important opportunity for all of us to discuss and highlight the impact of global advances on our own regions and practices.

I hope that you find the symposium both enjoyable and highly informative.

Paulo Hoff

Paulo Hoff, MD, PhD, FACP
Symposium Chair
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**29 November**

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<tr>
<td>09:30–09:40</td>
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| 09:40–09:50 | A new molecular taxonomy for colorectal cancer – What are the therapeutic implications?  
             | Presented at ESMO 2014 by Ultan McDermott; summarised by Alberto Sobrero |
| 09:50–10:00 | Liquid biopsies and “on treatment” markers                          |
|           | Presented at ESMO 2014 by Gerald Prager; summarised by Alberto Sobrero |
| 10:00–10:40 | Panel Discussion: Paulo Hoff, Anelisa Coutinho, Paulo Herman, Enrique Roca |
| 10:40–11:10 | Break                                                              |
| 11:10–11:20 | Are there any useful tools when deciding about adjuvant chemotherapy? |
|           | Presented at ESMO 2014 by Richard Adams; summarised by Alberto Sobrero |
| 11:20–11:30 | Final results from QUASAR2, a multicentre, international randomised phase III trial of capcitabine (CAP) +/- bevacizumab (BEV) in the adjuvant setting of stage II/III colorectal cancer (CRC) |
|           | Presented at ESMO 2014 by Rachel Midgely; summarised by Alberto Sobrero |
| 11:30–12:10 | Panel Discussion: Paulo Hoff, Anelisa Coutinho, Paulo Herman, Enrique Roca |
| 12:10–13:00 | CRC Case Study session: Anelisa Coutinho                            |
| 13:00–14:00 | Lunch                                                              |
| 14:00–14:10 | How to select first line chemotherapy in metastatic colorectal cancer |
|           | Presented at ESMO 2014 by Claus-Henning Koehne; summarised by Alberto Sobrero |
14:10–14:20  CALGB/SWOG 80405: PHASE III trial of irinotecan/5-FU/leucovorin (FOLFIRI) or oxaliplatin/5-FU/leucovorin (mFOLFOX6) with bevacizumab (BV) or cetuximab (CET) for patients (pts) with expanded ras analyses untreated metastatic adenocarcinoma of the colon or rectum  
Presented at ESMO 2014 by Heinz-Joseph Lenz; summarised by Alberto Sobrero

14:20–14:30  CALGB/SWOG 80405: Analysis of patients undergoing surgery as part of treatment strategy  
Presented at ESMO 2014 by Alan Venook; summarised by Alberto Sobrero

14:30–15:30  Panel Discussion: Paulo Hoff, Anelisa Coutinho, Paulo Herman, Enrique Roca

15:30–16:00  Break

16:00–16:10  Independent radiological evaluation of objective response, early tumor shrinkage, and depth of response in FIRE-3 (AIO KRK-0306) in the final RAS evaluable population  
Presented at ESMO 2014 by Sebastian Stintzing; summarised by Alberto Sobrero

16:10–16:30  Panel Discussion: Paulo Hoff, Anelisa Coutinho, Paulo Herman, Enrique Roca

16:30–16:40  Phase III RECOURSE trial of TAS-102 vs. placebo, with best supportive care (BSC), in patients (pts) with metastatic colorectal cancer (mCRC) refractory to standard therapies  
Presented at ESMO 2014 by Eric van Cutsem; summarised by Alberto Sobrero

16:40–17:00  Panel Discussion: Paulo Hoff, Anelisa Coutinho, Paulo Herman, Enrique Roca

17:00–17:15  Closing remarks: Paulo Hoff (Symposium Chair)
Faculty Biographies

**Paulo Hoff – Symposium Chair**

Paulo Hoff is a Professor of Oncology and General Director of the Instituto do Câncer do Estado de São Paulo (ICESP) at the University of São Paulo, and General Director of the Oncology Center at the Hospital Sírio Libanês, Brazil. An ASCO member since 1996, Dr Hoff has served on the Gastrointestinal Cancers Symposium Planning Committee, Cancer Education Committee and Scientific Program Committee, and the Journal of Clinical Oncology Editorial Board. In 1998, he received a Merit Award from ASCO for research presented at the ASCO Annual Meeting.

