

JOURNAL OF CLINICAL ONCOLOGY

Official Journal of the
American Society of Clinical Oncology

2011 ASCO Annual Meeting Proceedings

47th Annual Meeting
June 3-7, 2011
McCormick Place
Chicago, IL

47th
Annual Meeting of the
American Society of Clinical Oncology
June 3–7, 2011
Chicago, Illinois

2011 Annual Meeting Proceedings Part I
(a supplement to the *Journal of Clinical Oncology*)



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2011 ASCO Annual Meeting Proceedings

Vol 29, No 15S

May 20, 2011

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| | |
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American Society of Clinical Oncology 47th Annual Meeting

2011 Abstracts

Abstract Session Descriptions for Scheduled Presentations

Oral Abstract Sessions

Oral Abstract Sessions include didactic presentations of the abstracts determined by the Scientific Program Committee to be of the highest scientific merit. Experts in the field serve as Discussants to place the findings into perspective. The Plenary Session includes the abstracts selected by the Scientific Program Committee as having practice-changing findings.

Clinical Science Symposia

Clinical Science Symposia provide a forum for science in oncology, combining didactic lectures on a specific topic with the presentation of abstracts. Experts in the field classify studies on the basis of the strength of the evidence and critically discuss the conclusions in terms of their applicability to clinical practice.

Poster Discussion Sessions

Poster Discussion Sessions highlight selected abstracts of clinical research in poster format. The posters are grouped by topic and are on display for a specified time, followed by a discussion session in which experts provide commentary on the research findings.

General Poster Sessions

General Poster Sessions include selected abstracts of clinical research in poster format. The posters are grouped by topic and are on display for a specified time.

Trials in Progress Poster Session

The Trials in Progress Poster Session is designed to facilitate awareness of open, ongoing clinical trials. This session encourages discussions of new clinical research and the exchange of ideas on clinical trial design. There will be no presentation of trial results.

Publication-only Abstracts

Publication-only abstracts were selected to be published online in conjunction with the Annual Meeting, but not to be presented at the Meeting.

All presented and publication-only abstracts are citable to this Journal of Clinical Oncology supplement. For citation examples, please see the Letter from the Editor.

This publication contains abstracts selected by the ASCO Scientific Program Committee for presentation at the 2011 Annual Meeting. Abstracts selected for electronic publication only are available in full-text versions online at abstract.asco.org and JCO.org. The type of session, the day, and the session start/end times are located to the right of the abstract number for scheduled presentations. To determine the location of the abstract session, refer to the Annual Meeting Program or the ePlanner, the online version of the Annual Meeting Program, available at chicago2011.asco.org.

Dates and times are subject to change.

All modifications will be posted on ASCO.org (www.asco.org).

Letter from the Editor

The *2011 ASCO Annual Meeting Proceedings Part I* (a supplement to the *Journal of Clinical Oncology*) is an enduring record of the more than 2,500 abstracts selected by the ASCO Scientific Program Committee for presentation at the 47th Annual Meeting of the American Society of Clinical Oncology, held June 3–7, in Chicago, Illinois. Accepted abstracts not presented at the meeting are available in full-text versions on ASCO.org and are included in the May 20 *Journal of Clinical Oncology* supplement online at JCO.org. Both the presented abstracts and electronic-only abstracts were made publicly available on abstract.asco.org on May 18, 2011, at 6:00 PM (EDT).

The majority of abstracts selected for presentation are presented here in full and are categorized by scientific track. Abstracts are ordered numerically according to presentation type within a track. Presentation types include Clinical Science Symposia, Oral Abstract, Poster Discussion, General Poster, and Trials in Progress Poster Sessions. Abstracts include the presenting author only. The full list of abstract authors and their disclosure information can be found online at abstract.asco.org.

Certain abstracts are represented here by abstract title and presenting author. These include Clinical Review Abstracts (CRA), many of which will have important, and in some cases, immediate implications for patient

care; Late-breaking Abstracts (LBA), which feature results of phase III trials for which data were not available at the time of ASCO's regular submission deadline; and Plenary Abstracts, which were selected by the Scientific Program Committee for their potential to have practice-changing results. The full-text versions of abstracts within these three designations will be included in the *2011 ASCO Annual Meeting Proceedings Part II* (a supplement to the June 20 issue of *Journal of Clinical Oncology*). These abstracts will be available onsite at the Annual Meeting in print and CD-ROM formats, as well as on ASCO.org, on Saturday, June 4. They will be available on JCO.org starting in early June.

All of the abstracts carry *Journal of Clinical Oncology* citations. The following are citation examples for print and electronic abstracts:

J Clin Oncol 29:45s, 2011 (suppl; abstr 500)

J Clin Oncol 29, 2011 (suppl; abstr e12000)

Should you have any questions or comments about this publication we encourage you to provide feedback by contacting us at abstracts@asco.org.

Michael A. Carducci, MD
Editor, *2011 ASCO Annual Meeting Proceedings*
(Parts I and II)

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The abstracts published in the *2011 ASCO Annual Meeting Proceedings Part I*, as well as those published online only, were publicly released by ASCO at 6:00 PM (EDT) on Wednesday, May 18, 2011. These abstracts are publicly available online at www.asco.org, the official website of the Society. Plenary, Late-breaking, and Clinical Review Abstracts will be publicly released according to the following schedule:

- Plenary, Late-breaking, and Clinical Review Abstracts that are not part of ASCO's official Press Program will be publicly released at www.asco.org at 8:00 AM (EDT) on Saturday, June 4.
- Plenary, Late-breaking, and Clinical Review Abstracts selected for inclusion in the Press Program will be publicly released at the beginning of the News Briefing or at the beginning of the Scientific Session containing the research, whichever comes first. Any embargoes that have not lifted by 12:00 noon (EDT) on Sunday, June 5, will automatically lift at that time when the remaining abstracts are publicly posted on www.asco.org.

In the unlikely event that ASCO publicly releases an abstract in advance of the scheduled time, the release will be publicly announced on www.asco.org.

Abstract Notice

The *2011 ASCO Annual Meeting Proceedings Part I* (a supplement to *Journal of Clinical Oncology*) contains all of the abstracts to be presented at the 47th Annual Meeting of the American Society of Clinical Oncology with the exception of the Plenary, Late-breaking, and Clinical Review Abstracts. The full-text versions of these abstracts will be available to all Annual Meeting attendees in the *2011 Annual Meeting Proceedings Part II*, distributed onsite on Saturday, June 4.

ASCO Conflict of Interest Policy and Exceptions

In compliance with the guidelines established by the ASCO Conflict of Interest Policy (*J Clin Oncol.* 2006 Jan 20;24[3]:519–521) and the Accreditation Council for Continuing Medical Education (ACCME), ASCO strives to promote balance, independence, objectivity, and scientific rigor through disclosure of financial and other interests, and identification and management of potential conflicts. According to the ASCO Conflict of Interest Policy, the following financial and other relationships must be disclosed: employment or leadership position, consultant or advisory role, stock ownership, honoraria, research funding, expert testimony, and other remuneration (*J Clin Oncol.* 2006 Jan 20;24[3]:520). The ASCO Conflict of Interest Policy disclosure requirements apply to all authors who submit abstracts to the Annual Meeting.

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For more information about the ASCO Conflict of Interest Policy and the exceptions process, please visit www.asco.org/rwi.

ABSTRACTS
The American Society of Clinical Oncology
47th Annual Meeting
June 3–7, 2011
McCormick Place
Chicago, Illinois

SPECIAL AWARD LECTURE ABSTRACTS

David A. Karnofsky Memorial Award and Lecture
Saturday, June 4, 9:30 AM

Bench-to-bedside translation of targeted therapies in multiple myeloma.

Kenneth C. Anderson, MD; Dana-Farber Cancer Institute/Harvard Medical School, Boston, MA

Multiple myeloma (MM) is a remarkable example of rapid bench-to-bedside translation in new drug development. Over the past three decades, my studies have evolved from generation of monoclonal antibodies for identification of B and plasma cell surface diagnostic and therapeutic targets to development of in vitro and in vivo models of MM in the bone marrow (BM) microenvironment for characterization of molecular mechanisms whereby MM cells grow, survive, resist drugs, and migrate in the BM milieu. This progress has allowed for identification of potential therapeutic targets on the MM cell surface, within the tumor cell, and in the microenvironment, as well as validation of novel small molecules and monoclonal antibodies directed at these targets. To facilitate rapid bench-to-bedside translation of promising leads, we led collaborative efforts between academia, pharmaceuticals, regulatory agencies, and patient advocacy leading to new drug approvals. Our studies showed that bortezomib and lenalidomide target the MM cell in the BM microenvironment to overcome conventional drug resistance in laboratory and animal models, and rapid translation to clinical trials showed their efficacy in relapsed/refractory, relapsed, and newly diagnosed MM, as well as to consolidate and maintain response. Importantly, median survival of MM patients has extended from 3 to 7 years as a direct result. We are now applying oncogenomics to improve patient classification and develop personalized therapy; identify and validate next-generation novel agents targeting the tumor in its microenvironment; develop immune therapies; and inform design of rationally based combination targeted therapies. Moreover, synthetic chemistry capability within academia now allows for production of proof-of-principle targeted inhibitors and even more rapid translation of scientific advances to clinical application. Myeloma therefore represents a novel treatment paradigm targeting the tumor in its microenvironment to improve patient outcome, which has great potential in other hematologic malignancies and solid tumors as well.

Science of Oncology Award and Lecture
Sunday, June 5, 1:00 PM

The EMT and the pathogenesis of high-grade carcinomas.

Robert A. Weinberg, PhD; Whitehead Institute for Biomedical Research and Ludwig Center for Molecular Oncology at Massachusetts Institute of Technology, Cambridge, MA

The molecular and cellular mechanisms that allow primary carcinoma cells to metastasize have long been elusive. However, over the past 5 years, a cell biological program termed the epithelial–mesenchymal transition (EMT) has been found to play a key role in imparting to epithelial cancer cells many of the traits that are associated with mesenchymal cells, notably motility, invasiveness, and heightened resistance to apoptosis. These traits are precisely those of highly malignant carcinoma cells, and help to explain how primary carcinoma cells force epithelial cells—both normal and neoplastic—to acquire many of the traits of epithelial stem cells. The resulting self-renewing traits of such cells appear to play a key role in enabling these cells to spawn large cohorts of descendants that together form macroscopic metastases. These observations raise the question of how carcinoma cells in primary tumors are able to activate their EMT programs, which are active during embryogenesis and wound healing but otherwise silent. Certain EMT-inducing transcription factors (TFs) act as master regulators that choreograph EMT programs. Expression of these TFs is often induced by signals that individual cancer cells receive from the surrounding microenvironment and involve signaling by several well-studied paracrine signaling proteins, such as TGF- β , canonical Wnts, and noncanonical Wnts. When converging on carcinoma cells, these signals, working in concert, are able to induce expression of the

1548 General Poster Session (Board #3E), Sat, 2:00 PM-6:00 PM

Trends in survival by race for stomach cancer patients from the U.S. SEER cancer registry. Presenting Author: L. Chu, Genentech Inc., South San Francisco, CA

Background: Incidence and mortality rates for stomach cancer (SC) in the US have decreased steadily for years, but trends in survival are limited. The objective of this study was to describe median overall survival (OS) trends in US SC patients (pts) by race/ethnicity and tumor characteristics. **Methods:** SC pts diagnosed between 1978 and 2007 were identified in the Surveillance, Epidemiology, and End Results (SEER). SC was classified by tumor characteristics: tumor stage, anatomic site and sex. Frequency distribution and median OS (crude, unadjusted) were examined by time period (1978-1987, 1988-1997, 1998-2007) and race/ethnicity. **Results:** The median OS (months) by time period and race/ethnicity are presented in the table. Survival has increased modestly in the past 30 years, with Asians having the largest improvement. Asians have the longest median OS, followed by whites and blacks. Across all race/ethnicities, median OS in advanced stage pts has remained poor. **Conclusions:** Although survival has increased slightly over the last 30 years, prognosis for advanced stage SC remains particularly poor. In the U.S. SEER registry, Asian pts had longer median OS and greater improvement in survival over time compared to other ethnicities.