**Anelisa Coutinho**

Anelisa Coutinho is a Professor and Head of Department of the Division of Gastrointestinal Oncology, Assistência Multidisciplinar em Oncologia (AMO), Salvador, Bahia, Brazil. Dr Coutinho is President of the Brazilian Gastric Tumor Group (GTG) and a Medical Oncologist at the Assistência Multidisciplinar em Oncologia (AMO) where she is in charge of the Gastrointestinal Tumour Department.

**Paulo Herman**

Paulo Herman is Professor and Head of the Liver Surgery Unit at the University of São Paulo Medical School, São Paulo, Brazil. Dr Hermann obtained his Ph.D. in Medicine (Digestive Surgery) from the University of São Paulo. He is currently Director of the Department of Liver Surgery, Hospital das Clínicas, Faculty of Medicine, University of São Paulo. Dr Herman has experience in the area of Medicine, with a particular emphasis in Gastroenterology Surgery.

**Enrique Roca**

Enrique Roca is Professor and Head of the Oncology Service at Hospital Dr Carlos Bonorino Udaondo of the Autonomous City of Buenos Aires, Argentina. Dr Roca is a Principal Investigator at the Argentine Intergroup for the Treatment of Gastrointestinal Tumours (IATTGI).
Presenter Biography

Alberto Sobrero

Alberto Sobrero has been Head of the Medical Oncology Unit at Ospedale San Martino in Genova, Italy, since 2001. Before this, he was Associate Professor in Medical Oncology at the University of Florence and Udine, Italy. His main research interests include chemotherapy, targeted agents in gastrointestinal cancer, and the design of clinical trials.

Professor Sobrero is a member of the American Society of Clinical Oncology (ASCO), the American Association for Cancer Research (AACR), the European Society for Medical Oncology (ESMO), and several Italian scientific societies. Between 2002 and 2006, Professor Sobrero was Chairman of the Protocol Review Committee of the European Organisation for Research and Treatment of Cancer (EORTC). He has also served on the Editorial Board of the Journal of Clinical Oncology (2003–2006) and is presently a member of the Scientific Committee of ASCO as well as the Educational Committee of ESMO.
Abstracts

The following talks were presented at ESMO 2014 in Madrid, Spain, and will be summarised during this event in video interviews presented by Professor Alberto Sobrero (Genova, Italy).

A new molecular taxonomy for colorectal cancer – What are the therapeutic implications?

Ultan McDermott (Cambridge, United Kingdom)

In the past decade, advances in next-generation sequencing technologies have enabled us to define a new molecular taxonomy of human cancer based on the genetic and epigenetic basis of cancer. These efforts are based on the fundamental belief that a comprehensive knowledge of the genes that cause human cancer is a critical foundation for cancer diagnostics, therapeutics and clinical trial design. In particular, the International Cancer Genome Consortium and The Cancer Genome Atlas have collectively sequenced tens of thousands of cancers across a number of tumour types.

Colorectal cancer is a major cause of cancer mortality. The heterogeneity of response to therapy in this disease has led to speculation that as yet undiscovered molecular subtypes (each with differing clinical behaviour and outcomes) may be responsible. Thus, the detection of such subtypes could fundamentally alter the treatment paradigm for colorectal cancer. That such subtypes may regulate treatment response and outcome has been suggested by the discovery that approximately 15% of colorectal cancers are characterized by deficient DNA mismatch repair, leading to microsatellite instability (MSI) and mutations in critical genes involved in carcinogenesis, such as transforming growth factor-β type II receptor and BAX. Such tumours tend to be right-sided, BRAF mutant, have a better prognosis yet derive less benefit from standard 5FU chemotherapy. We now have a complete repertoire of the mutational, copy number, methylation and gene expression features that are dysregulated in colorectal cancer, and molecular subtypes that predict for clinical outcome are presenting themselves. Most recently, a combined analysis of gene expression data from 4,000 colorectal tumour samples has defined 4 consensus clusters, each with different patterns of mutations, dysregulated pathways and MSI status.