| | Stomach cancer median OS* (months) (95% CI) | | |
|------------------|---|-----------------------|-----------------------|
| | 1978-1987 N=19,641 | 1988-1997 N=25,905 | 1998-2007 N=49,723 |
| White | 8 (7, 8) | 8 (8, 9) | 9 (NE, NE) |
| Median age (yrs) | 71 | 72 | 71 |
| Anatomic site | | | |
| Cardia | 8 (8, 9) | 9 (9, 10) | 10 (10, 11) |
| Noncardia | 7 (7, 8) | 8 (NE, NE) | 8 (8, 9) |
| Tumor stage | | | |
| Localized | 38 (34, 43) | 39 (36, 42) | 36 (33, 38) |
| Regional | 12 (12, 13) | 14 (13, 14) | 16 (15, 16) |
| Distant | 4 (NE, NE) | 4 (NE, NE) | 4 (NE, NE) |
| Black | 8 (7, 9) | 8 (8, 9) | 8 (8, 9) |
| Median age (yrs) | 67 | 69 | 69 |
| Anatomic site | | | |
| Cardia | 6 (5, 7) | 6 (5, 8) | 8 (7, 9) |
| Noncardia | 8 (7, 9) | 9 (8, 9) | 9 (8, 9) |
| Tumor stage | | | |
| Localized | 32 (24, 44) | 39 (32, 49) | 33 (28, 41) |
| Regional | 13 (11, 14) | 14 (13, 17) | 14 (13, 15) |
| Distant | 4 (3, 4) | 3 (3, 4) | 4 (3, 4) |
| Asian | 11 (10, 12) | 13 (12, 14) | 15 (14, 16) |
| Median age (yrs) | 69 | 70 | 71 |
| Anatomic site | | | |
| Cardia | 9 (5, 12) | 11 (9, 12) | 13 (11, 15) |
| Noncardia | 12 (10, 13) | 13 (12, 14) | 15 (14, 16) |
| Tumor stage | | | |
| Localized | 103 (78, 124) | 115 (105, 132) | 89 (78, 101) |
| Regional | 17 (15, 19) | 18 (17, 19) | 23 (21, 24) |
| Distant | 5 (4, 5) | 4 (3, 4) | 5 (NE, NE) |

* Crude, unadjusted. Abbreviation: NE, not estimable.

1550 General Poster Session (Board #3G), Sat, 2:00 PM-6:00 PM

Does breast cancer in Asian geriatric patients have the same biological characteristics as in their Western counterparts? A comparison between Shanghai and Vienna. Presenting Author: M. M. Tea, Department of OB/GYN, Division of Senology, Medical University of Vienna, Vienna, Austria

Background: By 2030 it is estimated that 20% of the population will be 75 years of age or older. The incidence of breast cancer in Asia is especially rising, while the proportion of the population that is 60 and older is also growing. Today, approximately one-third of breast cancer occurs in women over the age of 65. The aim of this study was to investigate the biological differences of breast cancer older than 69 years in Asia and Europe. The results, including the stage at diagnosis and surgical therapy of Chinese breast cancer, were compared with their western counterparts. **Methods:** A total of 630 women, 70 years of age and older, with operable breast cancer were investigated in Shanghai and Vienna. Histopathological findings of 198 Austrian women seen during 2005 and 2010 were reviewed. The results were compared with 432 Chinese (time matched) obtained from the Breast Cancer Database in Shanghai. **Results:** The mean patients' age in the Viennese and Shanghai study groups were 75.3 and 77.9 years, respectively. In Austria, the breast-conserving surgery rate was 73.7% (n=146) compared to 10.4% (n=45) in China. Sentinel lymph node biopsy was performed in 40.4% (n=80) in Vienna vs. 8.3% (n=36) in Shanghai. More large tumors (tumor size > 2 cm) were detected in China (41.3%, n=169) than in Austria (34.7%, n=66) (p=0.033). No differences were found in axillary lymph node involvements (p=0.052). However, more grade-3 breast cancers were detected (p<0.001) in Austrians. Furthermore, there was a greater incidence of estrogen receptor-negative breast cancer in Chinese women (27%, n=113) compared to Austrian women (16.2%, n=32) (p<0.001). **Conclusions:** Chinese women present with a lower rate of grade-3 breast malignancies. As expected, a higher breast conserving surgery rate was noted in Vienna. Interestingly less Chinese women, although older than 69 years, were presented with estrogen receptor-positive breast cancer. We conclude that breast cancer characteristics are different in diverse ethnic groups. Ethnic-specific screening/therapy protocols may benefit this special Asian population, especially Asian women in western countries.

1549 General Poster Session (Board #3F), Sat, 2:00 PM-6:00 PM

Long-term history of fasting blood glucose level and the risk of pancreatic cancer: A nested case-control study. Presenting Author: K. Stefani, Department of Preventive Medicine, Yonsei University College of Medicine, Seoul, South Korea

Background: Many epidemiological studies have investigated the association between diabetes and pancreatic cancer (PC) as either a risk factor or manifestation, yet there have not been many studies which looked at long-term fasting blood glucose change before the diagnosis of PC. The aim of this study was to investigate the association between long-term changes in fasting blood glucose and PC risk in Korean adults. **Methods:** We performed a nested case-control study in the Korea Medical Insurance Corporation Study cohort which consists of 108,461 men and 64,119 women aged 35 to 59 years old. Fasting blood glucose levels were repeatedly measured every 2 years from 1990 to 2000. Hospital admissions or deaths due to PC were ascertained using the Death Statistics Database from the National Statistical Office and the National Health Insurance Claim Database. The cases consisted of 216 people with PC between 1993 and 2003, and the controls were 1080 people without PC at the time of sampling. We sampled five controls per each case using incidence-density sampling and matching for sex and age. Difference in the fasting glucose level between cases and controls were assessed using conditional logistic regression analyses. **Results:** Patients with PC had higher fasting glucose levels, compared to controls, from at least 10 years prior to the diagnosis. Moreover, patient's fasting glucose level started to increase 2-3 years prior to the diagnosis of PC. Mean difference (case's value minus control's value) was 6.2 mg/dl at 10-11 years prior to diagnosis (p=.007), 4.3 mg/dl at 8-9 years prior to diagnosis (p=.002), 6.8 mg/dl at 6-7 years prior to diagnosis (p=.077), 4.5 mg/dl at 4-5 years prior to diagnosis (p=.025), 10.2 mg/dl (p=.155) at 2-3 years prior to diagnosis, and 12.2 mg/dl (p=.026) at 0-1 year prior to diagnosis. **Conclusions:** These results support that long-term history of high fasting glucose level is associated with the risk of PC and an abrupt increase of fasting glucose level may be an early manifestation of PC. This study was supported by a grant of the Korean Health 21 R&D Project, Ministry of Health and Welfare, Republic of Korea (Grant No. A040152).

1551 General Poster Session (Board #3H), Sat, 2:00 PM-6:00 PM

Stroger Hospital of Cook County (CCH) AIDS malignancy project: The CHAMP study—A 13-year, single institution retrospective study of cancers in patients infected with HIV. Presenting Author: S. Gupta, John H. Stroger Hospital of Cook County, Chicago, IL

Background: CCH is the largest health provider for HIV patients in Chicago and has one of the largest HIV outpatient clinics in the United States, the Ruth M. Rothstein CORE Center (CC). Per year, over 5000 HIV + individuals are treated at the CC with over 500 HIV-infected individuals treated at CCH. Most studies of non-AIDS defining (NADC) and AIDS defining cancers (ADC) cover wide areas. Insights into the prevalence, type of cancer, and characteristics of HIV + cancer patients found in the inner city and underserved populations have been under represented in the literature. To this end, we examined all of the malignancies presenting to either the CC or CCH for the past 13 years. **Methods:** We identified via CCH, CC, and pharmacy databases all HIV infected patients with a cancer ICD9 codes from 1998-2010. HIV characteristics, cancer type, and patient demographics were confirmed by chart review. One-way Anova was used to show statistical significant between the prevalence among the different sub populations. **Results:** After initial screening, 404 cancers were confirmed, representing 21 malignancies. 56% were ADC, with 104 (25%) non-hodgkins lymphoma (NHL), 111(28%) Kaposi sarcoma, and 11(2.7%) cases of cervical cancer. Of the 104 NHL, the most common were DLBCL 61%, Burkitts (BL) 21%, and primary CNS lymphoma 7.6%. The most frequent NADC was anal cancer (n=30) and Hodgkin lymphoma (n=29). 70% of all cancers were stage III or above. HIV + patients at the CC were 71% African American (AA) and 19% Hispanic (Hsp) (compared to 26% and 23% in the Chicago population, respectively). In our study, AA represents 64% of all HIV cancers with Hsp and Caucasians 17% each. Though Caucasians with HIV and cancer in our cohort have the highest prevalence of cancer, 1.06%, vs. 0.69% AA vs. 0.60 Hsp (P< 0.001). The Male:Female ratio of all HIV malignancies in our cohort are 5.1:1. **Conclusions:** Our data show a race disparity isolated to AA compared to the general population, and a male predominance. Although the Caucasian-HIV population is the smallest, they have the highest cancer prevalence.

1553 General Poster Session (Board #4B), Sat, 2:00 PM-6:00 PM

The influence of sociodemographic factors on breast cancer tumor biology. Presenting Author: K. A. Dookeran, The Cancer Foundation for Minority and Underserved Populations, Chicago, IL

Background: African American (AA) race is often associated with lower socioeconomic status (SES) and features of aggressive tumor biology in breast cancer, including: ER/PR-negative (-) subtypes, p53-positive (+) disease, and high grade. We attempt to disaggregate the influence of race and SES on tumor biology. **Methods:** Logistic regression models [odds ratios (ORs), 95% confidence intervals (CIs)] were used to select and evaluate sociodemographic factors at diagnosis related to tumor biology in a cohort of 534 women (331 AA, 203 non-AA) with breast cancer. **Results:** Table shows baseline covariates. After adjustment for age and race, multivariate independent predictors were: (a) all ER/PR- subtypes: higher parity, with a borderline relationship with worsening poverty/education status; (b) p53+ disease: only younger age at menarche; and (c) grade 3 disease, not having a FH, with a borderline relationship with higher parity. **Conclusions:** Results suggest that early menarche may predispose diagnosis with p53+ disease [OR, 1.2; 95% CI, 1.06-1.37; p=0.005], while higher parity and worse poverty/education status may predispose diagnosis with high-grade, ER/PR- subtype disease, and race may exert its effects through such factors.

Tumor biology covariates.

| Sociodemographic factors | ER/PR- | | Triple negative | | Basal type | | p53+ | | Grade 3 | |
|--|--------|-----------|-----------------|-----------|------------|-----------|------|-----------|---------|-----------|
| | OR | 95% CI | OR | 95% CI | OR | 95% CI | OR | 95% CI | OR | 95% CI |
| Race (AA/non-AA) | 1.11 | 0.77-1.59 | 1.29 | 0.87-1.90 | 1.65 | 1.01-2.69 | 0.92 | 0.60-1.42 | 1.04 | 0.73-1.48 |
| Age (per year) | 0.98 | 0.96-0.99 | 0.98 | 0.96-0.99 | 0.97 | 0.95-0.99 | 0.99 | 0.97-1.01 | 0.99 | 0.98-1.01 |
| Comorbidity (+/-) | 0.79 | 0.54-1.15 | 0.71 | 0.48-1.07 | 0.68 | 0.42-1.11 | 0.97 | 0.62-1.52 | 0.96 | 0.65-1.40 |
| Family history (+/-) | 0.82 | 0.55-1.24 | 0.79 | 0.51-1.23 | 0.67 | 0.38-1.16 | 0.87 | 0.53-1.42 | 0.49 | 0.33-0.74 |
| Age at menarche (per year) | 0.97 | 0.88-1.08 | 0.98 | 0.88-1.10 | 0.97 | 0.85-1.10 | 0.83 | 0.73-0.94 | 0.97 | 0.88-1.08 |
| Parity (per birth) | 1.08 | 0.99-1.17 | 1.13 | 1.04-1.23 | 1.11 | 1.00-1.23 | 1.00 | 0.90-1.10 | 1.07 | 0.99-1.16 |
| BMI (≥ 30 / < 30) | 0.86 | 0.59-1.28 | 0.94 | 0.62-1.42 | 0.94 | 0.58-1.53 | 1.01 | 0.64-1.60 | 0.81 | 0.55-1.19 |
| Poverty status (> 1999 U.S. mean/ \leq) | 1.65 | 1.06-2.57 | 2.01 | 1.21-3.34 | 1.83 | 1.00-3.36 | 1.02 | 0.61-1.71 | 1.16 | 0.76-1.77 |
| Education status (< 1999 U.S. mean/ \geq) | 1.59 | 0.99-2.53 | 1.86 | 1.10-3.17 | 1.98 | 1.02-3.84 | 1.79 | 0.97-3.29 | 0.91 | 0.58-1.42 |