The challenge for the clinical research community will be to determine whether a new molecular taxonomy for colorectal cancer based on such subtypes has therapeutic implications and will play a role in our management of patients.

Liquid biopsies and “on treatment” markers

Gerald Prager (Vienna, Austria)

The discovery of molecular mechanisms for malignant transformation, angiogenesis and metastasis formation have opened an abundance of biologic insights and subsequent targeted therapeutic options, which have led to improved prognosis in colorectal cancers. Although this has been found beneficial for many patients with metastasized colorectal cancer, a substantial fraction of patients show secondary resistance after initial response, thus, limiting the clinical benefit of targeted treatment.

While assessment of somatic gene alterations from tumour-tissue biopsies is in routine clinical use for targeted treatment in colorectal cancer, it bears inherent limitations. Treatment of colorectal cancer leads to a dynamic molecular alterations due to selective pressure. Thus, a real-time characterization rather than a baseline snap-shot analysis is required. Recent evidence suggest that analysis of cell-free circulating tumour DNA (ctDNA) reflects an accurate, sensitive and specific way to track
The molecular tumour dynamic and the individual tumour burden. The advantage of early detection of acquired molecular alterations leading to treatment resistance in colorectal cancer might thereby soon be implemented into clinical routine as determination of ctDNA is cost-effective, less invasive and precise. The advantages as well as the challenges to implement liquid biopsies in to the routine clinical use of colorectal cancer treatment will be discussed.

CALGB/SWOG 80405: PHASE III trial of irinotecan/5-FU/leucovorin (FOLFIRI) or oxaliplatin/5-FU/leucovorin (mFOLFOX6) with bevacizumab (BV) or cetuximab (CET) for patients (pts) with expanded ras analyses untreated metastatic adenocarcinoma of the colon or rectum

Heinz-Joseph Lenz (Los Angeles, USA)

Aim: FOLFIRI or mFOLFOX6, combined with BV or CET, are 1st-line treatments for MCRC. The optimal antibody combination is unknown.

Methods: Pts with RAS wt (codons 12 and 13) MCRC and performance status 0-1 received FOLFIRI or mFOLFOX6 (MD/pt choice at enrollment) and randomized to either CET 400 mg/m2 X 1, then 250 mg/m2 qw or BV 5 mg/kg qw 2w. The original study included unselected MCRC pts receiving FOLFIRI or mFOLFOX6 and randomized to CET, BV or both. After 1420 pts accrued the study amended as follows: only pts w/ KRAS wt tumours (codon 12 and 13) were included. Accrual goal was 1142 pts Expanded RAS was tested in all wt ras exon 2 using beaming technology including KRAS exon 3, 4 and NRAS exon 2 and 3 with a detection sensitivity of 0.01%. Subsequent Rx not mandated. 1° endpoint was overall survival (OS).

Results: Between 11/2005 and 3/2012, 3058 unselected pts enrolled, 2334 KRAS wt pts randomized; final N =1137 (333 pre-amend eligible retrospective KRAS test, 804 post-amend), median f/u = xx mos; Median age–59 y; 61% male. Chemo/BV–559; chemo/CET–578. FOLFIRI = 26.6%, mFOLFOX6 = 73.4%. From xx patients tumour samples were available for expanded ras analyses.

Conclusions: Updated analysis will be shown at meeting.

Independent radiological evaluation of objective response, early tumour shrinkage, and depth of response in FIRE-3 (AIO KRK-0306) in the final R

Sebastian Stintzing (Munich, Germany)

FIRE-3 compared 1st-line therapy with FOLFIRI plus either cetuximab or bevacizumab in 592 KRAS exon 2 wild-type mCRC patients. An independent radiological review was carried out to evaluate tumour response according to RECIST 1.1 and to define early tumor shrinkage (ETS) and depth of response (DpR).

Methods: Extended RAS analysis was carried out in KRAS and NRAS exon 2, 3 and 4 (codons 12, 13, 59, 61, 117 and 146) using pyro-sequencing technique. Radiological data were evaluated independently. ETS was defined as a reduction in tumour diameter of more than 20% at first-tumour assessment after baseline (week 6). DpR was defined as the maximal tumour shrinkage observed in a patient. Reviewers were blinded to patient data. Calculations were done for both, the ITT- and the extended RAS wild-type population.