1555 General Poster Session (Board #4D), Sat, 2:00 PM-6:00 PM

Compliance to adjuvant hormone therapy for black and white women with breast cancer. Presenting Author: S. Bhatta, University of Chicago, Chicago, IL

Background: The use of adjuvant hormone therapy for estrogen receptor positive (ER+) breast cancer significantly decreases breast cancer recurrence and mortality. For reasons that are poorly understood, black women have lower risk but higher mortality from breast cancer. To better understand factors contributing to the disparate outcomes, we examined differences in compliance to adjuvant hormone therapy by race/ethnicity in women treated for ER+ breast cancer at a private University medical center. **Methods:** 381 women with ER+ breast cancer 2 to 10 years from their initial diagnosis identified from a University cancer center registry were asked to complete a mailed survey. Compliance to therapy was self-reported (see table). A proportional odds model was used to evaluate possible predictors of compliance including ethnicity, perceived risk for breast cancer recurrence, and perception of adjuvant hormone therapy importance on risk reduction. Multivariate models were used to adjust for age of diagnosis, education completed, income, and out of pocket expenses for prescription medications. **Results:** Among the 208 (55%) responses, black women had lower levels of therapy compliance compared to white women (adjusted odds ratio (OR) = 0.40, 95% CI: 0.19-0.83). Regardless of race, the most significant determinant of compliance was perception of hormone therapy importance in risk reduction (adjusted OR=1.58, 95% CI: 1.06-2.36). Other variables including age or fear of recurrence did not affect compliance. **Conclusions:** For both black and white women compliance to therapy is affected by perceived importance of treatment. This study underscores the need for more effective doctor-patient communication to improve adherence to adjuvant hormone therapy, especially among ethnic minorities.

Study sample.

| n (%) | White n=141 | Black n=67 | P value |
|------------------------------------|----------------|---------------|---------|
| Age (mean; range) | 57.9; 39-72 | 58.6; 42-73 | 0.4 |
| > some college education | 113 (80.4) | 45 (67.7) | 0.04 |
| Income $\geq 60K$ | 95 (67.3) | 17 (25.3) | <0.001 |
| Reported therapy compliance | | | |
| Never took | 4 (2.8) | 5 (7.8) | 0.005 |
| Missed > 1/week | 0 (0) | 3 (4.7) | |
| Missed < 2x/month | 11 (7.8) | 4 (6.3) | |
| Missed ≤ 1 month | 29 (20.6) | 22 (32.8) | |
| Never missed dose | 97 (68.8) | 32 (48.4) | |

1554 General Poster Session (Board #4C), Sat, 2:00 PM-6:00 PM

Multicenter, prospective, case-control, observational study on the influence of venous thromboembolism on patient outcome. Presenting Author: M. Mandala, Hospital of Bergamo, Bergamo, Italy

Background: The association between venous thromboembolism (VTE) and cancer is well known. VTE represents the second cause of death in cancer patients. In some retrospective analysis, carried out using population and hospital-based registries, it has been reported that patients with cancer and VTE have a worse survival than those with cancer without VTE. **Methods:** MASTER ONCOLOGY is a prospective, multicenter, national, observational, case-control study specifically designed to identify VTE risk factors and to evaluate the influence of VTE on outcome of cancer patients with locally advanced or metastatic solid tumours. Patients in case (cancer patients with VTE) and control (cancer patients without VTE) groups have been matched for gender, age, cancer site, cancer histology and extension of disease. Information on patients, tumour characteristics, concomitant diseases, treatments, risk factors were registered. Survival data have been prospectively collected at 10 months and assessed through both Kaplan Meier method and Cox proportional hazards regression. **Results:** 611 patients were enrolled in the study by 51 Italian Centers. Of these, 237 cases and 339 controls were evaluable. The mean age was 64.7 (range 23-84) and 64.5 (range 41-87) years in cases and controls groups, respectively; the percentage of males was 43.0% and 41.9%. The most frequent tumour sites were colon, breast, lung, pancreas and stomach. Overall survival was statistically better for control patients as compared to case patients (p=0.0021). The raw HR was statistically significant: case patients had a risk of death 1.56 larger than controls (95% CI 1.23-1.98). Considering the complete model (using backward selection), the HR, due to patients status after adjustment for covariates, was 1.29 (95% CI 1.01-1.66). **Conclusions:** The study results demonstrate, for the first time, in a prospective and specifically designed study, that patients with cancer and VTE have a reduced survival compared to cancer patients without VTE.

1556 General Poster Session (Board #4E), Sat, 2:00 PM-6:00 PM

The accuracy of tobacco assessment during definitive radiotherapy or chemoradiotherapy in patients with head and neck cancer. Presenting Author: M. R. Kudrimoti, University of Kentucky, Lexington, KY

Background: The accuracy of self-reported (SR) tobacco use at the time of cancer diagnosis and during treatment and the utility of biochemically confirmed (BC) tobacco use assessment during cancer treatment has not been established. **Methods:** Patients with squamous cell carcinoma of the head and neck treated with definitive radiotherapy or chemoradiotherapy (CRT) were eligible for voluntary unpaid enrollment on an Institutional Review Board approved study. Structured entry and weekly SR tobacco use was obtained and weekly patient serum was obtained for BC assessment using serum cotinine. **Results:** Of 50 patients, median age is 56, 80% are male, 92% are Caucasian, 92% are stage III-IVB, 92% have laryngeal or oropharyngeal cancer, and 84% were treated with CRT. Any tobacco use history was reported in 82%. At baseline, 39% of patients SR current tobacco, but 46% had BC tobacco use. The accuracy of tobacco use during treatment based upon baseline SR tobacco assessment ranged from 73-92%, but weekly SR tobacco assessment increased accuracy to between 85-92%. In patients with current SR tobacco use at baseline, 63% tested positive by BC during the final week of treatment. In patients who denied SR tobacco use at baseline, 16% tested positive by BC and 7% tested positive by BC during the final week of treatment. The average positive predictive value of weekly SR was 92% and the average negative predictive value of weekly SR was 87%. Evaluation of patient specific SR characteristics over the course of treatment demonstrates a consistent group of patients who repeatedly misrepresent weekly SR tobacco use. Weekly secondhand smoke exposure was higher (range 31-61%) in patients who report current SR tobacco use at diagnosis as compared with patients who deny SR tobacco use at diagnosis (range 7-19%). Patient compliance for completion of all assessments was 93%. **Conclusions:** Repeated SR tobacco use assessment during cancer treatment increases the accuracy of identifying true tobacco use as compared with baseline assessment alone, but BC assessments may be necessary to detect true tobacco use status in a subset of patients who consistently misrepresent SR tobacco use. Patient compliance for BC assessment was high.

10626 General Poster Session (Board #35C), Mon, 8:00 AM-12:00 PM

Correlation of CA 27.29 and circulating tumor cells before, at the end, and 2 years after adjuvant chemotherapy in patients with primary breast cancer: The SUCCESS trial. Presenting Author: P. G. M. Hepp, University Dusseldorf, Duesseldorf, Germany

Background: While evidence for the prognostic value of circulating tumor cells (CTC) in the adjuvant setting is swiftly increasing the role of tumor markers like CA27.29 in this setting is still controversial. In the SUCCESS Trial CTC and CA27.29 have been measured before, after and 2 years after adjuvant chemotherapy. Our goal was to examine the correlation of these 2 factors at the 3 points in time. **Methods:** The SUCCESS Trial compared FEC-docetaxel (Doc) vs. FEC-Doc-gemcitabine (Doc-G) regime and 2 vs. 5 year treatment with zoledronat in 3754 patients with primary breast cancer (N+ or high risk). CA27.29 has been measured with ST AIA-PACK Ca27.29 reagent using MUC-1 for AIA-600II (Tosoh Bioscience, Belgium). The cutoff for positivity of CA27.29 was >31 U/ml. CTC were assessed with the CellSearchSystem (Veridex, USA). After immunomagnetic enrichment with an anti-Epcam-antibody, cells were labeled with anti-cytokeratin (8,18,19) and anti-CD45 antibodies to distinguish between epithelial cells and leukocytes. The cutoff for positivity was >1 CTC/15ml. **Results:** CA27.29 and CTC data are available of 2011 patients before, 1525 after and 1000 pts 2 years after chemotherapy. Before CHT 7.86% of pts were CA27.29 positive and 9.40% were CTC positive. 1.29% were CA27.29 and CTC positive (p=0.0015). After CHT 20.92% of pts were CA27.29 positive and 8.59% were CTC positive. 1.18% were CA27.29 and CTC positive (p=0.0346). 2 years after CHT 2.60% of pts were CA27.29 positive and 7.40% were CTC positive. 0.40% were CA27.29 and CTC positive (p=0.1115). Concerning correlation of positive CA27.29 with other prognostic factors we found a significant (p<0.05) correlation to histology and menopausal status before CHT. After CHT there was a significant correlation to menopausal status only and 2 years after CHT no correlation to any other prognostic factor could be found. **Conclusions:** In conclusion there seems to be a relationship between CA27.29 and CTC before treatment which resolves in the course of the treatment and beyond. Therefore clarification of the prognostic value of CA27.29 and CTC after adjuvant treatment as independent tools is needed.

10628 General Poster Session (Board #35E), Mon, 8:00 AM-12:00 PM

Pathoepidemiological patterns of contralateral breast cancers in Black and White women. Presenting Author: H. Nsouli-Maktabi, George Washington University, Washington, DC

Background: Women with one primary breast cancer are at greater risk for developing a second cancer in the contralateral breast, despite the protective effect of Tamoxifen treatment. Breast cancer incidence exhibits a Black-to-White incidence crossover at age 40. The purpose of this study was to investigate whether the patho-epidemiological patterns of second primary contralateral breast cancers in Black and White women are similar to those of the first primary tumors according to age at diagnosis of the first primary breast cancer. **Methods:** The Surveillance, Epidemiology, and End Results' (SEER) Registry 9 database was used to follow a total of 455,551 women, 415,664 White (91.24%) and 39,887 Black (8.76%) female breast cancer survivors, diagnosed at age 19 or older, for the occurrence of a second primary contralateral breast cancer between 1973 and 2007. Black and White women with a first primary in-situ or invasive breast cancer and a second primary contralateral breast cancer were analyzed by age at diagnosis, histologic tumor type, histological grade, tumor size, tumor markers, and number of positive lymph nodes. The cumulative incidence of a second contralateral breast cancer, which accounts for the competing risks of death and second non-breast cancers, was also explored among Black and White breast cancer survivors. **Results:** Second contralateral breast cancers in Black women were characterized by an earlier onset (Blacks= 59 yrs; Whites=67 yrs), higher incidence (Blacks= 8.7%, 95% CI = 8.3- 9.1; Whites= 7.7%, 95% CI=7.6 - 7.8) and more aggressive clinical presentation than in Whites. In contrast to first primary breast cancers, second primary breast cancers are more common in Black than in White women of all ages. **Conclusions:** Our results point to the possible bilaterality of many cases of breast cancer, and to a possible shared etiology between cancers in the two contralateral breasts.