Results: Within the ITT population, 475 patients (80.2%) were successfully tested for all RAS locations. Primary outcome data for the RAS wt population (n = 400). ORR significantly favoured the FOLFIRI plus cetuximab arm (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015). The proportion of patients reaching ETS was significantly greater in the FOLFIRI plus cetuximab treated population (71.4% vs. 56.4%; two sided Fisher’s exact test p = 0.015).
was significantly greater in FOLFIRI plus cetuximab treated patients (48.2% vs. 33.0%, Wilcoxon test \( p = 0.0005 \)) and associated with post progression survival (Braavis Pearson test \( p < 0.0001 \)). Updated results will be presented.

Conclusions: Based on an independent radiological review, FOLFIRI plus cetuximab induced a significantly higher ORR, a greater rate of ETS, and an increased DpR compared to FOLFIRI plus bevacizumab. These response-related outcomes may in part explain the significant OS advantage of FOLFIRI plus cetuximab observed in FIRE-3.

**CALGB/SWOG 80405: Analysis of patients undergoing surgery as part of treatment strategy**

Alan Venook (San Francisco, USA)

Aim: Patients with metastatic colorectal cancer may be cured with multimodality therapy. The goal of this subset analysis is to determine the characteristics and the long-term outcome of patients enrolled on this first-line treatment study for metastatic disease but who underwent surgery following chemotherapy.

Methods: Pts with KRAS wt (codons 12 and 13) MCRC and performance status 0-1 received FOLFIRI or mFOLFOX6 (MD/patient choice at enrolment) and randomized to either CET 400 mg/m² X 1, then 250 mg/m² qw or BV 5 mg/kg q2w. At enrolment, the goal of therapy was chosen by the MD: palliative or as neoadjuvant therapy as part of a curative treatment plan. Rx continued until progression, death, unacceptable toxicity, surgery with curative intent; treatment holidays of 4 weeks permitted but patients in general came off study if they underwent surgery. Subsequent Rx not mandated. Accrual goal was 1142 pts. 1° endpoint was overall survival (OS).

Results: Between 11/2005 and 3/2012, 3058 unselected pts enrolled, 2334 KRAS wt pts randomized; final N =1137 (333 pre-amend eligible retrospective KRAS test, 804 post-amend), median follow-up = 32 mos; Median age–59 y; 61% male. At some point after chemotherapy, 179 patients (15.7%) went to surgery, median age 54, 62% male. 24.6% with intact primary, 36.9% with curative intent, 80% FOLFOX, and 58% CET. 130 of the 179 pts were NED immediately post-surgery (104 (58%) of these pts stopped protocol treatment to pursue surgery; 20 (11%) pts due to adverse events; 18 (10%) had surgery after progressing on study treatment.) Median time between study entry and surgery was 6.8 mos and median OS from randomization was 60 mos; 95% CI (49, 69). DFS from surgical resection among NED pts was 6.8 mos and median OS from randomization was 60 mos; 95% CI (49, 69). DFS from surgical resection among NED pts was 16.1 mos; 95%CI (10.5, 22.2) and from randomization was 26 mos; 95% CI (21, 34). 96 pts are alive post-surgery (50 remain NED) after a median of 37 mos post-surgical f/u.

Conclusions: 130 of 1137 pts enrolled on study reached NED after chemotherapy and surgery. The median OS in these patients was 60 months although many have recurred. We anticipate all-RAS status and plan on analysing the subset of patients who underwent surgery to identify possible predictive characteristics and also to determine if there is an explanation for the fact that more patients on CET went to surgery than did pts on BV.

**Final results from QUASAR2, a multicentre, international randomised phase III trial of capecitabine (CAP) +/- bevacizumab (BEV) in the adjuvant setting of stage II/III colorectal cancer (CRC)**

Rachel Midgely (Oxford UK)

Aim: The aims of Q2 were to assess whether the addition of BEV 7.5mg/kg q3/52 (12/12) to single agent CAP 1250mg/m², 14 of every 21/7 (6/12), increases disease-free (DFS) and overall survival (OS) in CRC patients after resection of the primary; and to validate suggested, or discover new, biomarkers of BEV efficacy and toxicity.

Methods: A phase III international randomised controlled trial, coordinated by the UK and recruiting in 6 countries. In addition to the collection of data
on toxicity, DFS and OS, a biobank comprising 1350 FFPE blocks and 1000 germline DNA samples was established. Hypothesis-driven biomarkers (MSI status and epithelial/stromal ratio) and hypothesis-driven biomarkers (chromosomal instability, ras, raf, POLE and an 80-gene ion torrent panel) were analysed to assess their prognostic and predictive (BEV) utility.

Results: 1941 patients were randomised in a 1:1 ratio and demographics and disease characteristics were well balanced between the two arms. DFS in the whole trial population demonstrates that BEV does not improve outcome in this setting (3 year DFS 75.2% for CAP/BEV vs 78.2% for CAP: HR = 1.06; p = 0.54). Similarly OS was not improved (3 year OS 85.5% for CAP/BEV vs 87.2% for CAP; p = 0.38; HR = 1.12). There may be a temporal trend in HRs (HRs: 1 year 0.83 [0.61-1.13], 2 year 0.87 [0.65-1.17], 3 year 1.32 [0.9-1.98]).

Biomarker analyses confirm that high tumour stromal content confers a worse prognosis (3 year DFS HR 1.58 [1.22-2.05]; p = 0.001). MSS positivity was associated with a worse DFS in patients treated with CAP/BEV compared to those treated with CAP alone (n = 840; HR 1.43; p = 0.005) suggesting a negative predictive effect for BEV. For MSI positive patients, there was no significant difference in DFS between the two arms (n = 135; HR 0.74; p = 0.42).

Conclusions: Q2 supports data from two other trials suggesting no role for BEV in the adjuvant setting of CRC. The Q2 biobank and linked database allows further collaborative biomarker hypotheses to be tested. There is a rationale for meta-analysis of all BEV adjuvant CRC studies to more fully explore the putative temporal effect of BEV administration on DFS.

Phase III RECOURSE trial of TAS-102 vs. placebo, with best supportive care (BSC), in patients (pts) with metastatic colorectal cancer (mCRC) refractory to standard therapies

Eric Van Cutsem (Leuven, Belgium)

Aim: TAS-102 is a combination of a novel oral nucleoside, trifluridine (FTD) with the thymidine phosphorylase inhibitor, tipiracil hydrochloride (TPI), which prevents the degradation of FTD, enabling sustained and effective FTD levels. RECOURSE (Sponsor: Taiho Oncology Inc. / Taiho Pharmaceutical Co. Ltd.) was conducted to evaluate the efficacy and safety of TAS-102 in pts with mCRC refractory to standard therapies.

Methods: mCRC-patients (ECOG PS 0-1) who failed fluoropyrimidines, irinotecan, oxaliplatin, bevacizumab and cetuximab/panitumumab (if KRAS wild-type) were eligible. The primary endpoint (overall survival, OS) and the key secondary efficacy endpoint (progression-free survival, PFS) were evaluated using univariate and multivariate analyses for prospectively defined subgroups and a retrospectively defined subgroup with prior regorafenib.

Results: 800 pts were randomized to TAS-102 (534 pts) or placebo (266 pts). The hazard ratios for OS and PFS were 0.68 (95% CI: 0.58 - 0.81; p < 0.0001) and 0.48 (95% CI: 0.41 - 0.57; p < 0.0001), respectively, both favouring TAS-102. OS and PFS benefit for TAS-102 was consistent across all subgroups. OS treatment effect in the multivariate model remained the same.

Safety results were previously presented [Yoshino T. et al., Ann Oncol 2014; 25 (Suppl 2; abstr O-0022)]. Time to worsening of ECOG PS status to 2 or more was significantly delayed with TAS-102 vs. placebo [medians of 5.7 vs. 4.0 months, HR = 0.66 (95% CI: 0.56–0.78)]. Post-study treatment was similar between arms (41.2% in TAS-102, 42.5% in placebo).