10627 General Poster Session (Board #35D), Mon, 8:00 AM-12:00 PM

Determining argininosuccinate synthetase (ASS) expression in patients with melanoma treated with arginine depleting therapy. Presenting Author: V. Dinh, University of Miami, Miami, FL

Background: In our phase II trial in advanced melanoma (ASCO 2010) with arginine depleting therapy using ADI-PEG20 (Polaris Pharmaceuticals), we found that response to therapy correlated with tumor expression of enzyme, ASS. Normal cells which possess ASS can recycle citrulline to arginine and hence evade cell death, whereas melanoma tumors lacking ASS do not. Thus, 11 of 17 patients (pts) with ASS(-) tumors (by immunohistochemistry or real-time PCR) had evidence of antitumor activity versus 1 of 10 pts with ASS (+) tumors, (P value = 0.01). To study why not all ASS(-) melanoma pts respond to ADI-PEG20, we have developed a sensitive method to detect small amounts of mRNA by PCR. **Methods:** Total RNA was prepared from primary culture or cell lines and used for cDNA synthesis. Real-time PCR of ASS was performed using specific primers. The reaction was based on SYBR Green and performed in a Bio-Rad iCycler PCR machine equipped with a MyiQ module. $\Delta\Delta C_t$ method was used to calculate the relative ASS mRNA level. Levels of ASS mRNA were corrected with that of GAPDH and normalized with that of BJ-1 normal skin fibroblast, the value of which was set as 1. **Results:** Real-time PCR was performed in a panel of 15 primary cultures derived from patients' melanoma samples. The relative mRNA expression ranged from 0.0005 to 4.9. 5 primary cultures were treated with ADI-PEG for three days and assayed for ASS expression. In 2/5 with ASS mRNA <0.003, ASS expression was not inducible. The ASSmRNA of less than 0.001 exhibit higher sensitivity to ADI-PEG20 treatment (ID50 <0.1 ug/ml). However, in one cell line with baseline ASS mRNA of 0.003, it increased to 0.06 upon arginine deprivation. In a panel of melanoma cell lines we have also found that immunocytochemistry cannot detect ASS when their mRNA is <0.09. Thus, it is possible that the ASS(-) melanoma pts who do not respond to ADI-PEG20 have low detectable levels of mRNA which can be induced upon arginine deprivation. **Conclusions:** While the assay for ASS mRNA is cumbersome, it may better define melanoma pts who will respond to arginine depleting therapy with ADI-PEG20. Other factors which regulate ASS expression such as HIF-1alpha and cMyc are also being investigated. Supported by 1R01CA109578.

10629 General Poster Session (Board #35F), Mon, 8:00 AM-12:00 PM

Thymidilate synthase gene copy number as predictive marker of capecitabine efficacy in patients with breast cancer. Presenting Author: R. Audet, VM Institute of Research, Montreal, QC, Canada

Background: Expression of key enzymes of the thymidilate synthase pathway may affect the efficacy of 5-FU and the pro-drug capecitabine (C). The gene copy number of thymidilate synthase (TS), thymidine phosphorylase (TP), and dihydrofolate reductase (DHFR) (high vs low defined by the median for each) was assessed and correlated with time-to progression (TTP) and progression-free survival (PFS). **Methods:** Adult female patients with pathologically confirmed breast cancer and locally advanced or metastatic disease were treated with C 1000 mg/m² BID days 1-14 of a 21-day cycle. Human epidermal growth factor receptor 2 (HER2) and estrogen receptor (ER) expression was assessed immunohistochemically. Custom made TS, TP and DHFR FISH probes (Dako, Glostrup, Denmark) along with centromeric reference probes were used to evaluate gene copy number and gene to reference ratios in at least 60 morphologically intact non-overlapping nuclei. Markers dichotomized as high/low by the median were correlated with TTP and PFS using Cox proportional hazard models in 75, 65 and 25 patients for TYMS, TYMP and DHFR probes respectively. **Results:** In the overall patients population, higher TS gene copy number per cell was significantly associated with both decreased TTP (HR 1.76, 95% CI 1.07 to 2.90, p=0.026) and PFS (HR 1.86, 95% CI 1.14 to 3.04, p=0.036). 2. Higher TS gene copy number was significantly associated with a decrease in TTP (HR 1.96, 95% CI 1.09 to 3.45, p=0.025) and PFS (HR 2.07, 95% CI 1.18 to 3.65, p=0.011) in HER2 negative patients but not in HER2 positive patients (HR 1.42 and HR 1.42). 3. Higher TS gene copy number was also significantly associated with a decrease in TTP (HR 2.46, 95% CI 1.16 to 5.19, p=0.019) and PFS (HR 2.74, 95% CI 1.34 to 5.58, p=0.005) in ER positive patients but not in ER negative patients (HR 0.81 and HR 0.81). 4. TP and DHFR gene copy number were not significantly associated with clinical outcome. **Conclusions:** High TS gene copy number appears to be a predictor of poor outcome in ER positive, Her2 negative patients. With further refinement, TS gene copy number, assessed by FISH, may prove to be a useful and easily accessible marker for C sensitivity in human breast cancer and warrants further investigation.