Are there any useful tools when deciding about adjuvant chemotherapy?

Richard Adams (Cardiff, UK)
Part of the Optimising the treatment of colorectal cancer ESMO 2014 Education session. Abstract not available.

How to select first line chemotherapy in metastatic colorectal cancer

Claus-Henning Koehne (Oldenburg, Germany)
Part of the Optimising the treatment of colorectal cancer ESMO 2014 Education session. Abstract not available.
Information for Delegates

Registration
Delegates should register at the registration desk in the lobby of the Hilton São Paulo Morumbi hotel. Registration is open from 16:00–22:00 on 28 November and from 08:00–09:30 on 29 November.

Welcome Reception
A welcome reception for all delegates will be held at the Hilton São Paulo Morumbi from 18:00–20:00 on 28 November.

Language and Translations
All Video Summaries and Panel Discussions will take place in English language. Simultaneous translation of the Video Summaries and Panel Discussions into Brazilian Portuguese is available for all delegates. Please pick up a headset at the entrance to the meeting room on the morning of 29 November.

Enduring Materials
Please note that all videos and slides from the programme will be made available to delegates on USB sticks at the end of the event and also online at http://education.springerhealthcare.com/colorectal-cancer-symposium-latin-america-2014/.

Point of Contact
If you have any questions during the meeting, please contact the registration desk or a Springer Healthcare representative, or alternatively email CRC_LATAM2014secretariat@springer.com.

Delegate List
A full list of delegates attending the event will be available upon registration.

Filming and Photography
Springer Healthcare may film or take photographs at the symposium that may be used in publicity and marketing materials. Your attendance at the meeting may mean you are featured in such photographs and films and, unless you notify the registration desk of your objection to this, your consent will be implied.
Conference Venue

The meeting and delegate accommodation are in the Hilton São Paulo Morumbi, Av. das Nações Unidas, 12.901 - São Paulo – SP 04578-000, Brazil.

Welcome Reception (28 November): Rooms 6/7 on the 3rd Floor of the hotel
Main Meeting (29 November): Morumbi Ballroom on the Ground Floor near the Lobby
Lunch (29 November): Rooms 6/7 on the 3rd Floor of the hotel
Airport Transfers

This hotel is 8 km from Congonhas National Airport (CGH) and 42 km from Guarulhos International Airport (GRU).

Transfers to and from the airport will be provided for all delegates arriving by plane. Upon arrival, please look for a driver displaying the symposium name in the airport’s Arrival Hall.

Transfers to the airport after the symposium will depart from the lobby of the Hilton São Paulo Morumbi – please refer to your pre-event email containing your personal travel arrangements to find the time of your own transfer. If you are unsure of your transfer time, please contact a member of the Springer Healthcare team, who can advise you on this.
Organisers, CME and Educational Grant

Organisers

This educational activity has been planned and independently implemented by Springer Healthcare, with collaboration on educational design and accreditation from Siyemi Learning.

Continuing Medical Education

The ‘Advances in the Management of Colorectal Cancer: A Latin-American Perspective’ is accredited by the European Accreditation Council for Continuing Medical Education (EACCME) to provide the following CME activity for medical specialists. The EACCME is an institution of the European Union of Medical Specialists (UEMS), www.uems.net.

The ‘Advances in the Management of Colorectal Cancer: A Latin-American Perspective’ is designated for a maximum of (or ‘for up to’) 6 hours of European external CME credits. Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity.

Through an agreement between the European Union of Medical Specialists and the American Medical Association, physicians may convert EACCME credits to an equivalent number of AMA PRA Category 1 Credits™. Information on the process to convert EACCME credit to AMA credit can be found at www.ama-assn.org/go/internationalcme.

Live educational activities, occurring outside of Canada, recognized by the UEMS-EACCME for ECMEC credits are deemed to be Accredited Group Learning Activities (Section 1) as defined by the Maintenance of Certification Program of The Royal College of Physicians and Surgeons of Canada.

Educational Grant

This programme is made possible thanks to an educational grant received from Merck Serono.