10630 General Poster Session (Board #35G), Mon, 8:00 AM-12:00 PM

Biomarkers affecting metastasis and survival in paired tissues of 107 patients with metastatic breast cancer. *Presenting Author: E. Kim, Department of Oncology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea*

Background: The chemokine, CXCL12, and its receptor, CXCR4, have been known to play important roles in metastasis of several kinds of carcinoma. In preclinical study, VEGF regulates both CXCR4 expression and invasiveness. Tumors with higher Ki67 and EGFR expression were more prone to recur or metastasis. CD44+/CD24- subpopulation of breast cancer cells has highly invasive properties and involves in early stage of metastasis. **Methods:** Between 1995 and 2009, among patients with breast cancer who underwent breast cancer surgery, during follow-up, patients who developed distant metastasis were screened for availability of metastatic sites tissue for immunohistochemistry (IHC) analysis. We reviewed patients' medical records and assessed CXCR4, CXCL12, VEGF, Ki67, EGFR, PTEN, CD24 and CD44 by IHC for in primary sites and metastatic sites of these 107 patients. **Results:** The median age was 48 years (range, 26 to 70 years). Most tumors were invasive ductal carcinoma (IDC) (98/107, 91.6%) and more than 2cm (78/107, 72.8%). 56 patients were positive axillary lymph nodes (56/107, 52.3%). 103 patients were assessed for HER2 expression by IHC, 35 (35/107, 32.7%) patients showed HER2 expression. CXCR4 were significantly relevant to brain metastasis (OR 5.1 [CI 1.0-24.5], p=0.04). ER, PR expression and good histologic grade of tumor tissues were correlated significantly with longer overall survival (86.5 vs. 33.5, p=0.002, 84.4 vs. 41.5, p=0.005, 74.4 vs. 40.9, p=0.032). **Conclusions:** Higher CXCR4 in primary breast cancer sites was related to brain metastasis. ER, PR expression and good histologic grade were favorable prognostic factors for survival in patients with breast cancer.

10631 General Poster Session (Board #35H), Mon, 8:00 AM-12:00 PM

Redefining CTCs: Detection of additional circulating tumor cells using an antibody capture cocktail and HER2 FISH. *Presenting Author: F. Z. Bischoff, Biocept Inc., San Diego, CA*

Background: Most circulating tumor cell (CTC) platforms rely on EpCAM for capture and cytokeratin (CK) for detection. However, an important population of cells that display an epithelial-mesenchymal transition (EMT) phenotype will be missed. We report a new strategy to efficiently isolate a more heterogeneous population of CTCs using an antibody cocktail. **Methods:** In the first prospective study, blood (20 mL) was collected from 23 patients diagnosed with various late stage metastatic/recurrent cancer (breast, CRC, lung, prostate) following IRB approval. PBMCs were incubated with either EpCAM alone or a mixture of 10 capture antibodies to target both epithelial and mesenchymal cells. CTCs were subsequently captured in the CEE channels and detected with cytokeratin (CK) and CD45. A second prospective IRB approved study involving 54 patients diagnosed with late stage metastatic/recurrent breast cancer was performed using similar detection strategies (CK cocktail mixture and anti-CD45) with the addition of HER2 FISH to determine amplification status among captured CK+/CD45- and CK-/CD45- cells (presumable EMT cells). **Results:** In the first study, overall detection of CK+ cells was 83% with EpCAM alone and 93% with antibody cocktail. In addition, a median of 0.4 CK+ cells/mL and 1.0 CK+ cells/mL was observed using EpCAM and antibody cocktail, respectively. In the second study, CK+/CD45- cells were detected in 43 of 54 cases (80%). Among the 43 cases in which CK+/CD45- cells were detected, high concordance (93%) in HER2 status between primary tumor and CTCs was observed with Her2 amplification noted in both CK+/CD45- (50%) and CK-/CD45- (50%) cells. **Conclusions:** We have developed a novel and robust method for CTC enumeration that utilizes a cocktail of antibodies for the detection of a heterogeneous population of CTCs in multiple cancer types. Our findings suggest an important population of CK- cells is being missed by current stain criteria. Data also demonstrate that recovery of CTCs from peripheral blood using the CEE platform is efficient and suitable for FISH-based testing.

10632 General Poster Session (Board #36A), Mon, 8:00 AM-12:00 PM

Proof of concept of immuno-PET molecular imaging of met using ⁷⁶Br- and ⁸⁹Zr-labeled MetMab. *Presenting Author: M. Merchant, Genentech Inc., South San Francisco, CA*

Background: Oncogenic signaling via the hepatocyte growth factor (HGF)/Met pathway promotes tumor cell proliferation, migration, invasion and survival. High levels of Met are poorly prognostic for multiple cancer subtypes, including non small-cell lung cancer (NSCLC) and pancreatic cancer. MetMab is a monovalent monoclonal antibody that binds Met and blocks activation by HGF. Recent Phase II clinical studies show that the combination of MetMab with erlotinib in 2nd-3rd line NSCLC significantly prolongs overall survival in patients whose archive tumor samples display high levels of Met. We investigated the feasibility of identifying diagnostically positive tumors in mouse xenografts by non-invasive immuno-PET imaging using either ⁷⁶Br- or ⁸⁹Zr-labeled MetMab. **Methods:** MetMab was labeled directly with ⁷⁶Br or conjugated with bifunctional chelator based on desferrioxamine B (Df-Bz-SCN) to form Df-MetMab (1:1) for labeling with ⁸⁹Zr. Met binding affinities of both forms of radiolabeled MetMab were determined using cultured cells. Biodistribution and imaging studies were done in mice bearing NCI-H441 (NSCLC), MKN-45 (gastric), U-87 MG (glioblastoma) or KP4 (pancreatic) xenografts at various time points. Plasma and tumor samples were taken to determine shed Met and tumor Met levels, respectively. **Results:** ⁷⁶Br-MetMab and ⁸⁹Zr-Df-MetMab retained nM binding affinity for human Met. Imaging and biodistribution studies showed rapid uptake and slow clearance of both tracers specifically in tumor xenografts. MKN-45 tumor uptake of ⁷⁶Br-MetMab correlated with tumor mass, Met abundance, and phosphoMet content. ⁷⁶Br-MetMab biodistribution was independent of plasma shed Met level, suggesting that plasma Met will not affect tumor imaging sensitivity. Tumor-to-muscle intensity ratio and signal stability values were also good for both tracers, and both robustly identified xenografts with high levels of Met, whereas ⁸⁹Zr-Df-MetMab was clearly superior for imaging the lower Met abundance U-87 MG xenografts. **Conclusions:** Radiolabeled MetMab displays several promising features and warrants further development as an immuno-PET reagent to non-invasively image Met in vivo for diagnostic and prognostic endpoints.

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